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ABSTRACT

Introduction. Different back muscle reflex assessment protocols have shown abnormally longer reflex latency responses of back muscles in chronic low back pain (CLBP). However, many confounding variables are difficult to control, such as the load magnitude and the preactivation of trunk muscles. The aims of this study were to evaluate, in 30 subjects with chronic low back pain (CLBP) and 30 healthy controls, the activation levels of back muscles during preloading and their reflex responses to sudden loading. Methods. After subjected to six practice perturbations, 20 sudden and unexpected forward perturbations of the trunk were applied in 30 CLBP subjects (14 women) and 31 controls (17 women), while attempting to minimize the confounding effect of preactivation level and perturbation amplitude. Reflex latency and amplitudes were computed from the surface EMG signals of four back muscles (bilaterally at L5, L3, L1, T10 vertebral levels). EMG was also collected from abdominal muscles. Results. Subjects with CLBP significantly increased the preactivation of back muscles (abdominal preactivation the same) relative to controls while no sex effect was observed. While adjusting statistically for these differences, reflex amplitude was significantly higher in subjects with CLBP and men, compared to healthy controls and women, respectively. Discussion. Interestingly, contrary to most of the literature available, no between-group effects were detected for reflex latency, which could potentially be explained by an appropriate control of confounding variables, but this remains to be clarified in future research.

Keywords: reflex latency, reflex amplitude, sudden load, lumbar impairment, muscle preactivation

INTRODUCTION

Various neuromuscular (motor-control) functions have been shown to be abnormal in subjects with CLBP, such as feedforward and feedback mechanisms, proprioception, psychomotor reaction time, balance and finally, muscle coordination patterns (Ebenbichler et al., 2001; Hodges and Moseley, 2003; van Dieen et al., 2003). The present study focuses on a feedback mechanism, namely the reflex contraction of back muscles in response to forward trunk displacement.

Delayed back muscle reflex latencies have been shown in subjects with CLBP, in response to sudden unexpected perturbations (Magnusson et al., 1996; Radebold et al., 2000; Reeves et al., 2005), whereas one study showed no difference (Leinonen et al., 2001). However, to date, various protocols have been proposed to elicit back muscle reflex responses, using a sudden loading (Leinonen, Kankaanpaa, Luukkonen, Hanninen, Airaksinen, and Taimela, 2001; Magnusson, Aleksiev, Wilder, Pope, Spratt, Lee, Goel, and Weinstein, 1996) or unloading paradigm (Radebold, Cholewicki, Panjabi, and Patel, 2000; Reeves, Cholewicki, and Milner, 2005), with the transmission of the load through the hands (Leinonen, Kankaanpaa, Luukkonen, Hanninen, Airaksinen, and Taimela, 2001; Magnusson, Aleksiev, Wilder, Pope, Spratt, Lee, Goel, and Weinstein, 1996) or directly on the trunk (Radebold, Cholewicki, Panjabi, and Patel, 2000; Reeves, Cholewicki, and Milner, 2005), and with the lower limbs and pelvis more
(Radebold, Cholewicki, Panjabi, and Patel, 2000; Reeves, Cholewicki, and Milner, 2005) or less
(Leinonen, Kankaanpaa, Luukkonen, Hanninen, Airaksinen, and Taimela, 2001; Magnusson,
Aleksiev, Wilder, Pope, Spratt, Lee, Goel, and Weinstein, 1996) stabilized. In fact, these different
perturbation conditions lead to more or less control on different potential sources of variation of
the reflex responses. Furthermore, the transmission of the load through the hands or directly on
the trunk might involve completely different reflex mechanisms. To date, between-group
comparisons (CLBP vs controls) have not been performed with a protocol involving a sudden
load paradigm where the load is applied directly on the trunk and where the lower limbs and
pelvis are well stabilized.

Other concerns are of major importance when eliciting reflex responses. Ideally, the magnitude of
the perturbation should be so that changes in the length and velocity of the tested muscle, namely
the variables influencing the sensory receptors mediating the reflex response, are standardized
across individuals. Contrary to what has been done so far, this would require adjusting the load
according to the trunk mass. For instance, sex differences have been obtained using a load
transmitted through the hands (Wilder et al., 1996) or applied directly on the trunk (Granata and
Rogers, 2007; Rogers and Granata, 2006), but the load was not adjusted according to the trunk
mass. Whether sex differences would remain while standardizing the trunk perturbation
parameters is unknown. The preactivation level of the trunk muscles should also be comparable
between subjects because increasing the preactivation of trunk muscles increases spinal stiffness
(Vera-Garcia et al., 2006) and consequently decreases trunk excursion (Vera-Garcia, Brown,
Gray, and McGill, 2006), decreases the frequency and amplitude of reflex responses (Stokes et
al., 2000; Vera-Garcia, Brown, Gray, and McGill, 2006) and delays reflex responses (Vera-
Garcia, Brown, Gray, and McGill, 2006). Knowing that at least some subjects with CLBP
increase the cocontraction of their trunk muscles (van Dieen, Selen, and Cholewicki, 2003) and
that the threat (fear) of pain alone can increase the cocontraction of trunk muscles (Moseley et
al., 2004; Moseley and Hodges, 2005), the control of this potential confounding variable appears
mandatory. Substantiating the trunk muscle activation level requires an adequate method to
normalize EMG amplitude values, which ideally requires a maximal EMG reference value
obtained during a maximal voluntary contraction (MVC). Unfortunately, MVCs may be biased
by pain-related fears in subjects with CLBP, which generates lower “maximal” EMG reference
values (Thomas et al., 2008). Submaximal EMG amplitude normalization protocols have to be
validated to circumvent this problem.

A final concern is related to the use of the traditional computerized procedure (standard deviation
– SD – method) to detect the electromyographic (EMG) onsets that generally lead to longer
latency estimates when higher levels of background muscle activity are present (Allison,
2003; Staude, 2001). Indeed, this could potentially bias the results from subjects with CLBP
showing more trunk muscle cocontraction. Fortunately, a relatively new computerized detection
method (Staude, 2001) has been shown to be unaffected by significant background activity (Lee
et al., 2007). However, this has not been tested in subjects with CLBP.
In the present study, a sudden-load paradigm where the load is transmitted directly through the trunk (forward perturbation) was used to elicit a reflex response of the back muscles in healthy controls and in subjects with CLBP. Experimental procedures have been implemented to attempt to minimize the different sources of bias identified above, and several trials \((n = 20\) perturbations) were performed to reduce the effect of random variations on the reflex responses. The main objective of this study was to compare trunk muscle preactivation and back muscle reflex responses between subjects with CLBP and healthy controls as well as between sexes. Two secondary objectives were also to assess whether the method to detect EMG onsets influences the reflex results and whether clinical or pain-related psychological variables (disability, pain intensity, fear of movement or re-injury, catastrophizing) are related to variations in the preactivation and reflex results.

