Clinical Biomechanics 29 (2014) 1131–1138

Contents lists available at ScienceDirect

Clinical Biomechanics

journal homepage: www.elsevier.com/locate/clinbiomech

Lumbar kinematic variability during gait in chronic low back pain and associations with pain, disability and isolated lumbar extension strength

James Steele [a,*], Stewart Bruce-Low [a], Dave Smith [b], David Jessop [a], Neil Osborne [c]

[a] Centre for Health, Exercise and Sport Science, Southampton Solent University, UK
[b] Department of Exercise & Sport Science, Manchester Metropolitan University, UK
[c] AECC Clinic, Anglo European College of Chiropractic, UK

ARTICLE INFO

Article history:
Received 1 May 2014
Accepted 29 September 2014

Keywords:
Lumbar spine
Gait variability
VICON
MedX

ABSTRACT

Background: Chronic low back pain is a multifactorial condition with many dysfunctions including gait variability. The lumbar spine and its musculature are involved during gait and in chronic low back pain the lumbar extensors are often deconditioned. It was therefore of interest to examine relationships between lumbar kinematic variability during gait, with pain, disability and isolated lumbar extension strength in participants with chronic low back pain.

Methods: Twenty four participants with chronic low back pain were assessed for lumbar kinematics during gait, isolated lumbar extension strength, pain, and disability. Angular displacement and kinematic waveform pattern and offset variability were examined.

Findings: Angular displacement and kinematic waveform pattern and offset variability differed across movement planes; displacement was highest and similar in frontal and transverse planes, and pattern variability and offset variability higher in the sagittal plane compared to frontal and transverse planes which were similar. Spearman's correlations showed significant correlations between transverse plane pattern variability and isolated lumbar extension strength ($r = -.411$) and disability ($r = .401$). However, pain was not correlated with pattern variability in any plane. The r^2 values suggested 80.5% to 86.3% of variance was accounted for by other variables.

Interpretation: Considering the lumbar extensors role in gait, the relationship between both isolated lumbar extension strength and disability with transverse plane pattern variability suggests that gait variability may result in consequence of lumbar extensor deconditioning or disability accompanying chronic low back pain. However, further study should examine the temporality of these relationships and other variables might account for the unexplained variance.

1. Introduction

Chronic low back pain (CLBP) is a highly prevalent musculoskeletal disorder (National Institute for Health and Clinical Excellence, 2009; Waddell and Burton, 2000; Walker, 2000; World Health Organisation, 1998) with costs amounting to billions worldwide (Ekman et al., 2005; Freburger et al., 2009; Guo et al., 1999; Katz, 2006; Maniadakis and Gray, 2000; Ricci et al., 2006; Stewart et al., 2003; van Tulder et al., 1995; Waddell et al., 2002). Despite its prevalence, in as much as 85% of LBP cases no specific patho-anatomical diagnosis can be found (White and Gordon, 1982). However, more recently it is acknowledged as a multifactorial condition with a variety of associated dysfunctions (National Research Council, 1998; National Research Council and The Institute of Medicine, 2001). One of the multifactorial dysfunctions reported is gait variability (Roffey et al., 2010; Vogt et al., 2001; Waddell et al., 1997). It has been suggested that deficiencies in motor control during gait may produce excessive stresses to the lumbar spine, which may contribute to development of CLBP (Vogt et al., 2001). However, a recent review has suggested that there is evidence against walking itself being causally associated with CLBP (Roffey et al., 2010).

Healthy participants demonstrate relatively low stride-to-stride variability in lumbar kinematic patterns during both level and incline gaits (Vogt and Banzer, 1999). However, greater stride-to-stride variability at the lumbar spine in all movement planes (Vogt et al., 2001), greater frontal plane coordination variability of the pelvis and trunk (Lamoth et al., 2006a; Seay et al., 2011a) and more rigid transverse plane coordination variability of the pelvis and trunk (Lamoth et al., 2002, 2006a; van Der Hoorn et al., 2012) are reported in participants with CLBP compared with healthy controls. It also appears that pain per se may not be responsible for these gait differences. Lumbar spine kinematics during gait appear to be complex and developed over time, as patterns are evident before pain is experienced (Anders et al., 2005) and both induced pain and fear of pain produce little change in muscle activity in CLBP patients (Lamoth et al., 2004). Indeed recently studies have shown that even

* Corresponding author at: Centre for Health, Exercise and Sport Science, Southampton Solent University, East Park Terrace, Southampton, Hampshire SO14 0YN, UK.
E-mail address: james.steele@solent.ac.uk (J. Steele).

http://dx.doi.org/10.1016/j.clinbiomech.2014.09.013
0268-0033/

those with a previous history of CLBP who are currently asymptomatic demonstrate abnormal gait patterns (Crosbie et al., 2012; Seay et al., 2011a). Thus pain per se may not be the factor responsible. There is contrasting evidence reporting no residual effect upon gait from an episode of low back pain in nurses returning to work with very low pain levels (Rowe and White, 1997); however this study lacked a directly comparable control group.

Evidence instead suggests that the lumbar extensor musculature might play a role in gait variability in CLBP (Arendt-Nielsen et al., 1996; Hanada et al., 2011; Lamoth et al., 2004, 2006a, 2006b; van Der Hulst et al., 2010a, 2010b, 2010c; Vogt et al., 2003). It appears that the kinematic patterns seen in participants with CLBP are combined with poorer erector spinae activity adaptability to unexpected perturbations (Lamoth et al., 2004), or walking velocity changes (Lamoth et al., 2002). In fact, the findings of numerous studies are suggestive of muscular dysfunction of the lumbar extensors during gait in those with CLBP compared with asymptomatic controls (Arendt-Nielsen et al., 1996; Lamoth et al., 2004, 2006a, 2006b; Vogt et al., 2003). Hanada et al. (2011) also report that where asymptomatic controls significantly activated their rectus abdominus and internal obliques more, symptomatic participants had significantly greater activation of the lumbar extensors. More recent work shows evidence of greater lumbar extensor activity in participants with CLBP compared with controls (van Der Hulst et al., 2010a), at a range walking velocities (van Der Hulst et al., 2010b), and that neither disability nor fear of movement is associated with this greater activity (van Der Hulst et al., 2010a). However, different coping strategies may be associated with either greater activity (catastrophizing) or greater relaxation during double support (distraction) suggesting some influence of cognitive control (van Der Hulst et al., 2010c).

