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## Individuals with non-specific low back pain use a trunk stiffening strategy to maintain upright posture

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### Abstract

There is increasing evidence that individuals with non-specific low back pain (LBP) have altered movement coordination. However, the relationship of this neuromotor impairment to recurrent pain episodes is unknown. To assess coordination while minimizing the confounding influences of pain we characterized automatic postural responses to multi-directional support surface translations in individuals with a history of LBP who were not in an active episode of their pain. Twenty subjects with and 21 subjects without non-specific LBP stood on a platform that was translated unexpectedly in 12 directions. Net joint torques of the ankles, knees, hips and trunk in the frontal and sagittal planes as well as surface electromyographs of 12 lower leg and trunk muscles were compared across perturbation directions to determine if individuals with LBP responded using a trunk stiffening strategy. Individuals with LBP demonstrated reduced peak trunk torques, and enhanced activation of the trunk and ankle muscle responses following perturbations. These results suggest that individuals with LBP use a strategy of trunk stiffening achieved through co-activation of trunk musculature, aided by enhanced distal responses, to respond to unexpected support surface perturbations. Notably, these neuromotor alterations persisted between active pain periods and could represent either movement patterns that have developed in response to pain or could reflect underlying impairments that may contribute to recurrent episodes of LBP.

### Keywords

Postural control; Automatic postural response; Joint torque; Surface electromyography; Ankle strategy; Hip strategy

### Introduction

Individuals with low back pain (LBP) demonstrate altered movement patterns that reflect changes in neuromuscular control of both voluntary [Coghlin and McFadyen, 1994; Gioftos

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and Grieve, 1996; Hubley-Kozey and Vezina, 2002; Lamothe, et al., 2006b] and reactive movements [Henry, et al., 2006; Radebold, et al., 2000]. Whether these movement alterations developed following the initial pain episode or may have contributed to the development of pain is unknown. Quantification of movement patterns in this population may help to clarify the relationship between neuromuscular impairments and recurrent episodes of pain and thus lead to more efficacious treatment. However, studying a population with recurrent pain can be difficult given the confounding effects of pain on movement. Individuals with LBP may choose to move in a guarded manner in order to minimize forces applied to painful structures [McGill, 2002; Porterfield and Derosa, 1998] or in the anticipation of pain [Al-Obaidi, et al., 2003; Moseley, et al., 2004], thus obscuring or exaggerating the underlying neuromuscular impairments that may contribute to the development or persistence of LBP. In addition, individuals with LBP may choose to move at slower velocities [Al-Obaidi, et al., 2003; Lamothe, et al., 2006b; Simmonds, et al., 1998] or with reduced force production [Verbunt, et al., 2005], but are still able to achieve higher than preferred movement velocities when challenged [Lamothe, et al., 2006a; Lamothe, et al., 2006b; Lee, et al., 2007]. These confounding factors are of particular concern when trying to determine underlying neuromuscular impairments resulting from the LBP episode.

To minimize the influence of volitional effects on neuromuscular measures, a paradigm that is less susceptible to volition, such as the support surface perturbation, can be used. Perturbation of the support surface has been used to study automatic postural responses (APRs; for a review, see [Horak and Macpherson, 1996]), which are obligatory responses to unexpected balance disruption. APRs are robust and well-documented in many different subject populations and provide a systematic means of investigating postural control. Through the study of APRs, we can investigate alterations in motor control in people with LBP without confounds that accompany volitional movement.

We have previously reported on APRs in subjects with non-specific, recurrent LBP elicited by support surface translations [Henry, et al., 2006]. Following unexpected, multi-directional perturbations, these subjects, compared to subjects without LBP, demonstrated delayed centers of pressure and mass responses, increased center of mass displacement, and a reduced margin of stability (center of pressure minus center of mass), largely in the sagittal plane. These results suggest that individuals with LBP may use an alternate strategy to maintain balance when confronted with unexpected changing environmental conditions; however, this strategy may afford them less stability in the sagittal plane. Although previous studies have demonstrated that individuals with LBP reduce their reliance on a hip strategy in standing posture [NW Mok, et al., 2004], which may negatively impact stability, little study has been devoted to understanding the unique roles of the trunk and hip in responding to unexpected balance perturbations.

