Published in final edited form as:

Hum Mov Sci. 2017 August ; 54: 210–219. doi:10.1016/j.humov.2017.05.007.

Task-related and person-related variables influence the effect of low back pain on anticipatory postural adjustments

Jesse V. Jacobs^{a,b}, Courtney A. Lyman^a, Juvena R. Hitt^a, and Sharon M. Henry^{a,c}

^aDepartment of Rehabilitation and Movement Science, University of Vermont, 305 Rowell Building, 106 Carrigan Dr., Burlington, Vermont, 05405, USA

^bCenter for Physical Ergonomics, Liberty Mutual Research Institute for Safety, 71 Frankland Rd., Hopkinton, MA, 01748, USA

^cDepartment of Rehabilitation Therapy, University of Vermont Medical Center, 111 Colchester Ave., Burlington, VT, 05401, USA

Abstract

Background—People with low back pain exhibit altered postural coordination that has been suggested as a target for treatment, but heterogeneous presentation has rendered it difficult to identify appropriate candidates and protocols for such treatments. This study evaluated the associations of task-related and person-related factors with the effect of low back pain on anticipatory postural adjustments.

Methods—Thirteen subjects with and 13 without low back pain performed seated, rapid arm flexion in self-initiated and cued conditions. Mixed-model ANOVA were used to evaluate group and condition effects on APA onset latencies of trunk muscles, arm-raise velocity, and pre-movement cortical potentials. These measures were evaluated for correlation with pain ratings, Fear Avoidance Beliefs Questionnaire scores, and Modified Oswestry Questionnaire scores.

Findings—Delayed postural adjustments of subjects with low back pain were greater in the cued condition than in the self-initiated condition. The group with low back pain exhibited larger-amplitude cortical potentials than the group without pain, but also significantly slower arm-raise velocities. With arm-raise velocity as a covariate, the effect of low back pain remained significant for the latencies of postural adjustments but not for cortical potentials. Latencies of the postural adjustments significantly correlated with Oswestry and Fear Avoidance Beliefs scores.

Interpretation—Delayed postural adjustments with low back pain appear to be influenced by cueing of movement, pain-related disability and fear of activity. These results highlight the importance of subject characteristics, task condition, and task performance when comparing across studies or when developing treatment of people with low back pain.

Corresponding Author Information: Jesse V. Jacobs, University of Vermont, 305 Rowell Building, 106 Carrigan Dr., Burlington, VT 05405; Jesse.Jacobs@med.uvm.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Keywords

posture; anticipatory postural adjustment; low back pain; cortex; EEG

1. INTRODUCTION

Chronic low back pain (LBP) represents one of the most common and disabling health conditions worldwide (Hoy, et al., 2014). The potential causes of chronic LBP appear complex and multifactorial, but altered mechanisms of motor function may be a contributing factor (Hodges, 2011; Langevin & Sherman, 2007). People with chronic LBP exhibit changes in muscle activation onset for the purpose of postural coordination that may include changes in the onset latency of the anticipatory postural adjustment (APA) (Hodges & Richardson, 1996, 1999; Jacobs, Henry, & Nagle, 2009, 2010; Lomond, et al., 2015; Masse-Alarie, Beaulieu, Preuss, & Schneider, 2015; Masse-Alarie, Flamand, Moffet, & Schneider, 2012; Sadeghi, Talebian, Olyaei, & Attarbashi Moghadam, 2016; Tsao, Danneels, & Hodges, 2011; Tsao, Galea, & Hodges, 2008). The APA is represented by muscle activations within the supporting body segments to stabilize the body against anticipated forces that result from voluntary movement (Massion, 1992). The APA is learned, feed-forward and centrally programmed, such that the activation of the APA occurs before the onset of any movement-induced postural perturbation (Massion, 1992). Thus, any changes in APA onset latency due to LBP could affect the movements and forces to which a person's trunk and spine are exposed during movement (Mok, Brauer, & Hodges, 2011; Silfies, Bhattacharya, Biely, Smith, & Giszter, 2009).

Concurrent with their altered APA timing, people with LBP also exhibit altered cortical representations of postural muscles (measured in response to transcranial magnetic stimulation) as well as altered cortical function (measured by electroencephalography (EEG)) during postural tasks (Jacobs, et al., 2010; Masse-Alarie, Beaulieu, Preuss, & Schneider, 2016a; Masse-Alarie, et al., 2012; Sadeghi, et al., 2016; Schabrun, Elgueta-Cancino, & Hodges, 2015; Tsao, et al., 2011; Tsao, et al., 2008). In some studies, the location and size of a postural muscle's cortical representation or the amplitude of cortical activity directly correlated with APA onset latencies (Jacobs, et al., 2010; Masse-Alarie, Beaulieu, Preuss, & Schneider, 2016b; Tsao, et al., 2008). Thus, changes in the APA with LBP represent centrally mediated effects that would suggest motor retraining as a method of treatment if such changes in postural coordination contribute to chronic LBP.

The ability to direct such motor retraining treatment, however, is challenged because the effects of LBP on the APA, and the APA's relation to pain and disability, are not consistent: some studies report significantly delayed APA onset latencies in people with LBP (Hodges & Richardson, 1996, 1999; Masse-Alarie, et al., 2012; Mehta, Cannella, Smith, & Silfies, 2010; Sadeghi, et al., 2016), whereas other studies report delays in only sub-groups of subjects or do not report significant delays at all (Gubler, et al., 2010; Jacobs, et al., 2010; Marshall & Murphy, 2010; Mehta, et al., 2016; Silfies, Mehta, Smith, & Karduna, 2009). Such discrepancies could reflect methodological considerations, such as observed task performance variables, evaluated task conditions, and subject sample characteristics. For

example, regarding variables of task performance, the effect of LBP on the APA is best observed during high-velocity movements that most require an APA (Hodges & Richardson, 1999). In addition, the extent to which the cortex is recruited in preparation for movement could influence the extent to which LBP alters the APA (Jacobs, et al., 2010). Regarding task condition, a self-initiated versus a cue-initiated movement could also influence the effect of LBP on APA onset latencies and cortical preparation (Jacobs, et al., 2010; Lariviere, Butler, Sullivan, & Fung, 2013). Lastly, APA onset latencies could relate to clinical characteristics of the subject sample, such as their self-reported levels of pain, disability and fear of movement (Lariviere, et al., 2013; Marshall & Murphy, 2010).