Human gait is normally quite robust in the face of muscular weakness of the lower limbs (van Der Krogt et al., 2012). The lumbar spine, however, may play a primary role in human bipedal gait (Gracovetsky, 1985). It is possible that the greater activation of the lumbar extensors, and altered lumbar spine kinematics during gait in participants with CLBP, are a manifestation of the lumbar extensor deconditioning (i.e. reduced lumbar extensor strength/endurance, atrophy, and excessive fatigability) commonly associated with CLBP (Steele et al., 2014). Greater activation in the face of fatigue due to deconditioning could be a compensatory attempt to maintain control of the lumbar spine during gait. Hart et al. (2009) demonstrate that inducing fatigue in the lumbar extensors impacts lumbar kinematics during running gait of healthy participants and participants with CLBP. Arjunan et al. (2010) also show significantly greater lumbar extensor activity during running gait in participants with CLBP. Indeed, prospective evidence has demonstrated that reduced lumbar extensor strength/endurance, atrophy, and excessive fatigability increase risk of low back injury and LBP in asymptomatic persons (Steele et al., 2014). Thus it may be responsible for the development of the gait variability associated with CLBP also.

Considering this it was therefore of interest in the present study to examine the relationships between lumbar kinematic variability during gait, with pain, disability and isolated lumbar extension (ILEX) strength. Previous research has focused upon trunk/pelvis co-ordination (Lamoth et al., 2002, 2006a, 2006b, 2008; Seay et al., 2011a, 2011b; Selles et al., 2001). Those interested in stride-to-stride variability of the lumbar spine with respect to the pelvis instead have utilised Winter's coefficient of variation (CV) (Winter, 1983) to examine the consistency of movement patterns using the ensemble average of the raw waveforms of repeated trials (Vogt and Banzer, 1999; Vogt et al., 2001). However, a new method of differentiating between pattern and offset variability has been recently suggested (O'Dwyer et al., 2009). A large mean offset value effectively deflates the value calculated for variability using the CV (O'Dwyer et al., 2009). Because of this O'Dwyer et al. (2009) have suggested the use of methods to differentiate the offset from calculation of the variability in the waveform pattern; the latter they suggest being far more representative of movement replication whereas the offset incorporates a greater degree of other variance sources (i.e. marker error). Thus this study in particular aimed to examine variation in lumbar kinematic pattern variability in relation to pain, disability and isolated lumbar extension (ILEX) strength.

2. Methods

2.1. Study design

The study was part of a wider investigation examining ILEX in participants with CLBP published in part elsewhere (Steele et al., 2013a). Gait data were also collected as part of this wider investigation. The present manuscript presents the cross-sectional data from the combined sample of the study collected at baseline.

2.2. Participants

Thirty eight participants (males n = 21, females n = 17) were initially identified and recruited into the wider investigation by posters, group email and word of mouth from a University and the surrounding locality. Direct referral was also provided from a local private chiropractor through posters in their practice. Inclusion criteria were as follows; participants suffered from non-specific low back pain having lasted longer than 12 weeks (Frymoyer, 1988) and had no medical condition for which resistance training would be contraindicated. Exclusion criteria were as follows; participants must have no medical condition for which movement therapy would be contraindicated. These included: acute (not re-occurring) low back injury occurring within the last 12 weeks, pregnancy, evidence of sciatic nerve root compression (sciatica), leg pain radiating to below the knee, paraesthesia (tingling or numbness), current tension sign, lower limb motor deficit, current disc herniation, previous vertebral fractures or other major structural abnormalities. All participants were cleared prior to involvement in the study by either their General Practitioner or the Chiropractor in the research group and provided written informed consent. The study was approved by the NHS National Research Ethics Service, Southampton & South West Hampshire Research Ethics Committee B (REC Reference: 11/H0504/9).

2.3. Equipment

Participants' stature was measured using a stadiometer (Holtan ltd, Crymych, Dyfed), body mass measured using scales (SECA, Germany) and Body Mass Index (BMI) calculated. Isometric ILEX strength testing and ROM were performed using the (MedX, Ocala, Florida; Fig. 1). Fig. 1 shows the restraint system. The has been shown to be reliable in assessing isometric strength at repeated angles in asymptomatic (Graves et al., 1990) and symptomatic participants (Robinson et al., 1992), and valid in measurement through removal of gravitational effects (Pollock et al., 1991) and pelvic movement (Inanami, 1991). Pain was measured using a 100 mm point visual analogue scale (VAS) (Ogon et al., 1996), and disability measured using the revised Oswestry disability index (ODI) (Fairbank et al., 1980). Gait kinematic variables were captured at 500 Hz using a 10 MX T20 camera three dimensional motion capture system (Vicon, Oxford) and analysed using both Vicon Nexus software version 1.4.116 (Vicon, Oxford), MATLAB version R2012a (MathWorks, Cambridge) and Microsoft Excel version 2010 (Microsoft, Reading).

2.4. Participant testing

For baseline testing participants visited the lab on three occasions. Participants were required to complete the VAS and the ODI on their first visit to the laboratory. The first two visits also involved testing of isometric ILEX strength. This was tested on separate days (at least 72 h apart in order to avoid the effects of residual fatigue or soreness).