To determine whether postural strategies are altered in individuals with LBP, net joint torque and myoelectric responses to these perturbations must be quantified so we can better understand how LBP impacts motor responses without volitional interference. Therefore, we compared the APRs of subjects with and without non-specific, recurrent LBP when their support surface was unexpectedly translated. Trunk myoelectric responses and net joint torques computed at the trunk, hip, knee, and ankle were quantified as indicators of the corrective strategy used to maintain balance following unexpected perturbation. In order to reduce the direct influence of pain on the postural responses we chose to study a cohort of subjects reporting a history of non-specific LBP and not in an active pain episode at the time of testing. Given that people with LBP demonstrated a reduced margin of stability [Henry, et al., 2006] and may choose to move in a guarded manner, we predicted that individuals with a history of LBP would demonstrate altered APRs that reflect stiffening at the trunk, as evidenced by increased trunk muscle co-activation and decreased trunk torque.

## Methods

### Subjects

Twenty subjects with non-specific, recurrent [Von Korff, 1994] LBP and 21 subjects without LBP (Table 1), who were recruited from the local community through posted advertisements, participated in this study. Subjects were included if they were between 21 – 55 years of age and able to stand and walk without assistance. Subjects with LBP were included if they had a history of non-specific LBP with or without recurrences for at least 12 months. Subjects were excluded if they had neurological disease or balance disorders, uncorrected vision problems, systemic infection, current pregnancy, cardiovascular disorders, severe musculoskeletal deformity (scoliosis or kyphosis), injury to the lower extremity that would interfere with testing or history of any surgery in the 3 months prior to testing. Control subjects were excluded if they had back pain during the prior 12 months that required medical attention or resulted in missed work. Subjects with LBP were also excluded if they had pain below the knee consistent with a disc herniation, presence of neurological signs, serious spinal complications (e.g., vertebral fracture, tumor or infection), spinal stenosis, previous spinal surgery, were receiving workers' or disability compensation or were in litigation due to their LBP. Subjects with LBP were tested when they were not in a recurrence of their LBP [McGorry, et al., 2000; Von Korff, 1994] Subjects with LBP demonstrated greater pain and disability than those without LBP as measured by the McGill Pain Questionnaire [Melzack, 1987], NPRS [Stratford and Spadoni, 2001] and Roland Morris Disability Questionnaire [Roland and Morris, 1983] ( $P < 0.01$ , Table 1). All subjects were currently employed at the time of testing or participating fully in their usual role. All subjects signed an informed consent document in accordance with University of Vermont Institutional Review Board policy.

### Equipment

A detailed description of the methods used in this study have been published previously [Jones, et al., 2008]. To summarize, two force plates (AMTI, Watertown, MA, USA) were mounted within the moveable platform that was driven by electromechanical motors (Compumotor, Parker Hannifin Corp., Rohnert Park, CA, USA). A 3-camera, passive marker system (BTS, Milan, Italy) was used to collect 3-dimensional body kinematic data (sampled at 50 Hz, dual pass 2<sup>nd</sup> order Butterworth low pass filter cut-off frequencies ranged from 2–5 Hz). An eight-segment, rigid link model was constructed for each subject [Zatsiorsky and Seluyanov, 1983], representing the feet, shanks and thighs bilaterally as well as the pelvis and trunk segments. Kinematics and force data were used to compute ankle, knee (sagittal only), hip and trunk (relative to pelvis) net joint torques in the frontal and sagittal planes (SD/Fast, Needham, MA, USA) through inverse dynamics techniques.

Surface electromyographs (EMG) of the left lower limb and the dorsal and ventral trunk, bilaterally, were recorded (BTS, Milan, Italy) using silver-silver chloride surface electrodes (Norotrode 20 bipolar, fixed 2 cm inter-electrode distance, Myotronics, Kent, WA, USA) placed over the Tibialis Anterior (TA) over the muscle belly approximately 2.5 cm lateral to the tibia, Medial Gastrocnemius (GA) over the most prominent part of the muscle belly oriented rostral-caudal, Rectus Abdominus (LRA and RRA corresponding to left and right RA, respectively) 3 cm lateral to the umbilicus oriented rostral-caudal, Internal Oblique (LIO, RIO) 2.5 cm medial and rostral to the anterior-superior iliac spine, rotated 45° toward the midline, External Oblique (LEO, REO) equidistant between the iliac crest and the lower ribs along the mid-axillary line, rotated posteriorly 45°, Erector Spinae at the 3<sup>rd</sup> lumbar segment (LES3, RES3), and 1<sup>st</sup> lumbar segment (LES1, RES1) 2.5 cm lateral to the lumbar segments oriented rostral-caudal. Skin was shaved and cleaned with alcohol prior to electrode placement. EMG signals were sampled at 1000 Hz in synchrony with the force

plate signals, amplified (2000–10000x), full-wave rectified and band-pass filtered from 35–200 Hz.