As a group of studies, the literature suggests a complex relationship between LBP and APA onset latency due to multiple potential confounding factors, but a lack of standardization across studies renders it difficult to disentangle these complications. Thus, it may be helpful in a single study to evaluate the potential influence of factors related to task condition, task performance, and individual clinical characteristics on LBP-related changes in APA onset latency and cortical function. The purpose of this study was to evaluate whether cueing condition, observed arm-raise velocity, and a person's clinical characteristics associate with LBP-related differences in APA onset latencies and pre-movement cortical function. We hypothesized that the effects of LBP on the APA and movement-related cortical preparation would be significantly affected by task condition, task performance, and individual clinical characteristics as evidenced by (a) significant group-by condition interactions, (b) significant changes in the effects of LBP on the APA and cortical function with the introduction of arm-raise velocity as a covariate, and (c) significant correlations of APA onset latencies and amplitudes of cortical potentials with self-reported scores of pain intensity, pain-related disability, and pain-related fear of activity. Understanding these relationships could help direct more efficacious treatments that seek to ameliorate postural impairment with LBP.

2. METHODS

2.1. Subjects

Thirteen subjects with chronic or recurrent LBP and 13 subjects without LBP participated in the study (Table 1). This sample size represents a convenience sample achieved within the duration of the funded study. Subjects with LBP were included if their LBP required them to seek treatment or limited them on at least 3 activities (as determined by the Patient Specific Functional Scale; (Maughan & Lewis, 2010)), and if they experienced chronic or recurrent episodes for at least one year. Subjects with either chronic or recurrent LBP were included because it is known that changes in postural coordination remain evident whether or not a person exhibits concurrent pain symptoms in people with a history of LBP (D'Hooge, et al., 2013; D. MacDonald, Moseley, & Hodges, 2009, 2010; D. A. Macdonald, Dawson, & Hodges, 2011). Subjects were excluded if they reported neurological, psychiatric, cardiovascular, or musculoskeletal disorders other than back pain as well as uncorrected vision problems, vertebral fracture, tumor or infection, spinal stenosis, previous spinal surgery, systemic infection, current pregnancy, history of any surgery in the three months prior to testing, scoliosis or kyphosis, injury to the lower extremity, or radiating pain below the knee. Subjects were also excluded if they were receiving disability compensation for

their LBP, or if they were in litigation because of the LBP. Subjects without LBP were excluded if they had any of the above-listed criteria as well as if they had a history of back pain that required them to seek treatment or resulted in limited activity. All subjects were currently employed or active as a full-time student or homemaker. Subjects participated only after providing written informed consent, and the protocol was approved by the local institutional review board.

2.2. Data Collection and Processing

2.2.1 Questionnaires—Subjects first completed a set of questionnaires that included a health history form, the Numeric Pain Rating Scale (Childs, Piva, & Fritz, 2005), the Modified Oswestry Disability Questionnaire (Fritz & Irrgang, 2001), and the Fear Avoidance Beliefs Questionnaire (FABQ; (Waddell, Newton, Henderson, Somerville, & Main, 1993)). These questionnaires were selected in order to provide insight regarding associations of APA onset latencies and cortical function with pain intensity, pain-related disability, and pain-related fear of activity. The Numeric Pain Rating identifies a person's reported pain intensity on a 0–10 point rating scale. The Oswestry questionnaire is reported as a percent possible level of disability following the summation of ten 0–5 point item ratings. The FABQ generates a physical activity score and a work score. The physical activity score represents the sum of four 0–6 point item ratings, and the work score represents the sum of seven 0–6 point item ratings. Higher scores represent greater pain severity, disability, and fear of activity.

2.2.2. Postural Task—The task was for the subjects to raise the dominant arm as fast as possible from an initial seated position with the arms hanging vertically to a final position with the arms outstretched horizontally in front of them – approximately 90 degrees of forward shoulder flexion (Fig. 1A). The subjects sat upright on a stable, flat, hard-surface, adjustable bench with their back unsupported and their gaze fixated on a point located at eye level approximately 2 meters in front of them. The subjects were initially positioned with the bench height at the subjects' lateral femoral epicondyle, and they sat with the midpoint of their thigh (defined from the lateral femoral epicondyle to the greater trochanter) aligned to the front edge of the seat as well as with their trunk (defined from the greater trochanter to the acromion) oriented vertically and perpendicular to the seat. The feet were placed in parallel at a heel-to-heel stance width equal to 11% of body height (McIlroy & Maki, 1997) and were positioned in the anterior-posterior plane so as to create 10 degrees of ankle dorsiflexion. This initial position was intermittently verified during testing by visual inspection with a goniometer and taped outlines of the feet. A seated task was chosen in order to localize postural control to the trunk, because it is known that people with LBP compensate with distal lower-limb control during standing postural tasks (Claeys, Brumagne, Dankaerts, Kiers, & Janssens, 2011; Jacobs, Henry, Jones, Hitt, & Bunn, 2011; Jacobs, Roy, Hitt, Popov, & Henry, 2016; Sadeghi, et al., 2016).

The subjects completed 40 trials of arm raises in a self-initiated condition and 40 trials in a cued condition. In the self-initiated condition, subjects were instructed to raise their arm as fast as possible approximately once every 10 seconds without explicitly counting, and that the experimenter would provide feedback about their rate if the interval between arm raises

was too short. This inter-trial interval was requested in order to allow for baseline signals to return before the start of the next trial. For the cued condition, subjects were instructed to respond as soon as possible and with as fast of an arm raise as possible to a single auditory cue that was provided at random, unpredictable inter-trial intervals of 8 to 15 seconds. The auditory cue was presented through earphones worn by the subjects. For both conditions, subjects were asked to rest after every 20 trials in order to prevent discomfort and fatigue. Subjects were also instructed to request rest when needed, but only one subject requested rest beyond that which was provided every 20 trials.