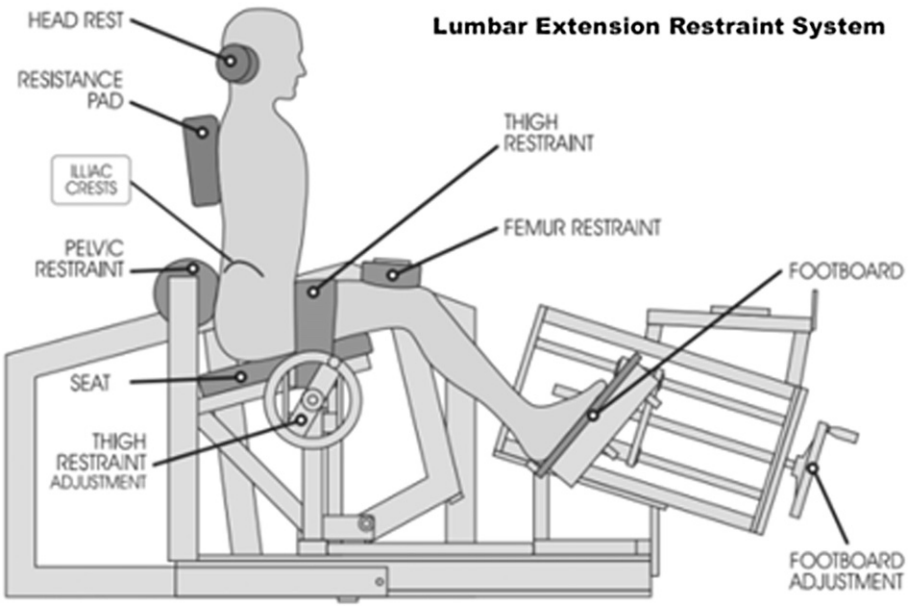

Fig. 1. Lumbar extension machine restraint system.

The first test acted as a familiarisation and the data from the second test was used for analysis. Each test using the lumbar extension machine involved maximal voluntary isometric contractions at various angles through the participant's full ROM. Details of the full test protocol using the lumbar extension machine and details of the restraint mechanisms have been documented previously elsewhere (Graves et al., 1990). Because of individual differences between participants for lumbar ROM, ILEX strength data was averaged across all angles tested. Gait data was collected using the Vicon system during the third visit to the laboratory (again at least 72 h after ILEX testing in order to avoid the effects of residual fatigue or soreness).

2.5. Three dimensional motion analyses

Due to the lumbar spine's capacity to rotate about three orthogonal axes, a three dimensional approach was used for data collection. Ten cameras were set up and angled in a manner so as to reduce hidden spots that might obscure data collection. The cameras were angled such that the centre of the runway used for walking trials was the volume calibrated for data capture. The cameras identified reflective markers attached to the participant and output three dimensional coordinates for each marker. Data were recorded for 5 walking trials both pre and postintervention. Participants walked barefoot from one end of a marked runway to the other that was 8 m in length at their free walking speed. The first full gait cycle captured where the participants entered the calibration volume during each walking trial was used.

2.6. Biomechanical model

The body of interest for the current study was the lumbar spine considered from S1 to T12 relative to the pelvis. For the purpose of analysis the lumbar spine was modelled as a rigid segment. The reasoning for not considering intervertebral segment movements was due to the small segments ranging from S2 to T10 always bending laterally towards the support leg with little variation between segments during gait (Syczewska et al., 1999). Lumbar spine data were collected through three axes using the same model previously described by Scache et al. (Schache et al., 2002a), which has been shown to have high overall repeatability of angular parameters (Schache et al., 2002b).

2.7. Marker setup

Reflective markers were placed over anatomical landmarks on the pelvis at both anterior superior iliac spines (ASIS) and at the midpoint of the posterior superior iliac spine (PSIS). Reflective markers were also used upon a thoracolumbar marker cluster similar to that used by Schache et al. (2002a, 2002b). As with the biomechanical model, this marker setup has been previously described elsewhere (Schache et al., 2002a, 2002b). The only alteration in this present study was the use of a flexible based wand marker for the thoraco-lumbar cluster. Two

Fig. 2. Pelvic and thoracolumbar cluster marker set-up.

Table 1
Participant demographics.

	n = 24
Age (years)	46 ± 14
Stature (cm)	174.01 ± 9.68
Body mass (Kg)	77.07 ± 16.50
BMI (Kg/m^2)	25.17 ± 3.36
Symptom duration (years)	14 ± 11
VAS (mm)	40.17 ± 24.54
ODI (pts)	32.50 ± 11.81
Lumbar extension strength (Nm)	190.05 ± 76.65

additional markers were secured equidistant either side of the midpoint of the wand marker base. This was placed over T12 with the mid-point of the base located over the spinous process. The ASIS and PSIS were identified by palpation after identifying the iliac crest and palpating along its length. T12 was first located and marked using the technique suggested in *Gray's Anatomy for Students* (Drake et al., 2010). This location was confirmed, whilst the participant was in a flexed standing position supporting themselves upon a stool, by palpation and counting of the spinous processes from this marked point down to the sacrum, and then double checked by counting back up to the marked spinous process. All markers and the base of the thoracolumbar marker cluster were secured using double sided adhesive tape. Markers were placed by the same investigator for all gait trials. Fig. 2 shows the marker set-up used.

2.8. Kinematic data

The variability of angular kinematics of the lumbar spine about the three described axes relative to the pelvic segment was of primary interest (i.e. movement of the thoraco-lumbar marker cluster with respect to the pelvic markers). The Vicon Nexus software was used to run a Bodybuilder (Vicon, Oxford) code pipeline to calculate joint angles as outputs using Cardan (Euler) angles. The angles were calculated in the following order: 1) sagittal, 2) frontal, and 3) transverse. As with the biomechanical model, the Bodybuilder code used was the same as used by Schache et al. (2002a, 2002b). Data were filtered using a low pass Butterworth filter (fourth order, cutoff frequency determined for each individual participant as sum of residuals closest to zero using 4 Hz, 6 Hz, 8 Hz, 10 Hz, and 12 Hz) and normalised to percentage gait cycle corresponding to initial right heel contact (0%) and subsequent right heel contact (100%). Heel contacts were identified as the lowest vertical displacement of a right heel marker. Stride duration and length were also calculated using the horizontal displacement of the right heel marker from initial right heel contact and subsequent right heel contact. Mean values for angular displacements, stride-to-stride intra-subject variability using CV_p and CV_o, were calculated for lumbar spine kinematics relative to the pelvis across all three planes of movement.