## Protocol

Subjects were instructed to stand, a foot on each of two force plates, at self-selected stance width and toe-out angle with arms hanging comfortably at their sides while looking forward. Subjects were given 2–3 practice trials in two translation directions (forward and leftward) prior to which they were told the direction of impending platform movement. Following practice trials, linear translations of the support surface in the transverse plane were randomly presented in 12 directions of 30° increments (Figure 1) with three trials in each direction (velocity 43 cm/s; peak acceleration 127 cm/s<sup>2</sup>). Any trials in which the subjects stepped were discarded and repeated randomly at a later point in the protocol.

## Data Processing

Peak torque magnitudes (stature and weight normalized) and latencies to peak torque in response to the surface translations were computed for each joint to characterize the torque responses used to restore balance. The absolute values of the peak torque magnitudes were computed to determine if the torque magnitude varied with perturbation direction, regardless of whether the torque polarity was negative or positive.

EMG integrals were computed for each muscle using the full-wave rectified, filtered EMG signals across three epochs: baseline (–250 to –50 ms relative to perturbation onset) and two 75 ms epochs spanning from 100–250ms following perturbation onset. Each epoch was divided by its duration to obtain an average value for each epoch that was then normalized to the maximum value across all perturbation directions and across the 3 epochs for each muscle, by subject; thus, the epoch with the largest integral, regardless of timing or direction was set equal to 100%.

## Data Analysis

Anthropometric measures (age, stature, body mass, BMI) were compared using independent samples t-tests, while the proportion of males vs. females was compared using a Chi-square test of independence. Measures of pain and disability were compared using a Wilcoxon Ranked Sum Test.

Responses to the perturbations were analyzed using a repeated measures analysis of variance for each joint torque and EMG epoch with perturbation direction as the repeated factor and LBP vs. NLBP as the grouping factor (SAS System for Windows, Cary, NC, USA). For sagittal plane torques, statistical comparisons of torques included only perturbation directions of 60°, 90°, 120°, 240°, 270° and 300°, and for frontal plane torques, comparisons included directions of 0°, 30°, 150°, 180°, 210°, 330° (Figure 1), while all 12 perturbation directions were compared for EMG variables. An alpha level of  $P \leq 0.05$  was considered statistically significant for main effect comparisons.

## Results

Following multi-directional platform perturbations, individuals with LBP modulated their torque responses with perturbation direction similar to individuals without LBP. However, in general, individuals with LBP reduced their trunk torque amplitude and used a greater proportion of their maximal response activation for trunk and ankle muscles.

## Peak Torque Responses

Individuals with and without LBP modulated their peak torque magnitudes with perturbation direction similarly at all joints in the sagittal and frontal planes (sagittal plane peak trunk torque,  $P=0.02$ ; all other joints,  $P<0.01$ ). However, individuals with LBP tended to reduce sagittal plane peak trunk torque amplitudes following forward/backward perturbations ( $P=0.09$ ; Figure 2a). In the frontal plane, individuals with LBP reduced peak trunk torque amplitudes across all perturbation directions ( $P=0.03$ ; Figure 2a). In contrast to the trunk torque response, the lower extremity peak torque magnitudes did not differ between groups (Figure 2b-d).

Individuals with LBP also demonstrated shorter latencies to right peak hip torque ( $P=0.024$ ) with a similar trend in the left hip ( $P=0.08$ ), in both extension and flexion, compared to healthy individuals. There were no group differences in either the frontal or sagittal planes for the latency to peak torques for all other lower extremity and trunk torques ( $P$ -values ranged from 0.11 to 0.86).

## Muscle Activation Patterns

Baseline muscle activation patterns for both groups did not vary across directions of the impending perturbation, as expected, given that the perturbations were randomly presented ( $P=0.99$ ). However, individuals with LBP used a greater proportion of their maximal response activation for their trunk and leg muscles compared to individuals without LBP ( $P<0.01$ ) at baseline.

In contrast, during the 100–175 ms and 175–250 ms epochs representing the APRs elicited by translations, subjects' responses differed based on both group and direction effects. During the 100–175 ms epoch, both groups varied the amplitude of muscle activation patterns for all proximal (Figure 3) and distal (Figure 4) muscles with perturbation direction ( $P$ -values ranged from  $<0.01$  to 0.03), such that one group of perturbation directions demonstrated a greater amplitude of muscle activation and the opposing perturbation directions demonstrated a lesser amplitude of activation. For example, muscle activation amplitudes of the ES at L1 were greater in response to perturbations with a forward component (i.e. 60°, 90°, 120°) with reduced activation amplitudes following perturbations with a backward component (i.e. 240°, 270°, 300°). This pattern was not evident at the RA muscles (left,  $P=0.52$ ; right,  $P=0.20$ ) given that the muscle activation amplitudes did not vary with direction. Throughout this epoch, individuals with LBP demonstrated a greater proportion of their maximal response activation for all trunk and leg muscles ( $P$ -values ranged from  $<0.01$  to 0.02) with the exception of the right IO ( $P=0.29$ ). In contrast to trunk muscle activation which was elevated across perturbation directions for both dorsal and ventral muscles, TA and GA muscles were elevated in opposing perturbation directions (e.g. TA was elevated in directions with a forward perturbation component, GA was elevated in directions with a backward perturbation component).