2.2.3. Electromyography (EMG)—For recording APA onset latencies during the arm-raise task (Fig. 1B), subjects were prepared for recording by shaving the skin overlying the recorded muscles and then cleaning the skin with a conductive gel to obtain impedances below 10 k Ω . Bipolar surface EMG electrodes (1-cm silver/silver-chloride disk electrodes with fixed 2-cm inter-electrode distance; Myotronics, Kent, WA, USA) were applied as previously reported (Jacobs et al., 2011) to the left and right lumbar erector spinae (ES), internal oblique (IO), and external oblique (EO) muscles. Electrodes were also placed over the anterior deltoid of the dominant arm (as described by www.seniam.org). All subjects were right-arm dominant except one subject with LBP, so EMG data are presented as contralateral or ipsilateral to the arm movement. We recognize that a surface EMG signal of the IO muscle also includes signal from the transversus abdominus muscle (Marshall & Murphy, 2003), but we maintain the IO naming convention for simplicity.

The EMG signals were sampled at 1000 Hz, pre-amplified by 1000 at the skin's surface and then amplified further for a total amplification of 5000–10000. EMG data were synchronously recorded with the motion capture data through VICON Nexus software (VICON, Denver, CO, USA). For offline processing using Matlab software (Matlab, Natick, MA, USA), the EMG signals were band-pass filtered at 30–400 Hz with a Butterworth filter, baseline corrected by subtracting the mean of the signal, and full-wave rectified. The high-pass limit was set to minimize cardiac artifact in the EMG signals of our evaluated trunk muscles (Drake & Callaghan, 2006). The integrated protocol method was used to identify EMG activation onset, in which the maximum difference between the integrated signal and an amplitude-normalized integral of the linear envelope is identified. The integrated protocol method is less susceptible to changes in baseline amplitude or to false onset detection from trunk muscles compared with traditional threshold techniques (Allison, 2003) and has been used before for APA onset detection during rapid arm raises of subjects with LBP (Marshall & Murphy, 2010). The onset of EMG activation was visually verified with an interactive graphing function. In some cases, an onset of EMG activation could not be identified. Specifically, the average incidence of EMG activation onset ranged from 74–94% of trials across muscles for each condition, and the incidence of EMG activation onset did not significantly differ between groups for any individual muscle within each condition [range of $T = 0.62$ – 1.97 ; range of $P = 0.069$ – 0.54]. For each trial, APA onset latencies were derived by subtracting the onset of deltoid EMG activation from that of the IO, EO, and ES muscles. Deltoid response latencies relative to the cue were also determined for the cue condition. The APA onset latencies were then averaged by subject and condition (self-initiated and cued) for analysis.

2.2.4. Kinematics—Subjects were also prepared for passive-marker motion capture (7-camera system; VICON, Denver, CO) that included placing reflective markers bilaterally over the subjects' acromion, lateral humeral condyle, styloid process, greater trochanter, lateral femoral condyle, lateral malleolus, and 5th metatarsal (Fig. 1A). The motion capture data were used to quantify the peak rotational velocity of the arm flexion in the sagittal plane.

The motion capture data were sampled at 100 Hz. Using Matlab software, marker position data were low-pass filtered at 10 Hz. The arm was defined as a single segment from the markers placed on the acromion and the styloid process in order to generate the sagittal angular displacement of the arm. The peak rotational velocity of the arm motion was determined by first differentiating the displacement signal and then identifying the maximum amplitude of the velocity signal for each trial. The peak arm-raise velocities were then averaged by subject and condition for analysis.

2.2.5. EEG—The subjects wore a Waveguard 128-channel EEG head cap (sintered silver/silver-chloride electrodes; standard 10/5 system placement (Oostenveld and Praamstra, 2001); Advanced Neuro Technology, Enschede, the Netherlands). A conductive electrode gel (Electro-gel; Electro-Cap International; Eaton, OH, USA) was used to obtain impedances below 10 k Ω . The EEG was recorded to derive the pre-movement negative voltage potential in order to gain insight into the neural preparation of movement by the cerebral cortex. In the self-initiated condition, a slow-wave negative voltage potential (the Bereitschaftspotential) precedes movement, which represents motor preparation by the supplementary and primary motor cortex (Kornhuber & Deecke, 1964; Shibasaki & Hallett, 2006). We refer to the potential recorded in this study as pre-movement negativity because the study also includes a single-cue condition that (1) does not represent the self-initiated condition that defines recording of the Bereitschaftspotential, and (2) does not involve the 2-stimulus paradigm that defines recording of contingent negative variation. Unpredictably timed, single-cue conditions are known to reduce or abolish pre-movement negativity (Walter, Cooper, Aldridge, McCallum, & Winter, 1964).

EEG data were collected at 1024 Hz using a DC amplifier and pre-processed using ASA software version 4.7.3 (Advanced Neuro Technology, Enschede, Netherlands). Following collection, blinking artifacts were removed using the artifact correction tool of the ASA software, selecting the two components that most represented the artifacts' characteristics. The EEG recordings were band-pass filtered from 0.05 to 30 Hz. The EMG signals were simultaneously input by BNC cable from the EMG amplifier to the EEG and Vicon systems. The signal from the deltoid EMG was used to epoch the EEG recordings. Epochs were spliced from the continuously recorded EEG data from 4 seconds before to 4 seconds after deltoid onset. The Cz (midline central) electrode was chosen for analysis based on a previous study that maximal potential amplitudes are derived from that location for this task when performed by subjects with and without LBP (Jacobs, et al., 2010).

The EEG signals were time realigned to the onset of the contralateral ES muscle, which represented the first muscle to activate, because pre-movement negativity is known to be greater in amplitude if aligned to the onset of the APA instead of to the prime movement

(Saitou, Washimi, Koike, Takahashi, & Kaneoke, 1996). The EEG signals were then baseline corrected by subtracting the average value of a baseline epoch defined from –3000 to –2950 ms prior to contralateral ES muscle onset. Trial data from the Cz electrode were evaluated for artifacts associated with amplifier drift, electrode movement, and muscle activity. On average (95% CI), 35 (34–37) of the 40 trials per condition were considered artifact free and used for analysis. No significant differences were evident between groups or conditions in the number of artifact-free trials used for analysis [group: $F = 0.04$, $P = 0.84$; condition: $F = 1.06$, $P = 0.31$; group-by-condition: $F = 0.02$, $P = 0.89$]. Trial data were averaged by condition and subject. From the average waveforms, the amplitude of the pre-movement negativity was determined as the average voltage amplitude of the final 100 ms preceding contralateral ES muscle onset (Fig. 1C).