Intra-subject variability was calculated using coefficient of variation of the ensemble average (average of the normalised time series data at each interval). As highlighted, O'Dwyer et al. (2009) note that the variability of mean offsets (CV_o) and waveform pattern variability (CV_p) should be calculated separately to account for the different information they provide; CV_o being determined by the reference frame used, identification of anatomical landmarks, markers and their configuration, whereas CV_p is more representative of repeatability of motor performance. Adding to this, the model used in this study has been examined for within-day repeatability previously and it was reported that marker reapplication errors and their effect upon daily mean offsets were the main source of concern (Schache et al., 2002b). Thus both CV_p and CV_o were also calculated using equations from O'Dwyer et al. (2009) to allow differentiation of offset variability from pattern variability, the former being better representative of motor performance repeatability.

2.9. Data analysis

Twenty four participants' data (Males, n = 13; Females, n = 11) were available for analysis after attrition. This number of participants combined with 5 trials per participant was sufficient for achieving adequate statistical power in a study of this kind (Bates et al., 1992). Isometric ILEX strength, recorded in units of torque, was measured across the participants' full ROM as foot pounds ($ft \cdot lbs^{-1}$) and converted to Newton metres (Nm) using a correction of 1.356. Kinematic variables (including means for displacements, stride-to-stride intra-subject variability using CV_p and CV_o), pain, disability, and ILEX strength were examined and Spearman's correlations were calculated among them as well as the square of the correlation coefficient. Statistical analysis was performed using SPSS statistics computer package (vs. 20) and $P \leq .05$ set as the limit for statistical significance.

3. Results

3.1. Participant demographics

Participant demographics, pain, disability and ILEX strength data are shown in Table 1.

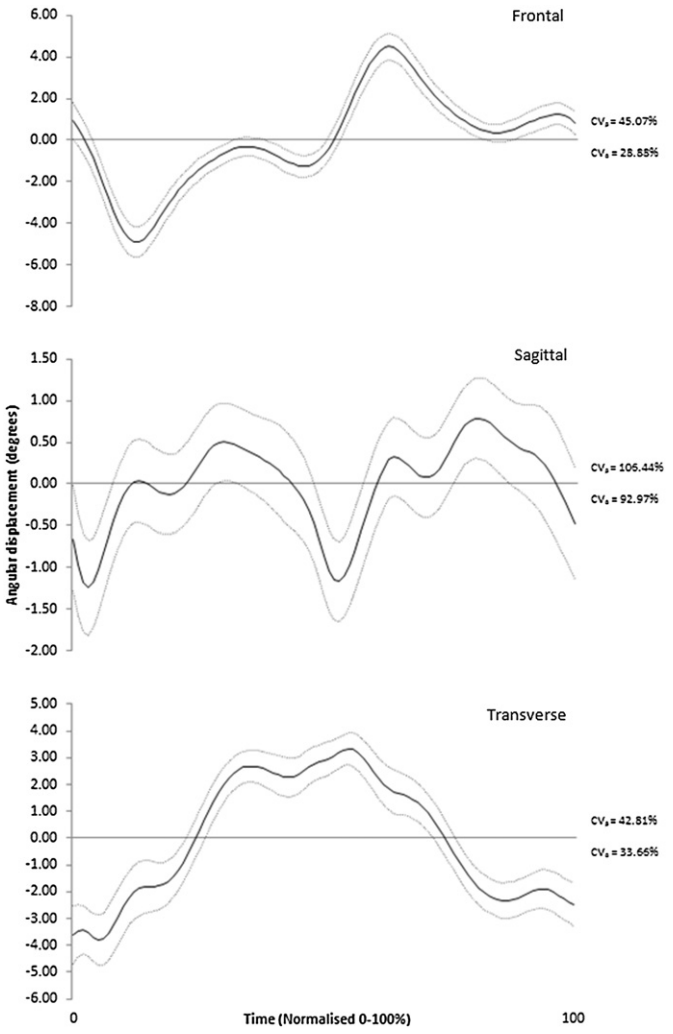

Fig. 3. Waveform patterns, CV_p and CV_o. From top to bottom graphs show mean (±standard deviation) with raw signals transformed to zero mean removing offsets and CV_p and CV_o for frontal, sagittal and transverse lumbar spine kinematics for pooled data (n = 24).

3.2. Kinematic data

Stride duration, length and gait velocity respectively were 1.03 ± 0.18 s, 1.30 ± 0.13 m, and 1.31 ± 0.34 m/s across the five walking trials.

Fig. 3 shows the waveform time-series including angular displacement, CV_p, and CV_o. Displacement was highest and similar in frontal and transverse planes. Contrastingly CV_p and CV_o were higher in the sagittal plane than in frontal and transverse planes which were both also similar.

3.3. Correlations between kinematic variables, VAS, ODI, and ILEX strength

Significant moderate positive correlations were found between ODI and transverse plane CV_p ($r = .401$, P 0.026). Significant moderate negative correlations were also found between ILEX strength and frontal plane CV_o ($r = -.370$, $P = 0.045$), transverse plane CV_p ($r = -.411$, $P = 0.029$), transverse plane CV_o ($r = -.378$, $P = 0.042$) and a significant moderate positive correlation with transverse plane displacement ($r = .442$, $P = 0.020$). Figs. 4–6 present scatter plots of the rank ordered data for transverse displacement and CV_p. The square of the correlation coefficients suggests that the degree of variance explained by ODI was 16.1%. The degree of variance explained ILEX strength was 13.7% for frontal place CV_o, 16.9% for transverse plane CV_p, 14.3% for transverse plane CV_o, and 19.5% for transverse plane displacement. Thus 80.5% to 86.3% of the variance was accounted for by other variables.