**MATERIALS AND METHODS**

**Subjects**

Thirty subjects with CLBP (15 women) and 30 healthy controls (15 women) aged between 18 and 55 and having a body mass index (BMI) less than 31.5 kg\(^2\)/m\(^2\) (women) or 33 kg\(^2\)/m\(^2\) (men) were recruited (Table 1). However, four subjects (1 healthy woman, 1 CLBP man, 2 CLBP women) were excluded from the analyses due to technical problems. Inclusion criteria for the subjects with CLBP were as follows: lumbar or lumbosacral pain with or without proximal radicular pain (limited distally at the knees); and presence of chronic pain defined as a daily or almost daily pain for at least three months. Exclusion criteria for the healthy subjects were as follows: back pain in the preceding year; surgery of the pelvis or spinal column; pregnancy. The CLBP were recruited through the Montreal Rehabilitation Institute and newspaper ads in Montreal, Quebec. Exclusion criteria for the subjects with CLBP were as follows: surgery of the pelvis or spinal column; scoliosis; systemic or degenerative disease; one positive response to the Physical Activity Readiness Questionnaire (Thomas et al., 1992); history of neurological diseases or deficits not related to back pain (e.g., stroke, peripheral neuropathies, balance deficits); anticonvulsive, antidepressive and anxiolytic medication (use of antispasmodic, anti-inflammatory and analgesic drugs for back pain were accepted); pregnancy; claustrophobia. All subjects were informed about the experimental protocol and its potential risks and gave written consent prior to their participation. The ethics committee of the Centre for Interdisciplinary Research in Rehabilitation of Greater Montreal (CRIR) approved the study and consent form.

**Procedures**

Each experimental session comprised the following: (1) subject questionnaires (details below), (2) anthropometric measures to document the general characteristics of the subjects and to determine the parameters (pre-load and sudden load) of the reflex test, (3) surface electrode positioning, (4) submaximal contractions to obtain reference EMG values (EMG amplitude normalization), (5) back muscle reflex testing protocol, (6) 30-min. rest, (7) isometric maximal voluntary contractions (MVC) in a dynamometer to measure back muscle extension strength, (8)
Results corresponding to the 9th step (fatigue test) will be presented elsewhere. The data from step 7 (back muscle extension strength) were used to normalize the back muscle EMG preactivation values using the maximal EMG amplitude obtained during these MVCs. This also represents an opportunity to compare back muscle preactivation results using both methods (using submaximal or maximal contractions) to normalize EMG amplitude values, which could help to validate the use of submaximal contractions. This comparison was not planned at the beginning of the study, which explains why maximal EMG activation of abdominals was not collected.

Table 1. Demographic, anthropometric and clinical characteristics of the participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>Healthy controls</th>
<th>CLBP subjects</th>
<th>P values *</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Men (n = 15)</td>
<td>Women (n = 14)</td>
<td></td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>38 (10)</td>
<td>39 (10)</td>
<td></td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.74 (0.07)</td>
<td>1.65 (0.09)</td>
<td></td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>78 (9)</td>
<td>63 (7)</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>26 (3)</td>
<td>23 (3)</td>
<td></td>
</tr>
<tr>
<td>Phys. activity level</td>
<td>8.5 (0.9)</td>
<td>8.0 (1.9)</td>
<td></td>
</tr>
<tr>
<td>Strength (Nm)</td>
<td>303 (36)</td>
<td>198 (64)</td>
<td></td>
</tr>
<tr>
<td>VAS pain (cm)</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>RDQ (%)</td>
<td>30 (18)</td>
<td>25 (24)</td>
<td></td>
</tr>
<tr>
<td>TSK (score/68)</td>
<td>48 (8)</td>
<td>37 (10)</td>
<td></td>
</tr>
<tr>
<td>PCS (score/52)</td>
<td>26 (10)</td>
<td>15 (13)</td>
<td></td>
</tr>
<tr>
<td>Pre-load (Nm)</td>
<td>16 (2)</td>
<td>12 (2)</td>
<td></td>
</tr>
<tr>
<td>Sudden-load (Nm)</td>
<td>40 (6)</td>
<td>29 (5)</td>
<td></td>
</tr>
</tbody>
</table>

* Probability values corresponding to the two-way ANOVAs used the differences between healthy and subjects with CLBP (PAIN-STATUS factor) and between sexes. T-tests were performed for the clinical variables (VAS, RDQ, TSK, PCS). BMI: Body Mass Index; VAS: Visual Analog Scale of pain intensity; RDQ: Rolland-Morris Disability Questionnaire; TSK: Tampa Scale of Kinesiophobia; PCS: Pain Catastrophizing Scale

**Questionnaires**

First, the physical activity level was assessed in all subjects (Baecke et al., 1982). Then, four clinical and pain-related psychological variables were assessed in subjects with CLBP: (1) pain intensity with a visual analog scale (VAS), (2) perception of disability with the Rolland-Morris disability questionnaire (RDQ) (Roland and Morris, 1983), (3) fear of movement or injury with the Tampa scale of kinesiophobia (TSK) (French et al., 2002), and (4) pain catastrophizing with the Pain catastrophizing scale (PCS) (Sullivan et al., 1995).

**Surface electromyography**

The EMG signals from four pairs of back muscles and two pairs of abdominal muscles were collected (bandpass filter: 20-450 Hz; preamplification gain: 1000; sampling rate: 1024 Hz) with active surface electrodes (Delsys Inc., MA). After the skin at the electrode sites was shaved and abraded with alcohol, the electrodes were positioned bilaterally over the multifidus at the L5 level (≈ 3 cm from the midline of the back), iliocostalis lumborum at L3 (≈ 5-6 cm from midline) and longissimus at L1 (3 cm from midline) following recommendations with regard to muscle fiber direction (Lariviere et al., 2001a). Two additional electrodes were positioned over the belly of
the longissimus at the T10 level (≈ 4-5 cm from midline). Electrodes on the abdominal muscles (rectus abdominis: RA; external oblique: EO) were positioned according to McGill (McGill, 1991). A reference silver-silver chloride electrode was positioned over the C7 spinous process. The difficulty capturing the multifidus muscle with surface electrodes (Stokes et al., 2003) is acknowledged and therefore the validity of the electromyographic signal was assigned to the landmarked location rather than to the multifidus muscle itself.

**Submaximal reference contractions**

In order to normalize selected EMG amplitude variables (preactivation assessment), submaximal referenced contractions were carried out according to published procedures that have been found to produce reliable results for abdominal (Dankaerts et al., 2004; O'Sullivan et al., 1998) as well as back muscles (Dankaerts, O'Sullivan, Burnett, Straker, and Danneels, 2004). Three 5-s contractions, separated by a 30-s rest interval, were performed for each muscle group. Briefly, the abdominals were recruited while performing a double leg raise exertion (1 cm) in a supine crook lying position with hips flexed at 45° and knees flexed at 90° (O'Sullivan, Twomey, and Allison, 1998). Back muscles were recruited while performing a double leg raise exertion (5 cm) in a prone position with the knees bent at 90° (Dankaerts, O'Sullivan, Burnett, Straker, and Danneels, 2004).