2.3. Statistical Analysis

Differences between groups in anthropometric and questionnaire measures were determined by two-tailed independent-samples t tests or Mann-Whitney U tests, depending on whether the data met assumptions for the t test. APA onset latencies, peak arm-raise velocities, and amplitudes of pre-movement negativity were analyzed with mixed-model ANOVA to determine differences between groups (2 levels; with and without LBP) and conditions (2 levels; self-initiated and cued). Analysis was conducted with and without peak arm-raise velocity as a covariate in order to determine the influence of arm-raise velocity on LBP-related differences in the APA and pre-movement cortical function. Greenhouse-Geisser corrections were applied to correct for any violations on the assumption of sphericity.

Within the group with LBP, Pearson's correlation coefficients were used to examine associations of questionnaire measures or arm-raise velocity with APA onset latencies and pre-movement negativity amplitudes found to be significantly different with LBP in order to provide insight about the influence of individual clinical characteristics or performance characteristics on APA onset latencies and pre-movement cortical function.

Measures were tested for the assumption of normality with Shapiro-Wilks tests. If data did not meet this assumption, non-parametric tests for group differences were evaluated by Mann-Whitney U tests, and Spearman's correlation coefficients were evaluated in place of the Pearson's coefficients. Statistical significance was set at a level of 0.05. The results of the parametric and non-parametric statistics were always consistent with regard to comparisons or correlations that did or did not reach the level of significance, thus we only report the parametric statistics. All analyses were performed in SPSS version-21 software (IBM, Armonk, NY, USA).

3. RESULTS

3.1. Comparisons Between Groups With and Without LBP

The subject groups' anthropometric measures were not statistically different, and the subjects with and without LBP significantly differed for Numeric Pain Rating scores, Oswestry Disability scores, and FABQ physical activity and work scores (Table 1).

Qualitatively, the group with LBP exhibited delayed APA onset latencies that appeared more evident in the cued condition than in the self-initiated condition (Fig. 2A). Statistically significant delays, however, were only evident at the contralateral EO muscle [group main effect: $F = 4.54$, $P = 0.044$; group-by-condition interaction: $F = 3.96$, $P = 0.058$]. When applying peak arm-raise velocity as a covariate, the group-by-condition interaction was significant [$F = 10.18$, $P = 0.0041$] such that delayed APAs with LBP were greater in the cued condition than in the self-initiated condition, independent of a significant effect of arm-raise velocity [$F = 7.33$, $P = 0.013$]. Even when accounting for the 6 ANOVAs that separately analyzed the latencies of each muscle with a Bonferroni-corrected alpha of 0.0083, this group-by-condition interaction remains significant. There were no significant group differences in contralateral ES muscle onset (the first muscle to activate) [group main effect: $F = 0.013$, $P = 0.91$; group-by-condition interaction: $F = 1.68$, $P = 0.21$] or in deltoid onset relative to the cue [$T = 0.49$, $P = 0.63$]; thus, reaction times or latencies between first muscle onset to deltoid onset were not significantly different between the groups with and without LBP.

The group with LBP exhibited significantly greater (more negative) pre-movement negativity amplitudes across both conditions (Fig. 2B) [group main effect: $F = 4.97$, $P = 0.036$; group-by-condition interaction: $F = 0.50$, $P = 0.49$]. Upon noticing that the group with LBP appeared to maintain pre-movement negativity in the cued condition, a post-hoc one-sample t-test verified that the pre-movement negativity was significantly different from zero for the group with LBP [$T = 3.11$, $P = 0.009$] but not for the group without LBP [$T = 0.24$, $P = 0.81$]. The effect of LBP on pre-movement negativity, however, did not remain significant with peak arm-raise velocity included as a covariate [group main effect: $F = 2.76$, $P = 0.11$; group-by-condition interaction: $F = 0.61$, $F = 0.45$] despite no significant independent effect of peak arm-raise velocity on pre-movement negativity [$F = 0.015$, $P = 0.90$]. Peak arm-raise velocity was significantly slower for the group with LBP across both conditions compared to the group without LBP (Fig. 2C) [group main effect: $F = 13.09$, $P = 0.0014$; group-by-condition interaction: $F = 0.22$, $F = 0.64$].

3.2. Correlations of APA Onset or Pre-Movement Negativity With Clinical Characteristics and Arm-Raise Velocity

Correlation analyses with clinical questionnaire scores were undertaken with the APA onset latencies of the contralateral EO muscle, the only muscle to exhibit significantly delayed APA onset latencies with LBP. APA latencies of the contralateral EO muscle significantly correlated with Oswestry scores and FABQ physical activity scores, but not with Numeric Pain Ratings or FABQ work scores (Fig. 3). When accounting for the 8 correlations analyzed between EO latencies and clinical measures, only the correlation with FABQ physical activity scores exceeded a Bonferroni-corrected threshold of 0.00625. Amplitudes of pre-movement negativity did not significantly correlate with any of the clinical questionnaire scores [range of $R = -0.24$ – 0.20 , range of $P = 0.43$ – 0.98], nor did peak arm-raise velocity [range of $R = 0.038$ – 0.38 , range of $P = 0.21$ – 0.90].

Correlations between arm-raise velocities and APA onset latencies of the contralateral EO were only significant for the group without LBP [self-initiated: $R = -0.76$, $P = 0.003$; cued:

−0.62, $P = 0.023$], but not for the group with LBP [self-initiated: $R = 0.005$, $P = 0.99$; cued: 0.13, $P = 0.67$], such that for the group without LBP, earlier APA onset latencies correlated with faster arm-raise velocities. Arm-raise velocities did not significantly correlate with amplitudes of pre-movement negativity for either group or condition [range of $R = -0.25$ –0.43, range of $P = 0.16$ –0.68].

4. DISCUSSION

As hypothesized, LBP-related delay in APA onset latency was affected by cue condition and correlated with the subjects' reported disability and fear of physical activity. Contrary to the hypothesis, however, the effect of LBP on APA onset latency remained significant despite differences in arm-raise velocity. Arm-raise velocity significantly correlated with APA onset latency for only the group without LBP, but not for the group with LBP, demonstrating that velocity's significant effect as a covariate on the analysis of APA onset latency was due to its relationship with APA onset latency for only the group without LBP, not the group with LBP. Arm-raise velocity was not correlated with pre-movement negativity amplitudes. Thus, although conditions of sub-maximal velocity can diminish both the requirement for an APA and the LBP-related effect on the APA (Hodges & Richardson, 1999), under conditions in which subjects are asked to raise their arm as quickly as possible, arm-raise velocity did not affect LBP-related differences in APA onset latency.