4. Discussion

This study of lumbar kinematic variability during gait in participants with CLBP yields several interesting and unique results which potentially offer further understanding of the nature of the relationships between CLBP, gait variability and lumbar extensor deconditioning. Waveform patterns and angular displacements of the lumbar spine exhibited by participants with CLBP in the present study were similar to those reported in other studies of both symptomatic (Lamoth et al., 2006a; Rowe and White, 1997; van Der Hoorn et al., 2012; Vogt et al., 2001) and healthy participants (Crosbie et al., 1997; Fowler et al., 2006; Krebs et al., 1992; Stokes et al., 1989; Thurston and Harris, 1983; Vogt and Banzer, 1999; Vogt et al., 2003; Whittle and Levine, 1999).

Within this study however the foremost interest was the repeatability of lumbar spine movement patterns exhibited (intra-subject stride-to-stride variability) as, despite similar average movements occurring at the lumbar spine, symptomatic participants appear less able to replicate these consistently (Vogt et al., 2001). Vogt et al. (2001) reported data suggesting that lumbar movement variability during gait was significantly higher in participants with CLBP compared with asymptomatic controls, and that both sagittal plane variability and transverse plane variability were greater than frontal plane variability. Vogt et al. (2001) however did not differentiate pattern and offset variability as we have done so here.

Schache et al. (2002b) have shown that although high within-day repeatability was displayed for the model adopted in the present study, angular parameters were most susceptible to marker reapplication errors that affected waveform offset. Our data highlights this showing that CV_p differs considerably from variation calculated using Winter's CV reported in earlier studies (Vogt et al., 2001). Sagittal plane CV_p (106.44%) is more than double the variation seen in the frontal (45.07%) and transverse planes (42.81%).

CV_p has not been calculated in participants with CLBP previously and thus it is not possible to verify whether this greater sagittal plane pattern variability is a typical characteristic of their gait. Nor is it possible to define the clinical meaning of this in comparison to healthy gait as CV_p has also not been reported on lumbar spine gait kinematics in asymptomatic participants to the author's knowledge. Yet, despite the high sagittal plane CV_p in comparison to other planes of movement, our correlation results suggest that there is instead a relationship between ILEX strength and transverse plane kinematics; lower transverse displacement and higher CV_p being associated with lower ILEX strength. It might be speculated upon that this relationship in participants with CLBP may be a consequence of the lumbar extensor deconditioning frequently associated with this population (Steele et al., 2014). Indeed it could be recalled that extensor fatigue impacts upon lumbar kinematics during gait emphasising the link between deconditioning and gait abnormality (Hart et al., 2009).

The lumbar spine has been considered to potentially play an important role in control of gait, from the early identification of movement about the pelvis being fundamental to controlling displacement of centre of mass during walking (Saunders et al., 1953), to Gracovetsky's (Gracovetsky, 1985) presentation of the 'Spinal-Engine' as being an important driver of human bipedal gait through all three planes of movement. Although, the specific role of movement about the pelvis in reducing centre of mass displacement (Della Croce et al., 2001; Hayot et al., 2013; Kerrigan et al., 2000; Kuo, 2007; Lin et al., 2014; Schache et al., 1999) has been questioned, and little corroboration for its role in driving locomotion exists (Rice et al., 2004). Yet despite this, considerable research has demonstrated that the lumbar spine in

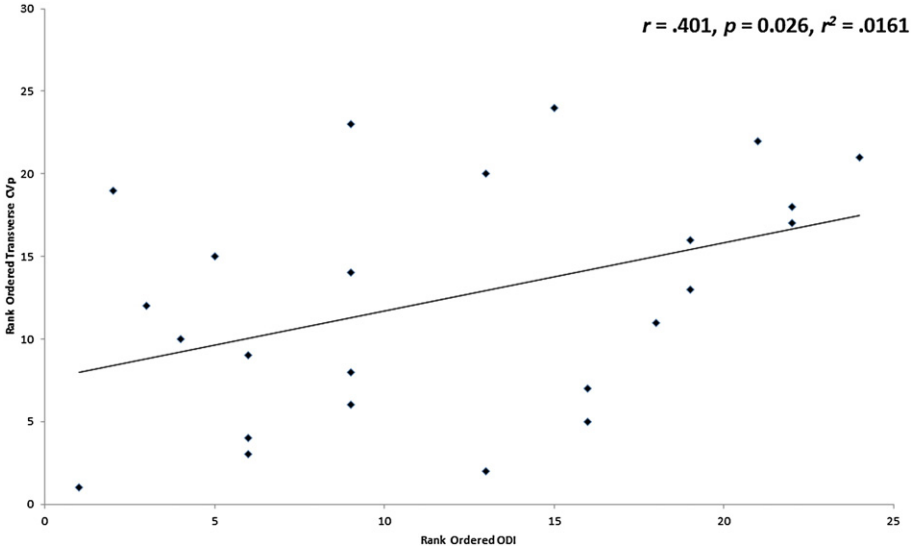

Fig. 4. Scatter plot of rank ordered data for ODI and transverse CV_p.

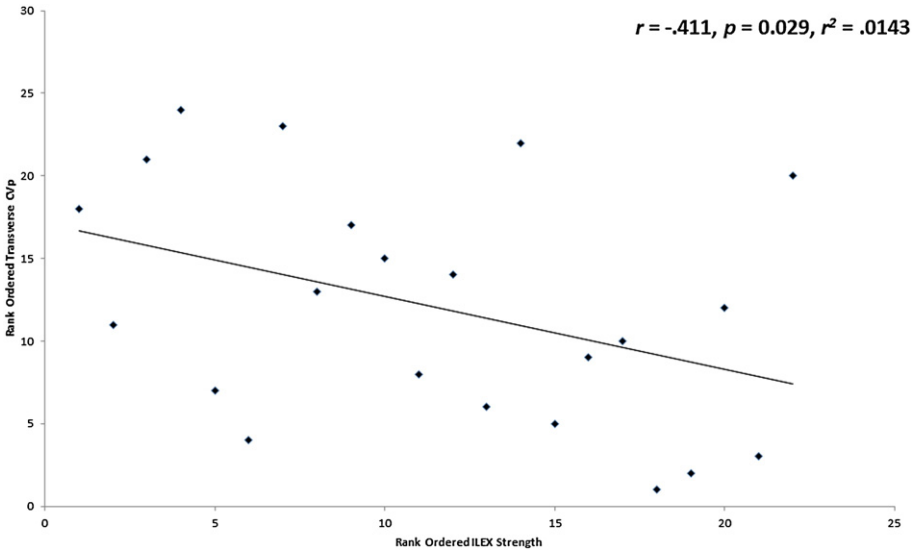

Fig. 5. Scatter plot of rank ordered data for ILEX strength and transverse CV_p.