During the 175–250 ms epoch, both groups varied the amplitude of all muscles with perturbation direction, in a similar sinusoidal fashion ( $P$ -values ranged from  $<0.01$  to 0.04) including the RA muscles bilaterally; however, subjects did not vary the amplitude of the right IO muscle ( $P=0.27$ ) with perturbation direction. Individuals with LBP continued to demonstrate a greater proportion of their maximal response activations for the EO bilaterally (left,  $P<0.01$ ; right,  $P=0.04$ ), the left TA ( $P=0.04$ ), ES at L1 bilaterally (left,  $P=0.03$ ; right,  $P=0.05$ ), and the left IO and RA muscles ( $P<0.01$  and  $P<0.01$  and, respectively), with the right RA trending toward significance ( $P=0.08$ ). There were no group differences in the proportion of the maximal response activation for the left GA ( $P=0.24$ ), the ES at L3, bilaterally (left,  $P=0.06$ ; right,  $P=0.35$ ), or the right IO ( $P=0.24$ ) muscles.

## Discussion

The results support our hypothesis that people with a history of non-specific LBP would demonstrate altered APR strategies that reflect stiffening at the trunk, as evidenced by decreased trunk torque amplitudes and increased co-activation of the trunk musculature. In addition, individuals with LBP demonstrated altered APRs distal to the site of pain indicated by enhanced muscle responses at the ankle (TA and GA muscles).

### Individuals with LBP demonstrate a trunk stiffening strategy

In response to multi-directional surface translations, individuals with a history of non-specific LBP demonstrated peak torque responses across lower extremity joints that were similar in pattern and in magnitude to those of healthy controls (Figure 2b-d) [Allum and Honegger, 1992; Jones, et al., 2008; Meyer, et al., 2004; Park, et al., 2004; Runge, et al., 1999]. However, individuals with LBP demonstrated reduced sagittal and frontal plane peak trunk torques (Figure 2a), and shorter latencies to peak hip torque, as well as increased co-contraction of the trunk musculature (Figure 3), compared to those of the control group. These results suggest that individuals with LBP respond to perturbations by utilizing a preparatory strategy of trunk stiffening, achieved through a greater baseline level of co-activation of agonist-antagonistic trunk muscles that is maintained throughout the early phases of the postural response. In addition, these individuals may use the hip joint to a greater extent (as indicated by earlier peak torque production) either as compensation for a trunk that is already constrained by muscle co-activation, or as a strategy to reduce the need for a perturbation-elicited trunk response, given the reluctance of these individuals to move the lumbar spine [NW Mok, et al., 2004; Shum, et al., 2005]. Previous studies have demonstrated increased levels of baseline [Stokes, et al., 2006] and perturbation-induced [Cholewicki, et al., 2005; Radebold, et al., 2000] co-activation of the trunk musculature, suggesting that individuals with LBP use a default strategy that aims to restrict trunk movement.

Previous investigations of perturbation-induced postural responses have reported that individuals with LBP reduce their reliance on a hip strategy when maintaining balance in unstable standing conditions [NW Mok, et al., 2004]. The reduced trunk torque magnitudes characteristic of the postural responses by individuals with LBP in the current study support this interpretation and, given the enhanced ankle muscle responses demonstrated by this cohort, may reflect a shift to a more ankle-driven response strategy. These results are consistent with our previous study [Henry, et al., 2006] in which people with LBP demonstrated reduced and delayed center of pressure responses with concomitant increased center-of-mass displacements, consistent with the use of an ankle strategy. This shift in strategy selection may reflect the reluctance of individuals with LBP to use a hip strategy [NW Mok, et al., 2004; N. W. Mok, et al., 2007] or to employ large movements or accelerations of the trunk [Thomas and France, 2007] that may result from the anticipation [Moseley, et al., 2004], or fear [Thomas and France, 2007], of impending pain. Unfortunately reliance on the ankle strategy may reduce postural stability, demonstrated by the reduced margin of stability elicited in response to unexpected perturbations in this population [Henry, et al., 2006], which may signal a reduction in robustness of the postural control system [Claeys, et al., 2011].