With regard to the factors found to influence the effect of LBP on APA onset latency, cue condition does enhance the ability to detect delayed APAs with LBP, and individuals with higher levels of reported disability and fear of physical activity also associate with greater delay of the APA. These findings are consistent with the literature, such that those studies that report significantly delayed APAs with LBP employed a cued paradigm (Hodges & Richardson, 1996, 1999; Masse-Alarie, et al., 2012; Mehta, et al., 2010; Sadeghi, et al., 2016), compared to the study that did not identify significant differences in a self-initiated paradigm (Jacobs, et al., 2010). Other methodological considerations may explain why other studies did not report significant delays, such as employing ultrasonography rather than EMG (Gubler, et al., 2010), or examining subjects with acute or sub-acute LBP (Mehta, et al., 2016) rather than subjects with chronic LBP. Increased delay of the APA with LBP was previously reported to associate with an increased fear of movement, although the direction of this relationship may be muscle specific (Lariviere, et al., 2013). Indeed, knowing that delayed muscle activation may relate to subgroups of people with LBP based on duration (acute, sub-acute, recurrent, or chronic), laterality, or spinal instability (Masse-Alarie, et al., 2016b; Mehta, et al., 2016; Silfies, Mehta, et al., 2009), we caution that our results and those of the literature at large may not easily generalize beyond the sample selection criteria.

Although our study qualitatively identified LBP-related delays at the IO and EO muscles, this study is in the minority to elicit the most significant effects at the EO muscle. Inter-study differences in whether statistically significant LBP-related delays are evident at the IO, EO or lumbar muscles could reflect any combination of task-related and person-related factors. For example, we utilized a seated task that may have enhanced the functional relevance of the EO compared to when standing; the arm raise was also approximately 90 degrees of motion at maximum voluntary speed and under self-initiated or simple reaction-

time conditions. Further, we did not weight the subjects' arms to enhance the postural challenge of the task, but subjects did perform 40 trials per condition. Our subjects had chronic or recurrent LBP over at least one year with varied, but generally low, levels of pain and disability at the time of testing. Because studies generally differ on many of these factors, it is not possible to determine why, for example, some studies report significant LBP-related delays at the IO muscle and others at the EO muscle. Further study on a large cohort sufficient for sub-group analyses of performance across multiple tasks may be necessary to clarify this question.

Contrary to our study in which we identified no significant correlation of APA onset latencies with numeric pain ratings but significant correlation with Oswestry disability scores, Marshall and Murphy (2010) found a significant positive correlation between pain severity and APA onset latency, but not with Oswestry disability scores; although their analysis focused on the IO muscle as opposed to the EO muscle that represented the primary analysis of this study. Further, APA onset latency correlated with FABQ physical activity scores but not work activity scores, which may reflect specificity regarding how the APA during a rapid arm raise relates to the type of physical activity. In specific, our subject sample's work activity may not be as relevant as other physical activities to the motor control of a ballistic arm movement. Thus, the relationship of APA onset latency to pain, disability, and fear of activity may be muscle dependent and activity dependent.

In addition to delayed APAs, the group with LBP exhibited enhanced amplitudes of pre-movement negativity. In a previous study of bereitschaftspotential amplitudes associated with self-initiated arm raises, a non-significant trend for increased amplitudes with LBP was reported (Jacobs, et al., 2010), whereas significantly larger amplitudes of contingent negative variation were reported in another study of cued arm raises (Sadeghi, et al., 2016). In this study, no significant group-by-condition interaction on pre-movement negativity amplitudes was identified, but the group without LBP exhibited no pre-movement negativity in the cued condition, whereas the group with LBP maintained a pre-movement negativity. Thus, LBP-related differences in pre-movement cortical function, similar to findings regarding APA onset latencies, may be more easily revealed under cued conditions.

Cued conditions render unique preparatory cortical functions compared to self-initiated conditions that might provide insight into why cued conditions elicit greater LBP-related differences in cortical preparation and postural coordination. Regarding the previous study on contingent negative variation with LBP (Sadeghi, et al., 2016), the contingent negative variation is evident in 2-cue paradigms in which a warning cue precedes an imperative cue to respond and the two cues are of a known inter-stimulus interval. Unlike the bereitschaftspotential, which is more specifically associated with the function of supplementary and primary motor regions of the cerebral cortex (Hamano, et al., 1997; Shibasaki & Hallett, 2006), the contingent negative variation includes a more diverse set of generators – supplementary motor, primary motor, parietal, occipital, temporal, prefrontal – and functionally associates with anticipation of movement as well as motor preparation (Hamano, et al., 1997; van Boxtel & Brunia, 1994). It is possible that, in our paradigm of an unpredictable reaction-time cue condition, the group with LBP maintained an enhanced level of cortical function because they maintained a greater level of anticipation for movement.

Although this speculation is not corroborated by correlations of pre-movement negativity with FABQ scores – which might be expected if fear of physical activity were to relate to pre-motor cortical function associated with anticipation for an impending movement – the FABQ represents a generalized proxy for fear of movement; thus, a more specific questionnaire on the fear to produce this study's arm-raise task may have been a more appropriate test of this speculation.

In addition to potential group differences in the cortical processing of anticipation, pre-movement cortical processing of movement velocity may also mediate the effects of group on pre-movement negativity. Previous studies have demonstrated that pre-movement negativity relates to the velocity of the subsequent movement (Khanmohammadi, Talebian, Hadian, Olyaei, & Bagheri, 2015; Lang, 2003), suggesting that motor cortical preparation may include processing of the impending movement's velocity. Thus, the lost statistical effect of group on pre-movement negativity amplitudes when including velocity as a covariate suggests that the group difference in cortical motor preparation may be at least partially mediated by cortical processing of the impending movement's velocity. Regardless of the mechanism underlying the increased pre-movement cortical activity of subjects with LBP, our findings corroborate previous interpretations that LBP associates with enhanced activity, and perhaps influence, of the cerebral cortex on postural coordination (Jacobs, et al., 2010; Jacobs, et al., 2016; Masse-Alarie, et al., 2016b; Sadeghi, et al., 2016).