**Back muscle reflex testing protocol**

The reflex response of the back muscles was measured using a sudden loading apparatus (details in Figure 1) designed to pre-load the back muscles and to give an anteriorly directed perturbation of the trunk in the upright position. The sudden load, sustained by a magnet, is released from a height of less than 1 cm so as to avoid ballistic impacts (safety issue). The upright and immobile reference position of the trunk was ensured with the use of visual feedback from a potentiometer attached to the harness behind the subject. To avoid anticipation of the sudden load, the loading system was hidden from the subject, and the forward sudden load, released by a magnet, was applied at a random time between 2 and 6 s after the subject had reached the reference upright position. On average, the subjects took from 3 to 10 s to reach and maintain the desired neutral posture, before the data collection was initiated. Consequently, considering the 2 to 6 s random time-interval, 5 to 15 s elapsed between the instruction to the subject to adopt the neutral position and the release of the sudden load. The subjects were instructed to maintain the trunk position that was reached after the perturbation for 1 s, so as to avoid overreacting by extending the trunk backward.

The sudden load was determined from the mass of the trunk and head, so as to produce an L5/S1 extension moment equivalent to 50% of the moment of these segments if they were horizontal. These estimations require only a few anthropometric measures [height, trunk (hips to C7) and head lengths] and the use of some body segment parameters (de Leva, 1996; Liu et al., 1971) to estimate the L5/S1 extension moment, as detailed elsewhere (Lariviere and Gagnon, 1999).
Figure 1: Experimental setup. The load was applied via a cable that was divided in front of the subject and attached on each side to a wood board positioned behind the subject and kept immobile at the level of T4 with shoulder straps. A load cell measured the applied load while a potentiometer measured trunk kinematics. After the subject reached the upright reference trunk posture (with the help of visual feedback of the potentiometer signal) while sustaining the pre-load, data collection was started and the sudden load was released at a random time between 2 and 6 s. The sudden load system was released by a noiseless electromagnet, allowing the hooks from the pre-load and sudden load, separated by approximately 1 cm, to make contact and to transmit the load to the trunk.

To standardize the preactivation of trunk muscles before the perturbation, two strategies were used: a small pre-loading of the back muscles was produced, and the biofeedback of the two abdominal muscles was displayed to help the subjects minimize their preactivation. The pre-load was determined in a manner similar to the sudden load but its magnitude was lower (20% of the L5/S1 extension moment produced by the trunk and head) to avoid the inhibition of reflex responses (Stokes, Gardner-Morse, Henry, and Badger, 2000). The raw signals from the right RA and right EO muscles were displayed on a screen in front of the subject, beside the feedback from the potentiometer. The instruction was to keep these signals as silent as possible. Data acquisition was begun only when there was evidence that these EMG signals were silent, or at least stable with very small amplitude.
The perturbation protocol consisted first of three practice perturbations to reassure the subject, followed by three practice perturbations to accustom the subject to the newly introduced EMG biofeedback. Then, 10 min. of rest was allowed before the final 20 test perturbations. Each perturbation was followed by approximately one min. rest. The total perturbation protocol (n = 26 trials), including rest intervals, took approximately 40 min.

Maximal voluntary contractions (MVC)

Three MVCs were carried out with the use of a static triaxial trunk dynamometer (Lariviere et al., 2001b). First, two to four submaximal trunk extension contractions were performed to warm up the subjects and to familiarize them with the apparatus and the visual feedback. Then, three MVCs that consisted of 7-s ramp extension efforts were performed, allowing a 2-min. rest period between trials. To avoid spuriously high transient forces resulting from jerky movements, the subject was asked to follow the visual feedback to allow a progressive (ramp) increase in the force toward the maximum. Verbal encouragement was given to the subject during each trial. The peak L5/S1 extension moment across the three MVC trials was defined as Strength.

Data processing

Only the signals from the test trials (last 20 trials) were processed. All EMG signals were first filtered using a notch filter to eliminate possible 60-Hz electrical noise and its harmonics (up to 420 Hz), further filtered using a wavelet method to remove ECG artifact (Bluthner et al., 2001), and then rectified. Afterwards, the following EMG variables were computed: EMG activation of all muscles before the perturbation, the reflex latency, and the amplitude of back muscle responses.

Quantification of trunk muscle preactivation.

The EMG root mean square (RMS) amplitude of each muscle was quantified using a 250-ms time-window positioned just before the perturbation (measured by the load cell), and then divided by the corresponding reference EMG RMS signal collected during the submaximal reference contractions (EMGref = mean RMS value of the three trials) and multiplied by 100 (variable: NRMSref). For back muscles only, the maximal EMG (EMGmax) RMS values (250-ms time-windows, 80% overlapped) were also used to normalize back muscle EMG preactivation (variable: NRMSmax).

Quantification of back muscle reflex latency.

The reflex latency was defined from the start of trunk movement (measured by the potentiometer) to the onset of the EMG response, as detected using two computer-based automated methods: the standard deviation (SD) method (Hodges and Bui, 1996) and the approximated generalized likelihood-ratio (AGLR) method (Staude, 2001). The AGLR method was applied using the following parameters: W=100, h=20 and Delta=10. These latency variables will hereafter be called LatencySD and LatencyAGLR. In both methods, the 250 ms preceding the force perturbation was used as the EMG reference signal, and the EMG onset was searched between 0
and 200 ms after the beginning of trunk movement. However, only responses that were reflexive (lying in the 30-150 ms time-window) were considered for further analysis. To use the SD method, the signal was first dual-pass (zero lag) Butterworth filtered (6th-order, 50 Hz cut-off frequency), and the processed EMG signal was assessed using a 25-ms sliding window. The EMG onset was defined as a signal exceeding a threshold of two SDs above the baseline mean. The AGLR algorithm uses the log-likelihood ratio in order to estimate the probability of a portion of an EMG signal to pertain to a certain reaction variance compared to the variance at rest (Staude and Wolf, 1999; Staude, 2001).

Quantification of back muscle reflex amplitude.
The reflex amplitude was quantified only when a reflex latency was detected in the 30-150 ms time-interval following trunk movement. The EMG signal was first rectified and then dual-pass second-order Butterworth filtered (2nd-order, 25 Hz cut-off frequency). The reflex amplitude was defined as the ratio of the first EMG peak amplitude value (searched after the EMG onset was detected using the SD method) divided by the EMG signal amplitude (250 ms) prior to trunk movement. This ratio will be called \( \text{RatioPeak} \), but the non-normalized EMG peak amplitude (\( \text{RawPeak} \)) was also considered.

Trunk kinematics.
The kinematics of perturbation was also quantified using the potentiometer signal and the distance from L5/S1 to the potentiometer cable: the trunk angle 150 ms after the beginning of trunk movement (variable: \( \text{Ang150ms} \)).

Because EMG variables (reflex and NRMS variables) are more prone to artifacts, the number of trials available (\( n = 20 \)) was used to increase their reliability. Outliers, defined as scores outside 3.0 of the interquartile range, were first eliminated and the remaining scores were averaged. Then, for the EMG variables, considering that no statistically significant difference (T-test, \( \alpha = 0.05 \)) was obtained between homologous (left and right) muscles, the scores were further averaged so as to obtain one score at each vertebral level (L5, L3, L1 and T10).