### Postural strategy selection by individuals with LBP may reflect altered central set

Individuals with LBP respond to unexpected translation of the support surface using a trunk stiffening strategy, mediated through co-activation of the trunk musculature that may require less specific knowledge of the nature of the postural perturbation. Alteration of the corrective strategy could be mediated through changes in central set, defined as “a central

preparatory state within the nervous system related to higher-level task-related intentions and expectations” that can influence postural responses, both expected and unexpected [Cacciatore, et al., 2005]. Horak, Diener and Nashner [Horak, et al., 1989] reported a “non-specific enhanced [postural] response” with unexpected perturbations of balance suggesting that healthy individuals set a higher default response magnitude in the absence of accurate information about the perturbation. Given the proprioceptive impairment demonstrated by individuals with LBP [S Brumagne, et al., 2000; S Brumagne, et al., 2004; Descarreaux, et al., 2005; Ginanneschi, et al., 2007; Newcomer, et al., 2000], it is plausible that these individuals may increase the gain of their postural responses irrespective of perturbation direction in order to compensate for a lack of accurate information and to avoid potentially injurious losses of balance. In addition, individuals with LBP may have a higher-level task-related intention to restrict lumbar movement, perhaps due to fear of movement or pain secondary to chronic LBP [Thomas and France, 2007; Thomas and France, 2008], which could influence the implementation of this corrective torque strategy.

Additional evidence for a central contribution (as opposed to solely a peripheral contribution, localized to the site of pain) is that individuals with LBP in the current study demonstrated heightened postural responses at the ankle for both the TA and GA muscles. Given that these alterations occur distal to the site of lumbar pain, it is likely that central set plays a role in shifting the postural responses to a more ankle-driven response. This distal compensation has been demonstrated by individuals with LBP through an increased reliance on distal proprioceptive feedback to maintain standing posture [S Brumagne, et al., 2004; S. Brumagne, et al., 2008; Claeys, et al., 2011], whereas a reverse phenomenon was observed in individuals with induced plantar sensation loss who compensated by increasing reliance on proximal joints to respond to postural perturbations [Meyer, et al., 2004]. The mutability of these postural responses suggests that regional alterations directly related to lumbar pain are unlikely to be the only explanation for the altered strategies used by individuals with LBP.

## Conclusions

In conclusion, our findings demonstrate that altered postural strategies for maintenance of posture persist following balance disturbances in a population of individuals with a history of LBP, who were not in an active pain episode. Individuals with LBP exhibit altered response patterns characterized by reduced peak trunk torque, increased co-activation of proximal musculature and enhanced distal muscle activation could reflect changes in central set. Although it is unknown whether these altered patterns contributed to the initial onset of LBP or are related to the lasting effects of the initial pain episode and underlying pathology, these results suggest that altered movement patterns persist even in the absence of an active pain episode and may be linked to recurrent episodes of LBP. Therapeutic interventions that address the underlying motor control impairments responsible for these altered movement patterns may contribute to the reduction of the probability, duration, and/or severity of recurrent episodes of pain.

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## Biographies

Stephanie Jones is currently a Postdoctoral Research Associate in the Motor Control Laboratory at the University of Massachusetts Amherst. She earned her Ph.D. in Rehabilitation Science from McGill University, her M.S. in Exercise Science from the University of Massachusetts Amherst and her B.Sc. (Honours) in Human Kinetics from the University of Guelph. Her primary research interests include the sensory and proprioceptive contributions to movement coordination in individuals with chronic pain and neurological disorders.



Sharon Henry, PT, PhD is a professor of physical therapy in the Department of Rehabilitation and Movement Science at the University of Vermont. Her primary research interests are motor control of human posture and movement, particularly as they relate to musculoskeletal impairments and injuries. Her recent work has focused on the examination the motor control strategies that are used by people with low back pain and on quantifying clinical outcomes and neuromuscular changes following different exercise based interventions in this patient population.



Dr. Christine C. Raasch is a Principal at Exponent, specializing in the biomechanical analysis of human injury, occupant protection systems, injury criteria, and accident reconstruction. Her work includes analysis of traumatic injury mechanisms, computer simulation of occupant motions, full-scale vehicle crash and sled testing using anthropomorphic test dummies, and specialized biomechanical studies such as helmet impact testing. Her research has focused on analysis of locomotion and reaching movements, and impaired control of movement after head injury or stroke utilizing complex computer models of muscle and skeletal dynamics, optimization techniques, and experiments involving motion analysis and electromyography.