Overall, although not the intended objective of this study, the results suggest a non-motor factor may possibly interact on the delayed APAs associated with LBP. In specific, the (a) effect of LBP on the coordination of the APA is altered by cue condition despite similar instruction regarding the type, extent, and speed of movement, (b) enhanced pre-movement cortical function did not seem strongly related to actual motor behavior and may reflect influence of non-motor processes, (c) APA onset was specific to movement velocity for the group without LBP, but this relationship of the APA to the primary movement was lost for the group with LBP, and (d) APA onset latencies correlated with disability and fear of physical activity, which can be influenced by or directly reflect cognitions about pain rather than purely reflecting actual physical capacity. Further study is required, however, to determine the role of pain-related cognitions on altering the APA with the experience of LBP.

When considering the clinical relevance of these findings, it might be tempting to recommend that specific motor control training focus on high-velocity, cued movements of individuals who report high levels of disability and fear of physical activity because these situations associated with a greater effect of LBP on the APA. However, because of the conflicting results across the literature on shared constructs tested in this study, and the potential that other factors untested in this study may also be relevant to the effect of LBP on the APA (e.g., the method of data collection, the choice of task or task characteristics other than velocity, and the muscles selected for assessment (Lariviere, et al., 2013; Masse-Alarie, et al., 2015; Mehta, et al., 2016)), it would be inappropriate to make such bold recommendations at this time without more independent confirmation of these relationships. Indeed, it has been theorized that LBP can render a redistribution of muscle coordination and mechanical behavior that is variable across individuals in attempt to offer short-term

protection against further injury or threat of injury, but perhaps to the detriment of long-term consequences for recurrent or chronic pain (Hodges, 2011). Due to the inter-individual variability of the response to LBP, the challenge to specify a sub-population of people with LBP who would benefit from motor control training interventions as well as the challenge to specify the mechanism of action for treatment infringes on the potential of motor control training interventions for the treatment of LBP. Nevertheless, given the multifactorial nature of the potential contributing factors to LBP (Langevin & Sherman, 2007), some type of motor component to treatment as part of a multimodal treatment plan is likely to be of some benefit. The nature of this motor component, however, and its relationship to non-motor components, remain unspecified (O’Keeffe, et al., 2016; Saragiotto, et al., 2016), thus behooving researchers and clinicians alike to continue seeking an optimal treatment approach through a combination of further mechanistic and trial studies.

4.1. Conclusions

In a single cohort, this study identified that factors related to cue condition and individual characteristics of disability and fear of physical activity appear to influence the effect of LBP on the APA. Although sub-maximal movement velocities may also affect the extent that LBP affects the APA (Hodges & Richardson, 1999), under conditions of maximal velocity that strongly elicit an APA, variations in arm-raise velocity do not seem to alter the effect of LBP on the APA. The lack of effect of velocity may be because the relationship of APA onset to movement velocity is lost with LBP. Although these findings provide some direction toward understanding the circumstances in which LBP most associates with impaired APA function in order to direct clinical intervention, further research is needed to independently confirm the relationships identified in this study and to elucidate other relevant factors that could not be addressed within the scope of this study. This study’s findings, however, highlight the challenges that exist in order to identify the people with LBP and activities in which they engage that would most benefit from interventions on postural coordination.

Acknowledgments

The authors wish to thank Bryana Park, Kim Copley, and Karen Lomond for their participation in data collection and processing. We also thank Dee Physical Therapy, Evolution Physical Therapy and Yoga, University of Vermont Medical Center (previously Fletcher Allen Health Care), Timberlane Physical Therapy, and Physical Therapy of Copley Hospital for help with subject recruitment.

Funding: The study was supported by the National Institutes of Health, National Center for Research Resources, award number P20 RR016435. The sponsor was not involved in study design, the collection, analysis and interpretation of data, writing of the report, or the decision to submit for publication

References

- Allison GT. Trunk muscle onset detection technique for EMG signals with ECG artefact. *J Electromyogr Kinesiol.* 2003; 13:209–216. [PubMed: 12706601]
- Childs JD, Piva SR, Fritz JM. Responsiveness of the numeric pain rating scale in patients with low back pain. *Spine (Phila Pa 1976).* 2005; 30:1331–1334. [PubMed: 15928561]
- Claeys K, Brumagne S, Dankaerts W, Kiers H, Janssens L. Decreased variability in postural control strategies in young people with non-specific low back pain is associated with altered proprioceptive reweighting. *Eur J Appl Physiol.* 2011; 111:115–123. [PubMed: 20824281]