Statistical analyses
Some of the variables (NRMSref, NRMSmax) were not normally distributed and thus were transformed before any statistical analyses (ANOVA, ANCOVA, Pearson correlations) were carried out. The transform that gave the best normal distribution as possible, according to the Wilk-Shapiro criterion, was systematically searched among log, exponential, power, trigonometric and hyperbolic functions. The best solution was power 1/3 for NRMSref and inverse hyperbolic cos transformation for NRMSmax. All statistical analyses were done with NCSS software (version 6.0 for Windows), using a significance level (alpha) of 0.05. Note, however, that values reported in tables and figures are untransformed values.

Pearson correlations were first carried out to assess whether clinical or pain-related psychological variables (VAS, TSK, PCS) could mediate the reflex assessment variables (preactivation, trunk
kinematics, reflex responses) and to test the association between trunk muscle preactivation, trunk kinematics and reflex responses.

All the dependent variables (preactivation: $NRMS_{ref}$, $NRMS_{max}$; trunk kinematics: $Ang_{150ms}$; reflex latency: $LatencySD$ and $LatencyAGLR$; reflex amplitude: $RatioPeak$, $RawPeak$) were compared between healthy and subjects with CLBP (PAIN-STATUS factor) and between sexes (SEX factor), using two-way ANOVAs ($2 \text{ PAIN-STATUS} 	imes 2 \text{ SEX}$). ANCOVAs were also applied, when appropriate, to account for possible confounding variables as identified through the above-described correlation analyses (more details in the results). The consideration of covariates in the ANCOVAs was based on the presumed effect of trunk muscle preactivation and trunk kinematics, and then on reflex responses, as suggested in the introduction. To test the potential confounding effect of muscle preactivation level on reflex latency detection, it would have been of interest to test the interaction between PAIN-STATUS and the EMG onset detection method (SD vs AGLR), as outlined in the introduction. Unfortunately, the presence of confounding variables forced the use of ANCOVAs, which is not possible in a mixed model (the detection method is a within-subject factor). It was thus decided to test both detection methods separately. The same comment applies to the possible between-muscle comparisons.

RESULTS

Demographic characteristics were comparable between the groups, except for the physical characteristics between sexes and for Strength (Table 1). Subjects with CLBP and women showed significantly lower strength than the healthy controls and men, respectively. Among subjects with CLBP, Strength was not correlated ($r$ ranging from -0.12 to 0.07) to VAS, TSK or PCS scores. Among subjects with CLBP, TSK and PCS were significantly different between the sexes (men > women). Results regarding the pre- and sudden loads, showing differences between sexes but not between healthy controls and patients, are in line with the consideration of the inertial properties of the trunk and head.

Identification of potential confounding variables

A few variables from the reflex assessment were significantly correlated ($n = 27$ subjects with CLBP) to the scores of some questionnaires (VAS, TSK, PCS). $Ang_{150ms}$ was correlated to TSK ($r = -0.55$, $P = 0.003$) and PCS ($r = -0.52$, $P = 0.005$). For the six trunk muscles, VAS was significantly ($P < 0.05$) correlated to preactivation at L5 ($r = -0.47$) and L3 ($r = -0.41$), and PCS was correlated to preactivation at L5 ($r = -0.44$), L3 ($r = -0.53$) and T10 ($r = -0.45$), but only for the $NRMS_{ref}$ variable (not $NRMS_{max}$ computed for back muscles). Finally, for the reflex responses ($LatencySD$, $LatencyAGLR$, $RatioPeak$, $RawPeak$ at each electrode site; $n = 16$ correlations), only two correlations reached statistical significance (VAS vs $LatencyAGLR$ at T10: $r = -0.47$; TSK vs $RatioPeak$ at L3: $r = 0.40$).

For the abdominal muscles (RA and EO), only preactivation of EO ($NRMS_{ref}$) was significantly correlated to $Ang_{150ms}$ ($r = 0.36$; $P = 0.007$). $NRMS_{ref}$ of abdominals was correlated to $RawPeak$ of all back muscles ($r$ ranging from -0.29 to -0.49) and to a lesser extent to $RatioPeak$.
Correlations between the three categories of variables extracted from the reflex assessment [(1) back muscle preactivation; (2) trunk kinematics; and (3) back muscle reflex responses] are presented in Table 2. Briefly, \( r = -0.31 \) and \(-0.29\) for RA and EO respectively), while no correlation to the latency variables was obtained.

Many correlations reached or almost reached statistical significance between back muscle preactivation and their corresponding (at the same electrode site) reflex responses. Positive and negative correlations were obtained with reflex latencies and amplitudes, respectively. The study of Table 2 also reveals that higher correlations were often obtained when \( NRMSmax \) was used instead of \( NRMSref \). Also of interest is that the preactivation level (\( NRMSref \) or \( NRMSmax \)) was more often (across electrode sites) significantly correlated to \( LatencySD \) than \( LatencyAGLR \).

**Table 2.** Pearson correlation coefficients (n = 56 subjects)* between the different categories of variables (trunk kinematics, back muscle preactivation level, back muscle reflex responses)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Electrode sites</th>
<th>L5</th>
<th>L3</th>
<th>L1</th>
<th>T10</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Trunk kinematics (( \text{Ang150ms} )) vs back muscle preactivation level</td>
<td>( NRMSref )</td>
<td>/</td>
<td>0.25</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td></td>
<td>( NRMSmax )</td>
<td>/</td>
<td>0.25</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>2. Trunk kinematics (( \text{Ang150ms} )) vs back muscle reflex responses</td>
<td>( LatencySD )</td>
<td>/</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td></td>
<td>( LatencyAGLR )</td>
<td>/</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td></td>
<td>( RatioPeak )</td>
<td>/</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td></td>
<td>( RawPeak )</td>
<td>-0.23</td>
<td>-0.31</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>3. Back muscle preactivation level (( NRMSref )) vs back muscle reflex responses †</td>
<td>( LatencySD )</td>
<td>/</td>
<td>0.32</td>
<td>0.33</td>
<td>0.31</td>
</tr>
<tr>
<td></td>
<td>( LatencyAGLR )</td>
<td>/</td>
<td>0.25</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td></td>
<td>( RatioPeak )</td>
<td>-0.33</td>
<td>/</td>
<td>-0.33</td>
<td>-0.28</td>
</tr>
<tr>
<td></td>
<td>( RawPeak )</td>
<td>/</td>
<td>/</td>
<td>0.22</td>
<td>/</td>
</tr>
<tr>
<td>4. Back muscle preactivation level (( NRMSmax )) vs back muscle reflex responses †</td>
<td>( LatencySD )</td>
<td>0.26</td>
<td>0.38</td>
<td>0.31</td>
<td>/</td>
</tr>
<tr>
<td></td>
<td>( LatencyAGLR )</td>
<td>0.30</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td></td>
<td>( RatioPeak )</td>
<td>-0.51</td>
<td>/</td>
<td>-0.50</td>
<td>-0.46</td>
</tr>
<tr>
<td></td>
<td>( RawPeak )</td>
<td>-0.27</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
</tbody>
</table>

* Only statistically significant correlations (\( P<0.05 \)) are identified, except for correlations for which \( P \) values approached significance (\( 0.05<P<0.10 \)), which are underlined. † These correlations were carried out between variables (\( NRMS \) vs reflex response) computed at the same electrode site.