healthy gait shows consistently reproducible sinusoidal waveform patterns between participants (Crosbie et al., 1997; Fowler et al., 2006; Krebs et al., 1992; Stokes et al., 1989; Thurston and Harris, 1983; Vogt and Banzer, 1999; Vogt et al., 2003; Whittle and Levine, 1999) and that healthy participants have high intra-stride reproducibility of these patterns (Vogt and Banzer, 1999; Vogt et al., 2003).

Such fine control of the lumbar spine during healthy gait is undoubtedly aided by the musculature. Most studies examining muscular contributions to gait have identified the active role that the lumbar extensor musculature plays (Callaghan et al., 1999; Thorstensson et al., 1982; Winter et al., 1993). Thorstensson and colleagues (Thorstensson et al., 1982) showed that the pattern of the multifidus and longissimus activation during gait involved two bursts of activity per cycle each corresponding to foot strike. They concluded that this activity in relation to the pattern of trunk movement suggested that the lumbar extensor muscles main function during gait is to control and restrict excessive trunk movement. Callaghan and colleagues (Winter et al., 1993) demonstrated similar bimodal activity corresponding to greater peak in the musculature ipsilateral to the contacting foot. The activity of the lumbar musculature appears to follow a particular pattern seemingly to stabilise superior segments against inertial and gravitational forces during both single foot contacts (Callaghan et al., 1999; Thorstensson et al., 1982; Winter et al., 1993).

It seems reasonable that in a pathology such as CLBP, wherein there is an associated deconditioning of what appears to be a critically important musculature for controlling gait, that the deconditioning of this musculature might be considered as potentially responsible for gait variability. Indeed our results tend towards supporting this with respect to transverse plane CV_p during gait. It might be noted that some authors have reported that transverse plane kinematics typically show *lower* variability in those with CLBP (Lamoth et al., 2002, 2006a; van Der Hoorn et al., 2012). However, these studies have examined the *coordination* of the trunk and pelvis and variability in the phase differences whereas the present study has instead examined the lumbar spine waveform relative to the pelvis. This difference in methodology may account for the difference in conclusions between these studies. Our results did also suggest that low ILEX was associated with smaller transverse displacements. Perhaps transverse movement is more rigid in CLBP, yet within that smaller range of movement there is a poor waveform pattern repeatability. The rigidity seen in transverse kinematic

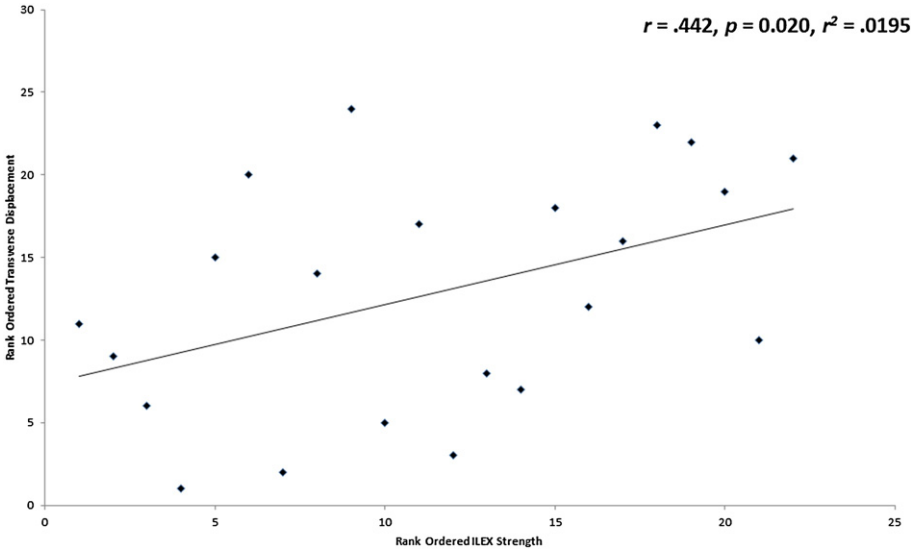

Fig. 6. Scatter plot of rank ordered data for ILEX strength and transverse displacement.

coordination in CLBP (Lamoth et al., 2002, 2006a; van Der Hoorn et al., 2012) may yet still be a manifestation of lumbar extensor deconditioning and may be a gross compensatory adjustment to reduce the impact of the reduced waveform pattern control by limiting the range of motion experienced. Considering this it may be of future interest to examine the relationship between ILEX and trunk/pelvis coordination in those with CLBP.

In addition, our results provide further evidence against the idea that pain per se may cause the variability seen during gait in CLBP. No significant correlation found between VAS and CV_p or any other kinematic variable supporting the findings of others that pain presence appears not to be associated with gait variability (Anders et al., 2005; Lamoth et al., 2004; Seay et al., 2011a). There was however also a significant correlation between ODI and transverse plane CV_p. Considering the multifactorial nature of CLBP it would be reasonable then to consider this evidence suggests that gait variability is potentially a dysfunction associated with CLBP that may result as a consequence of deconditioning of the lumbar extensors or the disability accompanying CLBP. However, it should be noted that the square of the correlation coefficients reported in the present study indicates that the independent variables examined and found to be significantly correlated with gait variables (ILEX strength and ODI) only accounted for a small degree of the variance. Between 80.5% and 86.3% of the variance was accounted for by other unexamined variables. Perhaps other cognitive variables account for some of the unexplained variance as, for example, different coping strategies (catastrophizing or distraction) in participants with CLBP have been shown to impact gait characteristics (van Der Hulst et al., 2010c).