- D'Hooge R, Hodges P, Tsao H, Hall L, Macdonald D, Danneels L. Altered trunk muscle coordination during rapid trunk flexion in people in remission of recurrent low back pain. *J Electromyogr Kinesiol.* 2013; 23:173–181. [PubMed: 23079004]
- Drake JD, Callaghan JP. Elimination of electrocardiogram contamination from electromyogram signals: An evaluation of currently used removal techniques. *J Electromyogr Kinesiol.* 2006; 16:175–187. [PubMed: 16139521]
- Fritz JM, Irrgang JJ. A comparison of a modified Oswestry Low Back Pain Disability Questionnaire and the Quebec Back Pain Disability Scale. *Phys Ther.* 2001; 81:776–788. [PubMed: 11175676]
- Gubler D, Mannion AF, Schenk P, Gorelick M, Helbling D, Gerber H, Toma V, Sprott H. Ultrasound tissue Doppler imaging reveals no delay in abdominal muscle feed-forward activity during rapid arm movements in patients with chronic low back pain. *Spine (Phila Pa 1976).* 2010; 35:1506–1513. [PubMed: 20431436]
- Hamano T, Luders HO, Ikeda A, Collura TF, Comair YG, Shibasaki H. The cortical generators of the contingent negative variation in humans: a study with subdural electrodes. *Electroencephalogr Clin Neurophysiol.* 1997; 104:257–268. [PubMed: 9186240]
- Hodges PW. Pain and motor control: From the laboratory to rehabilitation. *J Electromyogr Kinesiol.* 2011; 21:220–228. [PubMed: 21306915]
- Hodges PW, Richardson CA. Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. *Spine (Phila Pa 1976).* 1996; 21:2640–2650. [PubMed: 8961451]
- Hodges PW, Richardson CA. Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. *Arch Phys Med Rehabil.* 1999; 80:1005–1012. [PubMed: 10489000]
- Hoy D, March L, Brooks P, Blyth F, Woolf A, Bain C, Williams G, Smith E, Vos T, Barendregt J, Murray C, Burstein R, Buchbinder R. The global burden of low back pain: estimates from the Global Burden of Disease 2010 study. *Ann Rheum Dis.* 2014; 73:968–974. [PubMed: 24665116]
- Jacobs JV, Henry SM, Jones SL, Hitt JR, Bunn JY. A history of low back pain associates with altered electromyographic activation patterns in response to perturbations of standing balance. *J Neurophysiol.* 2011; 106:2506–2514. [PubMed: 21795622]
- Jacobs JV, Henry SM, Nagle KJ. People with chronic low back pain exhibit decreased variability in the timing of their anticipatory postural adjustments. *Behav Neurosci.* 2009; 123:455–458. [PubMed: 19331469]
- Jacobs JV, Henry SM, Nagle KJ. Low back pain associates with altered activity of the cerebral cortex prior to arm movements that require postural adjustment. *Clin Neurophysiol.* 2010; 121:431–440. [PubMed: 20071225]
- Jacobs JV, Roy CL, Hitt JR, Popov RE, Henry SM. Neural mechanisms and functional correlates of altered postural responses to perturbed standing balance with chronic low back pain. *Neuroscience.* 2016; 339:511–524. [PubMed: 27771534]
- Khanmohammadi R, Talebian S, Hadian MR, Olyaei G, Bagheri H. Preparatory postural adjustments during gait initiation in healthy younger and older adults: Neurophysiological and biomechanical aspects. *Brain Res.* 2015; 1629:240–249. [PubMed: 26453832]
- Kornhuber, HH., Deecke, L. PFLUGERS ARCHIV-EUROPEAN JOURNAL OF PHYSIOLOGY. Vol. 281. SPRINGER VERLAG; 175 FIFTH AVE, NEW YORK, NY 10010: 1964.
Hirnpotentialänderungen beim Menschen vor und nach Willkurbewegungen dargestellt mit Magnetbandspeicherung und Rückwärtsanalyse; p. 52
- Lang, W. The Bereitschaftspotential. Springer; 2003. Surface recordings of the Bereitschaftspotential in normals; p. 19-34.
- Langevin HM, Sherman KJ. Pathophysiological model for chronic low back pain integrating connective tissue and nervous system mechanisms. *Med Hypotheses.* 2007; 68:74–80. [PubMed: 16919887]
- Lariviere C, Butler H, Sullivan MJ, Fung J. An exploratory study on the effect of pain interference and attentional interference on neuromuscular responses during rapid arm flexion movements. *Clin J Pain.* 2013; 29:265–275. [PubMed: 23369930]

- Lomond KV, Jacobs JV, Hitt JR, DeSarno MJ, Bunn JY, Henry SM. Effects of low back pain stabilization or movement system impairment treatments on voluntary postural adjustments: a randomized controlled trial. *Spine J.* 2015; 15:596–606. [PubMed: 25452017]
- MacDonald D, Moseley GL, Hodges PW. Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. *Pain.* 2009; 142:183–188. [PubMed: 19186001]
- MacDonald D, Moseley GL, Hodges PW. People with recurrent low back pain respond differently to trunk loading despite remission from symptoms. *Spine (Phila Pa 1976).* 2010; 35:818–824. [PubMed: 20228708]
- Macdonald DA, Dawson AP, Hodges PW. Behavior of the lumbar multifidus during lower extremity movements in people with recurrent low back pain during symptom remission. *J Orthop Sports Phys Ther.* 2011; 41:155–164. [PubMed: 21212497]
- Marshall P, Murphy B. The validity and reliability of surface EMG to assess the neuromuscular response of the abdominal muscles to rapid limb movement. *J Electromyogr Kinesiol.* 2003; 13:477–489. [PubMed: 12932422]
- Marshall P, Murphy B. Delayed abdominal muscle onsets and self-report measures of pain and disability in chronic low back pain. *J Electromyogr Kinesiol.* 2010; 20:833–839. [PubMed: 19853473]
- Masse-Alarie H, Beaulieu LD, Preuss R, Schneider C. Task-specificity of bilateral anticipatory activation of the deep abdominal muscles in healthy and chronic low back pain populations. *Gait Posture.* 2015; 41:440–447. [PubMed: 25482033]
- Masse-Alarie H, Beaulieu LD, Preuss R, Schneider C. Corticomotor control of lumbar multifidus muscles is impaired in chronic low back pain: concurrent evidence from ultrasound imaging and double-pulse transcranial magnetic stimulation. *Exp Brain Res.* 2016a; 234:1033–1045. [PubMed: 26708518]
- Masse-Alarie H, Beaulieu LD, Preuss R, Schneider C. 2016bThe side of chronic low back pain matters: evidence from the primary motor cortex excitability and the postural adjustments of multifidi muscles. *Exp Brain Res.*
- Masse-Alarie H, Flamand VH, Moffet H, Schneider C. Corticomotor control of deep abdominal muscles in chronic low back pain and anticipatory postural adjustments. *Exp Brain Res.* 2012; 218:99–109. [PubMed: 22311467]
- Massion J. Movement, posture and equilibrium: interaction and coordination. *Prog Neurobiol.* 1992; 38:35–56. [PubMed: 1736324]
- Maughan EF, Lewis JS. Outcome measures in chronic low back pain. *Eur Spine J.* 2010; 19:1484–1494. [PubMed: 20397032]
- McIlroy WE, Maki BE. Preferred placement of the feet during quiet stance: development of a standardized foot placement for balance testing. *Clin Biomech (Bristol, Avon).* 1997; 12:66–70.
- Mehta R, Cannella M, Henry SM, Smith S, Giszter S, Silfies SP. Trunk Postural Muscle Timing Is Not Compromised In Low Back Pain Patients Clinically Diagnosed with Movement Coordination Impairments. *Motor Control.* 2016:1–37.
- Mehta R, Cannella M, Smith SS, Silfies SP. Altered trunk motor planning in patients with nonspecific low back pain. *J Mot Behav.* 2010; 42:135–144. [PubMed: 20207604]
- Mok NW, Brauer SG, Hodges PW. Postural recovery following voluntary arm movement is impaired in people with chronic low back pain. *Gait Posture.* 2011; 34:97–102. [PubMed: 21530268]
- O’Keeffe M, Purtill H, Kennedy N, Conneely M, Hurley J, O’Sullivan P, Dankaerts W, O’Sullivan K. Comparative Effectiveness of Conservative Interventions for Nonspecific Chronic Spinal Pain: Physical, Behavioral/Psychologically Informed, or Combined? A Systematic Review and Meta-Analysis. *J Pain.* 2016; 17:755–774. [PubMed: 26844416]
- Sadeghi M, Talebian S, Olyaei GR, Attarbashi Moghadam B. Preparatory brain activity and anticipatory postural adjustments accompanied by externally cued weighted-rapid arm rise task in non-specific chronic low back pain patients and healthy subjects. *Springerplus.* 2016; 5:674. [PubMed: 27350911]