Subjects with CLBP preactivated their back muscles more than the healthy controls, while no difference was obtained for the abdominals (Table 3; example in Figure 2A for L3 electrode site). Regarding back muscles, very similar statistical findings were obtained using \( NRMSref \) and \( NRMSmax \). The correlations between \( NRMSref \) and \( NRMSmax \) were moderate (\( r = 0.33 \) to 0.59 across electrode sites), but still all statistically significant. Men and women preactivated their back and abdominal muscles to a similar extent according to both variables (\( NRMSref \), \( NRMSmax \) in back muscles). The results in Table 3 (preactivation) allowed us to conclude that only the
preactivation of back muscles (not abdominal muscles) may affect between-group comparisons for trunk kinematics and reflex responses. Also, for back muscles, even though NRMSref and NRMSmax showed similar between-group effects, NRMSmax would represent a better covariate in the following analyses because the correlations with the other variables (Table 2) were higher than NRMSref. This would be more consistent with the link relating muscle preactivation with trunk kinematics and reflex responses (better logical or face validity), as substantiated in the introduction. Finally, considering that back muscle preactivation was different between subjects with CLBP and healthy controls, the RatioPeak variable (reflex amplitude) would be biased, contrary to RawPeak, because the denominator of this ratio is back muscle preactivation per se.

Trunk kinematics (Ang150ms) was also compared between groups (Figure 2B), using an ANCOVA to adjust for back muscle preactivation (NRMSmax-L3 was selected, according to the statistical results in Table 3). While NRMSmax-L3 just failed to reach significance ($P = 0.065$) in the ANCOVA, a significant PAIN-STATUS × SEX interaction ($P = 0.034$) was observed. Post-hoc analyses (separate ANCOVAs adjusting for NRMSmax-L3) revealed a PAIN-STATUS effect only for men (healthy > CLBP) and a SEX effect only for subjects with CLBP (women > men), as depicted in Figure 2B. Considering these significant effects, Ang150ms has to be considered, in addition to the NRMSmax of back muscles, as a covariate when assessing between-group differences regarding back muscle reflex responses.

**Figure 2:** Boxplot representation of different variables corresponding to healthy controls, subjects with CLBP, men (trapezoid boxes) and women (square boxes) subgroups for different variables associated with the reflex assessment: (A) back muscle preactivation at L3 (NRMSmax variable), (B) angle of the trunk 150 ms after the perturbation (Ang150ms), (C) reflex latency at L3 (approximated generalized likelihood-ratio – AGLR - EMG-detection method), and (D) reflex amplitude at L3 (RawPeak variable). Statistical results ($P$ values) corresponding to main effects ($P =$ PAIN-STATUS; $S =$ SEX) and to the interaction ($P \times S =$ PAIN-STATUS × SEX) are presented on the upper part of each plot.
Table 3. Descriptive statistics [median (interquartile range: 25th and 75th percentiles)*] and statistical results (P values) corresponding to the comparisons between healthy and subjects with CLBP and between men and women for the preactivation of trunk muscles.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Muscle</th>
<th>Healthy controls</th>
<th>CLBP subjects</th>
<th>P values (ANOVA) ††</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Men (n = 15)</td>
<td>Women (n = 14)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Men (n = 14)</td>
<td>Women (n = 13)</td>
</tr>
<tr>
<td>NRMSref</td>
<td>L5</td>
<td>18 (16, 19)</td>
<td>17 (6, 19)</td>
<td>20 (15, 27)</td>
</tr>
<tr>
<td></td>
<td>L3</td>
<td>11 (9, 14)</td>
<td>15 (6, 21)</td>
<td>15 (12, 21)</td>
</tr>
<tr>
<td></td>
<td>L1</td>
<td>20 (17, 23)</td>
<td>17 (7, 25)</td>
<td>24 (14, 31)</td>
</tr>
<tr>
<td></td>
<td>T10</td>
<td>18 (11, 22)</td>
<td>21 (9, 30)</td>
<td>25 (18, 33)</td>
</tr>
<tr>
<td></td>
<td>RA</td>
<td>18 (10, 39)</td>
<td>31 (15, 35)</td>
<td>20 (10, 28)</td>
</tr>
<tr>
<td></td>
<td>EO</td>
<td>19 (10, 34)</td>
<td>24 (16, 36)</td>
<td>15 (8, 28)</td>
</tr>
<tr>
<td>NRMSmax</td>
<td>L5</td>
<td>6 (5, 8)</td>
<td>8 (6, 12)</td>
<td>10 (6, 21)</td>
</tr>
<tr>
<td></td>
<td>L3</td>
<td>3 (2, 4)</td>
<td>4 (2, 5)</td>
<td>5 (2, 8)</td>
</tr>
<tr>
<td></td>
<td>L1</td>
<td>5 (4, 7)</td>
<td>7 (4, 9)</td>
<td>11 (4, 19)</td>
</tr>
<tr>
<td></td>
<td>T10</td>
<td>4 (3, 6)</td>
<td>5 (4, 7)</td>
<td>6 (4, 12)</td>
</tr>
</tbody>
</table>

* Because the data were not normally distributed before transformed and statistically assessed (please see methods), these untransformed values were reported using median and interquartile range.
† Statistically significant differences (P<0.05) are identified in bold characters, while trends (0.05<P<0.10) are underlined.

Reflex responses: between-group comparisons

Between-group comparisons of reflex responses were carried out with (ANCOVA) and without (ANOVA) adjustment for potential covariates (NRMSmax of back muscles, Ang150ms), to better appreciate their importance. Regarding reflex latency, LatencySD and LatencyAGLR led to very similar statistical findings (Table 4). Figure 2C depicts the LatencyAGLR results at L3. Simple ANOVAs revealed only one SEX effect at T10 (no PAIN-STATUS effect), which disappeared in the ANCOVA. No between-group difference was obtained using ANCOVAs adjusting for Ang150ms and NRMSmax. In these ANCOVAs, only NRMSmax was significant but to a greater extent for LatencySD (at L5, L3; L1 almost reached statistical significance) than for LatencyAGLR (at L5 only).

Regarding reflex amplitude, since RawPeak does not account for the filtering effect of subcutaneous tissue thickness because it is not normalized to a reference EMG value (contrary to RatioPeak), an additional covariate (BMI) was considered. BMI was used as a surrogate for subcutaneous tissue thickness according to previous findings (Nordander et al., 2003). RatioPeak and RawPeak led to different findings (Table 5). Figure 2D depicts the RawPeak results at L3. For RatioPeak, ANCOVAs disclosed one more significant effect than ANOVAs (PAIN-STATUS effect at L1), the NRMSmax covariate being significant in all cases except at L3 (P = 0.063). For RawPeak, the role of the NRMSmax covariate was apparently not as important as suggested by the higher and non-significant P values (same results without considering the BMI covariate), which is consistent with the fact that RawPeak is not biased by the preactivation level. In fact, adjusting for NRMSmax changed the main effects slightly, compared to ANOVAs. More specifically, subjects with CLBP showed higher reflex amplitudes than healthy controls at L3.
Also, men showed higher reflex amplitudes than women at L5, L3 and at L1, but the ANCOVA just failed ($P = 0.062$) to show a significant effect at L1.