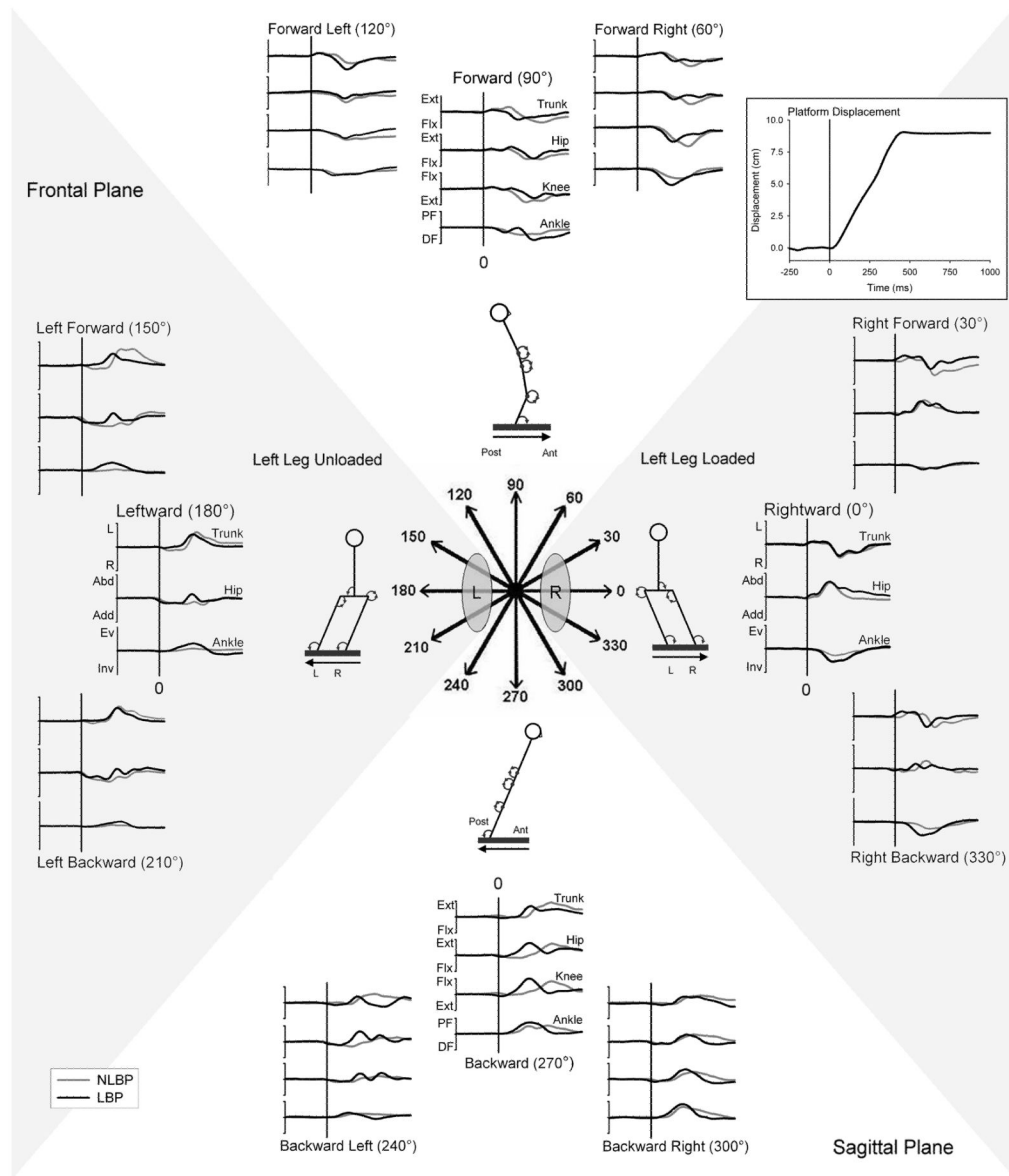


Juvena R. Hitt received a BS degree in physics from Loyola University Maryland in 1995. She is currently a senior research technician in the Department of Rehabilitation and Movement Science at the University of Vermont. She has numerous years of research experience in the areas of biomechanics and biomedical engineering in both academic and industrial settings. Her current work has focused on the biomechanics of postural control in the low back pain patient population.



Dr. Janice Y. Bunn has been an Associate Research Professor at the University of Vermont since 2000, with appointments in the Departments of Medical Biostatistics and Rehabilitation and Movement Sciences. Her continued interest is in the areas of research study design and statistical analysis as a means of improving the integrity of health-related research studies.