The results of this study however might suggest that exercise based interventions aimed at addressing the lumbar extensor deconditioning in CLBP may be a justified approach to address a range of dysfunctions associated with CLBP. Indeed previous studies have provided support for exercise based interventions on improving aspects of gait variability including muscle activation (Tsao and Hodges, 2008), ground reaction force parameters (da Fonseca et al., 2009) and displacements during gait (Carpes et al., 2008). However, none has examined specifically lumbar kinematic variability during gait, nor has prior work utilised specific exercise designed to isolate the lumbar extensors. Recent work has found that improvement in ILEX strength resulting from a strengthening programme predicts improvement in gait endurance in participants with CLBP (Vincent et al., 2013). Indeed, ILEX based exercise has recently been suggested as optimal for conditioning of the lumbar extensors (Steele et al., 2013b). Future studies might look to whether such exercise interventions can improve the gait variability seen in participants with CLBP.

A limitation within the present study was the lack of a comparable healthy control group. Earlier studies have suggested greater lumbar spine variability in participants with CLBP compared to healthy controls (Vogt and Banzer, 1999; Vogt et al., 2003). Thus it might seem reasonable to speculate that CV_p data would likely be greater in the participants with CLBP in this study if compared with healthy controls also. However, CV_p has not been calculated for lumbar spine kinematics in healthy participants as of yet to the author's knowledge. Thus future work in healthy participants should utilise this method (O'Dwyer et al., 2009) in order to produce normative data to conduct comparisons and also provide data in order to judge improvement from clinical intervention.

5. Conclusions

The results of this study have provided novel information on lumbar spine kinematic variability during gait in CLBP. These new findings are in contrast to earlier ones and instead suggest that the highest variability is observed in sagittal plane lumbar movement during gait in CLBP. Further to this, there was a significant relationship between both ILEX strength and ODI with transverse plane lumbar CV_p. And, a lack of relationship between VAS and CV_p in any plane measured during gait. Further research should aim to provide comparable data from healthy controls. This would allow a better understanding of whether the pattern variability seen here is typical or associated specifically with CLBP. In addition, the relationship between the gait characteristics examined here and other variables should be examined in order to investigate what might be responsible for the unexplained variance in the relationships reported. However, the relationship found between ILEX strength and transverse plane lumbar variability suggests that the lumbar extensors might play an important role in controlling gait in CLBP and thus future work might examine the effect of specific exercise for this musculature upon lumbar spine kinematic variability during gait.

References

Anders, C., Scholle, H.C., Wagner, H., Puta, C., Grassme, R., Petrovitch, A., 2005. Trunk muscle co-ordination during gait: relationship between muscle function and acute low back pain. Pathophysiology 12 (4), 243–247.

Arendt-Nielsen, L., Graven-Nielsen, T., Svarrer, H., Svensson, P., 1996. The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. Pain 64 (2), 231–240.

Arjunan, S.P., Kumar, D.K., Poon, W.M., Rudolph, H., Hu, Y., 2010. Variability in surface electromyogram during gait analysis of low back pain patients. J. Med. Biol. Eng. 30 (3), 133–138.

Bates, B.T., Dufek, J.S., Davis, H.P., 1992. The effect of trial size on statistical power. Med. Sci. Sports Exerc. 24 (9), 1059–1068.

Callaghan, J.P., Patla, A.E., McGill, S.M., 1999. Low back three-dimensional joint forces, kinematics, and kinetics during walking. Clin. Biomech. 14, 203–216.

Carpes, F.P., Reinehr, F.A., Mota, C.B., 2008. Effects of a program for trunk strength and stability on pain, low back and pelvis kinematics, and body balance: a pilot study. J. Bodyw. Mov. Ther. 12, 22–30.

Crosbie, J., Vachalanthiti, R., Smith, R., 1997. Patterns of spinal motion during walking. Gait Posture 5, 6–12.

Crosbie, J., de Faria Negrao Filho, R., Nascimento, D.P., Ferreira, P., 2012. Coordination of spinal motion in the transverse and frontal planes during walking in people with and without recurrent low back pain. Spine 38 (5), E286–E292.

da Fonseca, J.L., Magini, M., de Freitas, T.H., 2009. Laboratory gait analysis in patients with low back pain before and after a Pilates intervention. J. Sport Rehabil. 18, 269–282.

Della Croce, U., Riley, P.O., Lelas, J.L., Kerrigan, D.C., 2001. A refined view of the determinants of gait. Gait Posture 14 (2), 79–84.

Drake, R.L., Vogl, A.W., Mitchell, A.W.M., 2010. Gray's Anatomy for Students. Churchill Livingstone, New York.

Ekman, M., Johnell, O., Lidgren, L., 2005. The economic cost of low back pain in Sweden in 2001. Acta Orthop. 76 (2), 275–284.

Fairbank, J.C., Couper, J., Davies, J.B., O'Brien, J.P., 1980. The Oswestry low back pain disability questionnaire. Physiotherapy 66 (8), 271–273.

Fowler, N.E., Rodacki, A.L.F., Rodacki, C.D., 2006. Changes in stature and spine kinematics during a loaded walking task. Gait Posture 23, 133–141.

Freburger, J.K., Holmes, G.M., Agans, R.P., Jackman, A.M., Darter, J.D., Wallace, A.S., et al., 2009. The rising prevalence of chronic low back pain. Arch. Intern. Med. 169 (3), 251–258.

Frymoyer, J., 1988. Back pain and sciatica. N. Engl. J. Med. 318, 291–300.

Gracovetsky, S., 1985. An hypothesis for the role of the spine in human locomotion: a challenge to current thinking. J. Biomed. Eng. 7, 205–216.