**Table 4.** Statistical results ($P$ values*) corresponding to the comparisons between healthy and subjects with CLBP and between men and women, for the two methods for EMG onset detection: latency variables.

<table>
<thead>
<tr>
<th>Electrode sites</th>
<th>LatencySD</th>
<th></th>
<th></th>
<th>LatencyAGLR</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pain (P)</td>
<td>Sex (S)</td>
<td>P XS</td>
<td>/</td>
<td>Pain (P)</td>
<td>Sex (S)</td>
</tr>
<tr>
<td>L5</td>
<td>0.657</td>
<td>0.151</td>
<td>0.808</td>
<td>/</td>
<td>0.535</td>
<td>0.904</td>
</tr>
<tr>
<td>L3</td>
<td>0.928</td>
<td>0.255</td>
<td>0.442</td>
<td>/</td>
<td>0.700</td>
<td>0.078</td>
</tr>
<tr>
<td>L1</td>
<td>0.475</td>
<td>0.282</td>
<td>0.631</td>
<td>/</td>
<td>0.210</td>
<td>0.178</td>
</tr>
<tr>
<td>T10</td>
<td>0.663</td>
<td>0.047</td>
<td>0.241</td>
<td>/</td>
<td>0.956</td>
<td>0.046</td>
</tr>
</tbody>
</table>

ANOVA results

<table>
<thead>
<tr>
<th>Electrode sites</th>
<th>ANCOVA results ††</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pain (P)</td>
<td>Sex (S)</td>
<td>P XS</td>
<td></td>
</tr>
<tr>
<td>L5</td>
<td>0.603</td>
<td>0.530</td>
<td>0.141</td>
<td>0.430</td>
</tr>
<tr>
<td>L3</td>
<td>0.007</td>
<td>0.032</td>
<td>0.692</td>
<td>0.669</td>
</tr>
<tr>
<td>L1</td>
<td>0.016</td>
<td>0.598</td>
<td>0.889</td>
<td>0.436</td>
</tr>
<tr>
<td>T10</td>
<td>0.327</td>
<td>0.709</td>
<td>0.393</td>
<td>0.721</td>
</tr>
</tbody>
</table>

*Statistically significant differences ($P<0.05$) are identified in bold characters, while trends ($0.05<P<0.10$) are underlined.

†† Two covariables were considered ($Ang150ms$ and $NRMSmax$ corresponding to the same electrode site) in the ANCOVAs.

**Table 5.** Statistical results ($P$ values*) corresponding to the comparisons between healthy and subjects with CLBP and between men and women, for the reflex amplitude variables ($RatioPeak$ and $RawPeak$).

| Electrode sites | RatioPeak | | | RawPeak | | |
|-----------------|-----------|-----------|-----------|-----------|-----------|
|                 | ANOVA     | | | ANOVA (covariate: BMI) | | |
|                 | Pain (P)  | Sex (S)   | P XS      | /         | Pain (P)  | Sex (S)   | P XS      |
| L5              | 0.475     | 0.405     | 0.544     | /         | 0.932     | 0.000     | 0.772     |
| L3              | 0.255     | 0.009     | 0.513     | /         | 0.008     | 0.000     | 0.339     |
| L1              | 0.523     | 0.677     | 0.373     | /         | 0.093     | 0.032     | 0.739     |
| T10             | 0.997     | 0.564     | 0.664     | /         | 0.290     | 0.309     | 0.189     |

ANOVA (covariates: $Ang150ms$, $NRMSmax$)

<table>
<thead>
<tr>
<th>Electrode sites</th>
<th>ANOVA (covariates: $Ang150ms$, $NRMSmax$)††</th>
<th></th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Pain (P)</td>
<td>Sex (S)</td>
<td>P XS</td>
<td></td>
</tr>
<tr>
<td>L5</td>
<td>0.603</td>
<td>0.530</td>
<td>0.141</td>
<td>0.430</td>
</tr>
<tr>
<td>L3</td>
<td>0.007</td>
<td>0.032</td>
<td>0.692</td>
<td>0.669</td>
</tr>
<tr>
<td>L1</td>
<td>0.016</td>
<td>0.598</td>
<td>0.889</td>
<td>0.436</td>
</tr>
<tr>
<td>T10</td>
<td>0.327</td>
<td>0.709</td>
<td>0.393</td>
<td>0.721</td>
</tr>
</tbody>
</table>

*Statistically significant differences ($P<0.05$) are identified in bold characters, while trends ($0.05<P<0.10$) are underlined.

†† $NRMSmax$ corresponding to the same electrode site
DISCUSSION

The main findings of the present study were: (1) that subjects with CLBP preactivated their back muscles to a greater extent than healthy subjects before the sudden load event, (2) that contrary to reflex latency, back muscle reflex amplitude was affected by sex and by the pain status at specific electrode sites, and (3) that the consideration of confounding variables associated with the presence of CLBP can change the interpretation of reflex responses.

Assessment of potential confounding variables

This study put a lot of emphasis on the control of potential confounding variables, especially regarding (1) the a priori determined testing parameters (trunk muscle preactivation, pre-load and sudden load), and (2) the a posteriori controlled residual confounding variables, through statistical analyses (ANCOVAs).