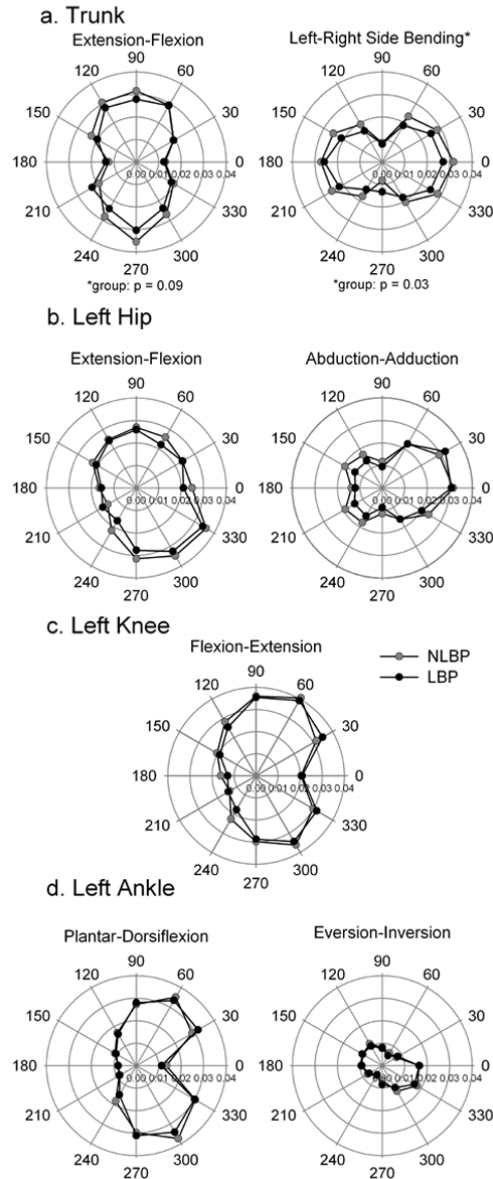




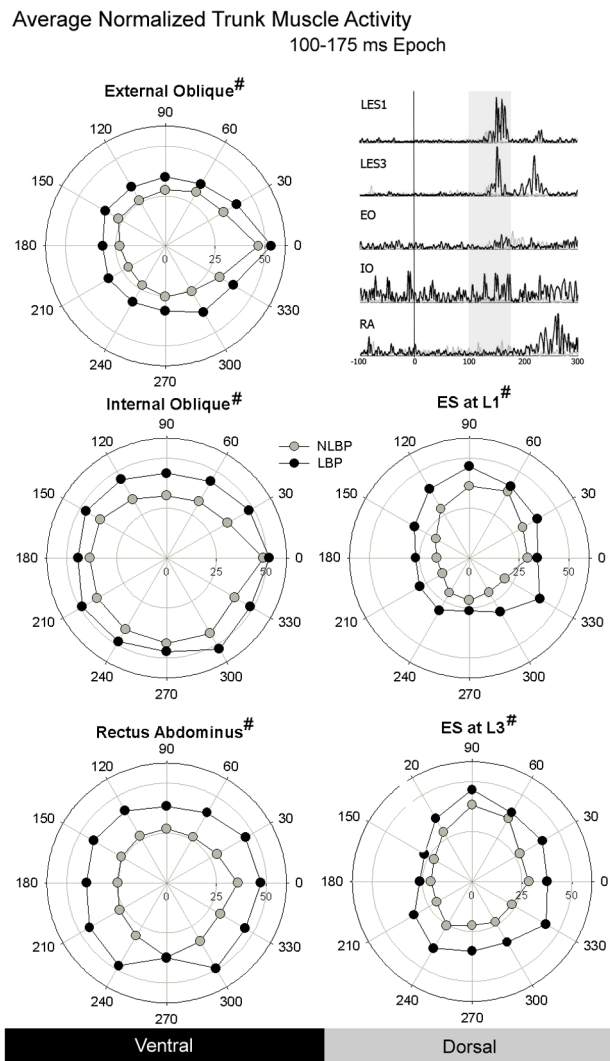
**Figure 1.**

Time series of joint torque responses to perturbations across all perturbation directions for two exemplar subjects; an individual with LBP (black) and an individual without a history of LBP (gray). Sagittal plane torques of the trunk and left hip, knee and ankle are presented in response to predominately forward or backward perturbations (unshaded). Frontal plane torques of the trunk, and left hip and ankle are presented in response to primarily lateral perturbations (shaded). Traces range from 250 ms prior to platform perturbation to 1000 ms post-perturbation onset on the horizontal axis (vertical line represents the onset of platform movement) and  $\pm 50$  Nm on the vertical axis. Schematic stick figures (depicted with the figure facing to the right for the sagittal plane views and viewed from the back for the frontal plane views) demonstrate the resulting direction of body sway and the dominant torque demonstrated at each joint for each of the cardinal directions. The boxed inset contains a displacement (cm) vs. time (ms) trace for platform movement during a rightward (0°) perturbation, as determined by the force plate kinematic markers.