- Saitou K, Washimi Y, Koike Y, Takahashi A, Kaneoke Y. Slow negative cortical potential preceding the onset of postural adjustment. *Electroencephalogr Clin Neurophysiol*. 1996; 98:449–455. [PubMed: 8763504]
- Saragiotto BT, Maher CG, Yamato TP, Costa LO, Menezes Costa LC, Ostelo RW, Macedo LG. Motor control exercise for chronic non-specific low-back pain. *Cochrane Database Syst Rev*. 2016; 1:CD012004.
- Schabrun SM, Elgueta-Cancino EL, Hodges PW. Smudging of the Motor Cortex is related to the Severity of Low Back Pain. *Spine (Phila Pa 1976)*. 2015
- Shibasaki H, Hallett M. What is the Bereitschaftspotential? *Clin Neurophysiol*. 2006; 117:2341–2356. [PubMed: 16876476]
- Silfies SP, Bhattacharya A, Biely S, Smith SS, Giszter S. Trunk control during standing reach: A dynamical system analysis of movement strategies in patients with mechanical low back pain. *Gait Posture*. 2009; 29:370–376. [PubMed: 19046882]
- Silfies SP, Mehta R, Smith SS, Karduna AR. Differences in feedforward trunk muscle activity in subgroups of patients with mechanical low back pain. *Arch Phys Med Rehabil*. 2009; 90:1159–1169. [PubMed: 19501348]
- Tsao H, Danneels LA, Hodges PW. ISSLS prize winner: Smudging the motor brain in young adults with recurrent low back pain. *Spine (Phila Pa 1976)*. 2011; 36:1721–1727. [PubMed: 21508892]
- Tsao H, Galea MP, Hodges PW. Reorganization of the motor cortex is associated with postural control deficits in recurrent low back pain. *Brain*. 2008; 131:2161–2171. [PubMed: 18669505]
- van Boxtel GJ, Brunia CH. Motor and non-motor aspects of slow brain potentials. *Biol Psychol*. 1994; 38:37–51. [PubMed: 7999929]
- Waddell G, Newton M, Henderson I, Somerville D, Main CJ. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain*. 1993; 52:157–168. [PubMed: 8455963]
- Walter W, Cooper R, Aldridge V, McCallum W, Winter A. Contingent negative variation: an electric sign of sensori-motor association and expectancy in the human brain. *Nature*. 1964; 203:380–384. [PubMed: 14197376]

HIGHLIGHTS

- Evaluated factors that influence the effect of low back pain (LBP) on posture
- People with and without LBP performed rapid arm raises
- postural adjustments (APAs) more delayed in cued than self-initiated conditions
- APA onset associated with reported disability and fear of physical activity
- Effects of LBP on APAs influenced by task condition and disability status

Fig. 1.

(A) Illustration of the arm-raise task while recording EEG (black circles), EMG (black ovals), and kinematics (gray circles). (B) Representative traces of average EMG waveforms for a subject with LBP (gray lines) and a subject without LBP (black lines), illustrating the APA onset of trunk muscles relative to deltoid onset (time = 0). (C) A subject's average EEG signal prior to first-muscle onset (contralateral erector spinae onset, time = 0) in the self-initiated condition (solid line) and cued condition (dashed line), illustrating the epoch (gray box) from which the average amplitudes of EEG pre-movement negativity were derived.

(A) Muscle Onset Latencies After Deltoid Onset **(B) Pre-Movement Negativity** **(C) Peak Arm-Raise Velocity** **Fig. 2.**

Group mean (95% confidence intervals) **(A)** APA onset latencies, **(B)** amplitudes of pre-movement negativity, and **(C)** peak arm-raise velocities in the self-initiated (self) and cued conditions. Gray lines and symbols represent the group with LBP; black lines and symbols represent the group without LBP. IO = internal oblique, EO = external oblique, ES = erector spinae, contra = contralateral to the arm raised, and ipsi = ipsilateral to the arm raised.

Fig. 3.

Scatter plots illustrating associations of APA onset latencies at the contralateral EO muscle with Modified Oswestry Disability scores, Numeric Pain Ratings, and FABQ scores. Filled circles represent subjects with LBP in the self-initiated condition; open circles, in the cued condition. The solid trend line represents that of the self-initiated condition; the dashed trend line, the cued condition.

Table 1

Group Characteristics

	Participant Group		Statistic (P-Value)
	With LBP	Without LBP	
Number (Female, Male)	13 (8, 5)	13 (9, 4)	Fisher's $\chi^2 = 0.70$ (P = 1.0)
Mean (95% CI) Age, yr	37 (31–43)	35 (29–40)	T = 0.62 (P = 0.54)
Mean (95% CI) Height, m	1.69 (1.63–1.75)	1.66 (1.61–1.72)	T-Test = 0.68 (P = 0.51)
Mean (95% CI) Weight, kg	65 (59–71)	68 (60–77)	T-Test = 0.61 (P = 0.55)
Mean (95% CI) Duration of LBP, yr	8.67 (4.23–13.10)	0 (0–0)	Mann Whitney Z = 4.64 (P < 0.0001)
Median (range) Numeric Pain Rating	2 (0–6)	0 (0–3)*	Mann Whitney Z = 2.66 (P = 0.016)
Median (range) Modified Oswestry Percent Score	12 (0–22)	0 (0–0)	Mann Whitney Z = 4.36 (P < 0.0001)
Median (range) Fear Avoidance Beliefs Questionnaire Physical Activity Score	14 (0–21)	0 (0–0)	Mann Whitney Z = 4.36 (P < 0.0001)
Median (range) Fear Avoidance Beliefs Questionnaire Work Score	9 (0–30)	0 (0–0)	Mann Whitney Z = 4.08 (P < 0.0001)

* One subject indicated lower-extremity soreness following exercise the previous day; the subject was allowed to participate and did not elicit outcomes that suggested outlier behavior.