The trunk muscle preactivation level has been assessed only once in subjects with CLBP (Stokes et al., 2006) and has been suggested as a potential confounding variable to be considered in future studies (Lee, Cholewicki, and Peter, 2007). In partial agreement with the present results, Stokes (Stokes, Fox, and Henry, 2006) showed a higher preactivation of back and abdominal muscles in subjects with CLBP than in controls. Unfortunately in that study, EMG signals were normalized with the use of maximal EMG obtained during MVCs, which might be questionable since it may artificially increase the estimated activation level if their subjects with CLBP had pain-related fears (Thomas, France, Sha, and Wiele, 2008). EMG amplitude normalization is a tricky task, especially in subjects with CLBP. In our study, we tried to control preactivation levels by asking subjects to relax their abdominal muscles, thus reducing the activation of abdominals and the required back muscle activation that would be required to counteract the resulting flexion moment. This was apparently successful, as substantiated by the absence of difference in the contraction levels of the abdominal muscles between the healthy and CLBP groups. However, the CLBP group showed higher preactivation levels of their back muscles. The possibility that the \( NRMS_{\text{max}} \) variable was biased by the presence of pain-related fears in subjects with CLBP has to be considered, since their TSK scores were not negligible and subjects with CLBP were “weaker” than healthy controls (Table 1). However, none of the assessed pain-related psychological variables (VAS, TSK, PCS) were correlated to \( \text{Strength} \) or \( NRMS_{\text{max}} \), which supports the validity of \( NRMS_{\text{max}} \) in the present study, knowing that such a relationship depends heavily on the subject’s sample recruited in each study. Furthermore, \( NRMS_{\text{ref}} \), which is not dependent on an EMG amplitude normalization method relying on maximal reference contractions, produced between-group effects very similar to \( NRMS_{\text{max}} \), though statistically significant correlations were obtained between \( NRMS_{\text{ref}} \) and VAS and PCS. These latter correlations suggest that even though between-group systematic effects can be detected in a similar fashion as \( NRMS_{\text{max}} \), which uses the best available method to normalize EMG while allowing between-subject comparisons (at least in healthy subjects), inter-individual variations in \( EMG_{\text{ref}} \) values collected during the submaximal reference contractions might be induced by pain-related psychological influences. Unfortunately, this weakens the validity of this
submaximal methodology to normalize EMG amplitude values. Consequently, the significant
correlations obtained between NRMSRef and VAS and PCS might be biased by the submaximal
EMG reference value used to normalize EMG preactivation values. In summary, it appears likely
from these analyses that subjects with CLBP preactivated their back muscles more, and according
to the correlations (Table 3) and ANCOVAs (NRMSmax covariate statistically significant; Tables
4 and 5), this has an influence on back muscle reflex responses. The lack of association between
NRMSmax and pain-related psychological variables suggests that psychological variables may
not explain the increased activation of back muscles in subjects with CLBP. This also suggest
that the preactivation level cannot explain the significant correlation ($r = -0.55$) that was obtained
between TSK and Ang150ms. Effectively, an increased preactivation due to pain-related fears, a
possible physiological manifestation of guarding behaviors, could have increased spinal stiffness
and in turn decreased trunk excursion, as suggested by previous findings in healthy subjects
(Stokes, Gardner-Morse, Henry, and Badger, 2000; Vera-Garcia, Brown, Gray, and McGill,
2006), but this was not the case here. The sole alternative explanation we see would be that the
pre-load, which is determined according to body (or trunk and head) size, would represent a
higher proportion of their strength, which would be in line with the Strength results (CLBP <
healthy subjects), keeping in mind that their body size (height and weight) was equivalent to that
of healthy subjects.

Recent evidence shows that back muscle reflex responses are largely accounted for by the
movement velocity of the trunk (Moorhouse and Granata, 2007), so it is recommended that
between-subject differences be investigated, and more particularly sex effects, using protocols
that control trunk movement rather than applied force (Granata and Rogers, 2007). In the present
study, adjusting the pre-load and sudden load according to trunk and head mass was not enough
to eliminate between-sex differences for the Ang150ms variable, a type of velocity variable.
Determining the sudden load without accounting for trunk and head moments of inertia (inertial
factors) may partly explain these results. However, Ang150ms was not statistically significant in
any ANCOVA results, suggesting that even though sex differences were detected for Ang150ms,
these differences were not large enough to affect back muscle reflex responses. This further
suggests that our method to determine the sudden load was good enough to eliminate the
confounding effect of subject anthropometrics, as relating to the statistical results, but still
warrants some adjustments to further help reduce the inter-subject variability of reflex responses
(increase diagnostic capability).

As explained in the statistical analysis section (methods), it was impossible to directly compare
the two detection methods to define EMG onset ($\text{LatencySD}$ and $\text{LatencyAGLR}$). However,
interestingly enough, the preactivation level (NRMSRef or NRMSmax) was more often (across
electrode sites) significantly correlated to $\text{LatencySD}$ than $\text{LatencyAGLR}$, which was obviously
further substantiated in the ANCOVAs (NRMSmax covariate more often significant for
$\text{LatencySD}$). Of course, increasing the preactivation levels increase trunk stiffness, which would
lead to a decrease in trunk displacement and an increase in reflex latency (Vera-Garcia, Brown,
Gray, and McGill, 2006). However, for $\text{LatencySD}$ and $\text{LatencyAGLR}$, these unequal correlation
results suggest that the SD method is more influenced by the background EMG activity than the AGLR method, which apparently generates a detection artifact, as previously shown using artificially modified back muscle EMG signals (Lee, Cholewicki, and Peter, 2007). However, while these findings suggest that the AGLR method might be better than the SD method, between-group comparisons leading to different results would have provided a more convincing demonstration, but this was not the case as discussed next.

**Between-group effects**

No PAIN-STATUS effect was obtained for back muscle reflex latencies, which is contrary to previous findings (Magnusson, Aleksiev, Wilder, Pope, Spratt, Lee, Goel, and Weinstein, 1996; Radebold, Cholewicki, Panjabi, and Patel, 2000; Reeves, Cholewicki, and Milner, 2005) but consistent with another study (Leinonen, Kankaanpaa, Luukkonen, Hanninen, Airaksinen, and Taimela, 2001). The use of different testing protocols (loading/unloading paradigms; load applied through the hands or through the trunk; pelvis and lower limbs stabilized/unrestricted) could explain these differences, particularly when there is no control of EMG preactivation levels or trunk kinematics. In the two studies involving sudden loading (instead of sudden release), the load was transmitted through the hands (Leinonen, Kankaanpaa, Luukkonen, Hanninen, Airaksinen, and Taimela, 2001). In the study where the pelvis was stabilized (Magnusson, Aleksiev, Wilder, Pope, Spratt, Lee, Goel, and Weinstein, 1996), the increased reflex latency observed in the subjects with pain could also be explained by absorption of the load through the arms to decrease the impact on the back muscles. Unfortunately, trunk kinematics was not quantified in these studies, so this interpretation cannot be verified. However, it should be noted that the abdominal muscles investigated here (RA and EO) were similarly activated in both groups. An alternative explanation would be that an increased cocontraction of the abdominals, which is sometimes observed in subjects with CLBP (van Dieen, Selen, and Cholewicki, 2003), was present in the other studies and would explain their abnormal longer latencies because of increased spinal stiffness, as explained earlier. Minimizing abdominal cocontraction through EMG biofeedback in subjects with CLBP might decrease these latencies to normal values, but may also elicit higher reflex amplitude, as discussed in the next paragraph. Clearly, to clarify this issue, we would need to test whether providing EMG biofeedback or not mediates the results obtained from subjects with CLBP.

The higher reflex amplitude at L3 observed in subjects with CLBP, compared to healthy controls, was consistent across the various analyses (ANOVA vs ANCOVA applied on RatioPeak and RawPeak variables) reported in Table 5, which adds to the credibility of this finding. In fact, the possibility that potential confounding variables (NRMSSmax, Ang150ms) have affected these results being excluded with the use of ANCOVAs, this finding can be ascribed to the presence of CLBP per se. More specifically, this may be explained by the need to compensate for a loss of lumbar stability (or stiffness) due to soft tissue injury and/or corrupted signals from injured mechanoreceptors. In both cases, lumbar stability is hypothesized to rely more on the preactivation of trunk muscles (Panjabi, 2003; Panjabi, 2006), especially through the cocontraction of abdominals. However, such abdominal cocontraction was not allowed in the
present study (minimized through EMG biofeedback), and the preactivation of back muscles was accounted for in the ANCOVA. Since trunk stiffness is dependent on its intrinsic stiffness (muscle + passive tissue stiffness) and the reflex response (Davarani et al., 2007), a higher reflex amplitude could compensate here for insufficient intrinsic trunk stiffness and more specifically, for insufficient passive tissue stiffness. This would support previous results (Cholewicki et al., 2000; Davarani, Shirazi-Adl, Hemami, Mousavi, and Parnianpour, 2007). The fact that this finding was obtained only at the L3 electrode site might be related to the biomechanical function of the various back muscles.