Polar Plots of Normalized Peak Torque Magnitudes



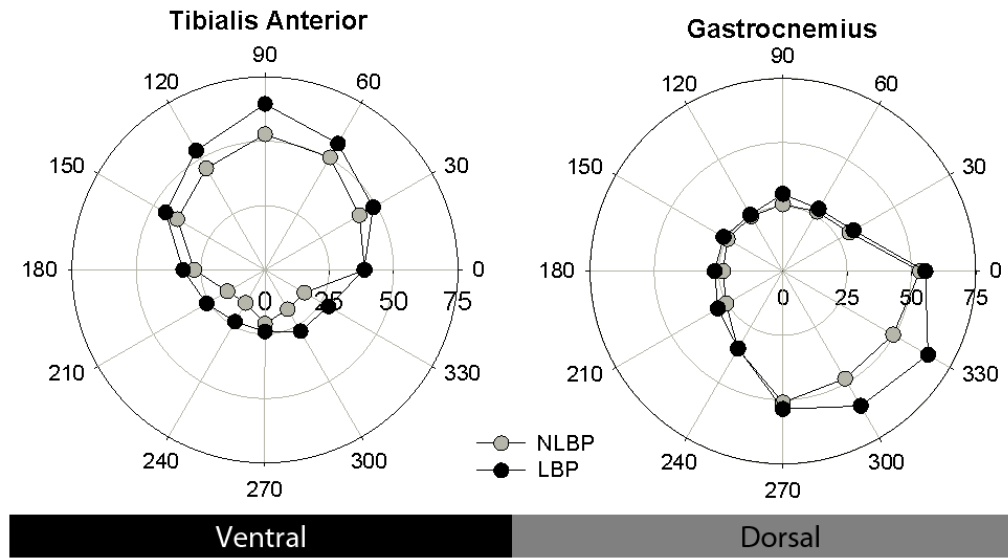
**Figure 2.** Peak joint torque responses in the sagittal and frontal planes at the trunk (a), and left lower limb (b, hip; c, knee, and d, ankle). Polar plots depict group means of the absolute values of normalized peak torque magnitudes comparing individuals with LBP (black circles) and without LBP (NLBP; gray circles). All joints demonstrated significant main effects of direction ( $P < 0.01$ ). Trends or significant main effects for group differences (\*) are indicated by superscripts.



**Figure 3.** Average normalized trunk muscle activity for the 100–175 ms epoch following platform perturbations. Polar plots depict group means of the muscles of the left ventral (left panel) and left dorsal (right panel) trunk comparing individuals with LBP (black circles) and without LBP (NLBP; gray circles). Significant group effects ( $P < 0.05$ ) are denoted by #. Inset depicts normalized, rectified, filtered EMG traces for two exemplar participants (LBP, black; NLBP, gray) in response to a forward ( $90^\circ$ ) perturbation; shaded area corresponds to 100–175 ms epoch of interest.

### Average Normalized Ankle Muscle Activity

100-175ms Epoch



**Figure 4.** Average normalized Tibialis Anterior and Gastrocnemius muscle activity for the 100–175 ms epoch following platform perturbations. Polar plots depict group means comparing individuals with LBP (black circles) and without LBP (NLBP; gray circles). Both muscles demonstrated significant direction and group effects ( $P < 0.05$ ).



**Table 1**

## Subject Characteristics

Parameter [Mean ( SD)]	LBP (n = 20)	NLBP (n = 21)	P-value
Age (years)	39.9 (14.5)	33 (10.5)	0.09
Stature (m)	1.73 (0.14)	1.71 (0.08)	0.56
Body Mass (kg)	75.9 (16.8)	68.1 (12.9)	0.10
BMI (kg/m <sup>2</sup> )	25.2 (3.2)	23.2 (3.4)	0.07
Gender (# Male/# Female)	9/11	8/13	0.65
McGill Pain Questionnaire (# of Words Circled)	3.45 (2.78)	0.05 (0.22)	<0.01
Numeric Pain Rating Scale (/10)	2.00 (1.37)	0.19 (0.40)	<0.01
Roland Morris Disability Questionnaire (/18)	2.35 (2.30)	0 (0)	<0.01