Regarding SEX effects, only the reflex amplitude (not latency) was affected, revealing lower values for women at almost three electrodes sites (RawPeak, ANCOVA adjusting for BMI, Ang150ms and NRMSmax), considering that the effect just failed to reach statistical significance at L1. This finding partially agrees with one study showing longer latencies in addition to lower reflex amplitudes in women (Wilder, Aleksiev, Magnusson, Pope, Spratt, and Goel, 1996), but also contradicts others (Granata et al., 2005; Granata and Rogers, 2007; Rogers and Granata, 2006). However, in these latter studies, the sudden load was fixed (70 or 75 N) instead of being determined according to trunk mass properties, leading to greater acceleration and muscle strain velocities in women relative to men, as discussed by the authors (Granata and Rogers, 2007). This in turn led to higher reflex amplitudes in women (Granata, Rogers, and Moorhouse, 2005; Granata and Rogers, 2007; Rogers and Granata, 2006) and comparable latencies (Granata, Rogers, and Moorhouse, 2005). In the present study, even though we attempted to solve this problem, Ang150ms was still significantly higher in women but, fortunately, not to the extent to influence the reflex responses according to the ANCOVA results. The literature on this topic is mixed, showing shorter latencies and higher amplitudes in women for the jaw muscles (Peddireddy et al., 2006; van Selms et al., 2005), while no sex effect was found for the triceps surae (Blackburn et al., 2008) and quadriceps (Moore et al., 2002). The available literature does not support an explanation based on differences in spinal stiffness. More explicitly, considering that the effective spinal stiffness is similar in both sexes (Moorhouse and Granata, 2005) and that “Effective stiffness = intrinsic stiffness + reflex response,” the decrease in the reflex response observed here in women would have been compensated for by an increase in intrinsic muscle stiffness. However, intrinsic stiffness depends on muscle activation stiffness and passive stiffness (passive muscle components and all other passive tissues) and none of these elements is apparently sex dependent. In fact, muscle preactivation was not sex dependent (Table 3) and the possible sex differences in passive stiffness of the trunk are unlikely to be involved here since they account for a small fraction of total trunk stiffness in an upright posture (Cholewicki, Simons, and Radebold, 2000). Nevertheless, the sole study that has looked at sex differences in passive stiffness in the three motion planes did not obtain a difference for spinal flexion (McGill et al., 1994). In the present study, the lower reflex amplitude may be explained, at least in part, by the smaller diameter of back muscle fibers (type I and type II) in women (Mannion et al., 1997; Thorstensson and Carlson, 1987), considering that smaller fibers produce smaller action potentials (Hakansson, 1956).
Limitations
The more or less suitable control of potential confounding variables has already been discussed at the beginning of the discussion section. In addition, the current results may apply only to a sudden load paradigm where the load is applied directly on the trunk and where the lower limbs and pelvis are well stabilized. Whether different reflex responses could be obtained using different combinations of experimental conditions is unknown, although some previous results suggest that this might be the case (Leinonen, Kankaanpaa, Luukkanen, Hanninen, Airaksinen, and Taimela, 2001). The reflex response of deep back muscles, such as the multifidus, would have added relevant information. Knowing the preactivation level of the deep transversus abdominis, a potential lumbar spine stabilizer, would also have been of value. Finally, the CLBP subject sample was likely not large enough to represent the wide heterogeneity of the non-specific CLBP population, preventing further subgroup analyses.

CONCLUSION
Back muscle reflex responses were assessed in subjects with CLBP and in healthy controls with the use of a sudden loading paradigm, while attempting to minimize the potential confounding effect of load magnitude and trunk muscle preactivation. However, subjects with CLBP preactivated their back muscles more than healthy controls, and the final trunk perturbation (kinematics) was greater in women relative to men. While adjusting statistically for these differences, it was found that the reflex amplitude was significantly higher in subjects with CLBP and men compared to healthy controls and women, respectively. Interestingly, contrary to most of the literature available, subjects with CLBP were not shown to have larger reflex latencies than healthy controls. These results could potentially be explained by an appropriate control of confounding variables.

ACKNOWLEDGEMENTS
The present research project was funded by the Occupational Health and Safety Research Institute Robert-Sauvé (IRSST) and the “Réseau provincial de recherche en adaptation-réadaptation (REPAR)” of Quebec (Canada). Special thanks to Flavia Dell’Oso and Marielle Trottier for data collection and to David McFadden, Jean-François Pilon and Hakim Mecheri for technical (engineering) assistance.
ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGLR</td>
<td>Approximated generalized likelihood-ratio</td>
</tr>
<tr>
<td>ANOVA</td>
<td>Analysis of variance</td>
</tr>
<tr>
<td>ANCOVA</td>
<td>Analysis of covariance</td>
</tr>
<tr>
<td>Ang150ms</td>
<td>Trunk angle 150 ms after the beginning of trunk movement</td>
</tr>
<tr>
<td>CLBP</td>
<td>Chronic low-back pain</td>
</tr>
<tr>
<td>EMG</td>
<td>Electromyography</td>
</tr>
<tr>
<td>EMGref</td>
<td>EMG RMS signal corresponding to submaximal reference contractions</td>
</tr>
<tr>
<td>EMGmax</td>
<td>Maximal EMG RMS signal collected during the MVCs</td>
</tr>
<tr>
<td>EO</td>
<td>External oblique</td>
</tr>
<tr>
<td>LatencySD</td>
<td>Reflex latency using the computer-based SD method to detect EMG onsets</td>
</tr>
<tr>
<td>LatencyAGLR</td>
<td>Reflex latency using the computer-based AGLR method to detect EMG onsets</td>
</tr>
<tr>
<td>MVC</td>
<td>Maximal voluntary contraction</td>
</tr>
<tr>
<td>NRMSref</td>
<td>Muscle preactivation in RMS values, normalized using EMGref</td>
</tr>
<tr>
<td>NRMSmax</td>
<td>Muscle preactivation in RMS values, normalized using EMGmax</td>
</tr>
<tr>
<td>PCS</td>
<td>Pain catastrophizing scale</td>
</tr>
<tr>
<td>RA</td>
<td>Rectus abdominis</td>
</tr>
<tr>
<td>RatioPeak</td>
<td>Reflex amplitude corresponding to the ratio between RawPeak and the EMG signal amplitude (250 ms) prior to trunk movement</td>
</tr>
<tr>
<td>RawPeak</td>
<td>Reflex amplitude corresponding to the peak reflex amplitude (in µV)</td>
</tr>
<tr>
<td>RDQ</td>
<td>Rolland-Morris disability questionnaire</td>
</tr>
<tr>
<td>RMS</td>
<td>Root mean square</td>
</tr>
<tr>
<td>SD</td>
<td>Standard deviation</td>
</tr>
<tr>
<td>TSK</td>
<td>Tampa scale of kinesiophobia</td>
</tr>
<tr>
<td>VAS</td>
<td>Visual analog scale</td>
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</table>

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