Graves, J.E., Pollock, M.L., Carpenter, D.M., Leggett, S.H., Jones, A., MacMillan, M., et al., 1990. Quantitative assessment of full range of motion isometric lumbar extension strength. Spine 15 (4), 289–294.

Guo, H.R., Tanaka, S., Halperin, W.E., Cameron, L.L., 1999. Back pain prevalence in US industry and estimates of lost workdays. Am. J. Public Health 89, 1029–1035.

Hanada, E.Y., Johnson, M., Hubley-Kozey, C., 2011. A comparison of trunk muscle activation amplitudes during gait in older adults with and without chronic low back pain. PM R 3 (10), 920–928.

Hart, J.M., Kerrigan, D.C., Fritz, J.M., Ingersoll, C.D., 2009. Jogging kinematics after lumbar paraspinal muscle fatigue. J. Athl. Train. 44 (5), 475–481.

Hayot, C., Sakka, S., Lacouture, P., 2013. Contribution of the six major gait determinants on the vertical center of mass trajectory and the vertical ground reaction force. Hum. Mov. Sci. 32 (2), 279–289.

Inanami, H., 1991. Iwai Orthopedic Hospital rehabilitation program. Paper Presented at International Spinal Rehabilitation Update 1991 Symposium, Daytona.

Katz, J.N., 2006. Lumbar disc disorders and low back pain: socioeconomic factors and consequences. J. Bone Joint Surg. Am. 88 (Suppl. 2), 21–24.

Kerrigan, D.C., Della Croce, U., Marciello, M., Riley, P.O., 2000. A refined view of the determinants of gait: significance of heel rise. Arch. Phys. Med. Rehabil. 81 (8), 1077–1080.

Krebs, D.E., Wong, D., Jevsevar, D., O'Riley, P., Hodge, W.A., 1992. Trunk kinematics during locomotor activities. Phys. Ther. 72 (7), 505–514.

Kuo, A.D., 2007. The six determinants of gait and the inverted pendulum analogy: a dynamic walking perspective. Hum. Mov. Sci. 26 (4), 617–656.

Lamoth, C.J., Meijer, O.G., Wuisman, P.I., van Dieen, J.H., Levin, M.F., Beek, P.J., 2002. Pelvis–thorax coordination in the transverse plane during walking in persons with non-specific low back pain. Spine 27 (4), E92–E99.

Lamoth, C.J., Daffertshofer, A., Meijer, O.G., Lorimer Moseley, G., Wuisman, P.I., Beek, P.J., 2004. Effects of experimentally induced pain and fear of pain on trunk coordination and back muscle activity during walking. Clin. Biomech. 19 (6), 551–563.
Lamoth, C.J., Meijer, O.G., Daffersthofer, A.D., Wuisman, P.I., Beek, P.J., 2006a. Effects of chronic low back pain on trunk coordination and back muscle activity during walking: changes in motor control. Eur. Spine J. 15, 23–40.
Lamoth, C.J., Daffertshofer, A.D., Meijer, O.G., Beek, P.J., 2006b. How do persons with chronic low back pain speed up and slow down? Trunk–pelvis coordination and lumbar erector spinae activity during gait. Gait Posture 23 (2), 230–239.
Lamoth, C.J., Stins, J.F., Pont, M., Kerckhoff, F., Beek, P.J., 2008. Effects of attention on the control of locomotion in individuals with chronic low back pain. J. Neuroeng. Rehabil. 25 (5), 13.
Lin, Y., Gfoehler, M., Pandy, M.G., 2014. Quantitative evaluation of the major determinants of human gait. J. Biomech. 47 (6), 1324–1331.
Maniadakis, N., Gray, A., 2000. The economic burden of back pain in the UK. Pain 84 (1), 95–103.
National Institute for Health and Clinical Excellence, 2009. Low Back Pain: Early Management of Persistent Non-Specific Low Back Pain. Royal College of General Practitioners, London.
National Research Council, 1998. Work-Related Musculoskeletal Disorders: A Review of the Evidence. National Academy Press, Washington, DC.
National Research Council, The Institute of Medicine, 2001. Musculoskeletal Disorders and the Workplace: Low Back and Upper Extremities. National Academy Press, Washington, DC.
O'Dwyer, N., Smith, R., Kalaki, M., Rattanaprasert, U., 2009. Independent assessment of pattern and offset variability of time series waveforms. Gait Posture 29, 285–289.
Ogon, M., Krismer, M., Sollner, W., Kantner-Rumplmair, W., Lampe, A., 1996. Chronic low back pain measurement with visual analogue scales in different settings. Pain 64 (3), 425–428.
Pollock, M.L., Graves, J.E., Leggett, S.H., Young, W.G., Gazzarella, L., Carpenter, D.M., 1991. Accuracy of counter weighting to account for upper body mass in testing lumbar extension strength. Med. Sci. Sports Exerc. 23, S66.
Ricci, J.A., Stewart, W.F., Chee, E., Leotta, C., Foley, K., Hochberg, M.C., 2006. Back pain exacerbations and lost productive time costs in United States workers. Spine 31 (26), 3052–3060.
Rice, J., Kaliszer, M., Walsh, M., Jenkinson, A., O'Brien, T., 2004. Movements at the low back during normal walking. Clin. Anat. 17 (8), 662–666.
Robinson, M.E., Greene, A.F., O'Connor, P., Graves, J.E., MacMillan, M., 1992. Reliability of lumbar isometric torque in patients with chronic low back pain. Phys. Ther. 72 (3), 186–190.
Roffey, D.M., Wai, E.K., Bishop, P., Kwon, B.K., Dagenais, S., 2010. Causal assessment of occupational standing or walking and low back pain: results of a systematic review. Spine J. 10 (3), 262–272.
Rowe, P.J., White, M., 1997. Three dimensional lumbar spinal kinematics during gait following mild musculoskeletal low back pain in nurses. Gait Posture 4, 242–251.
Saunders, J.B., Inman, V.T., Eberhart, H.D., 1953. The major determinants in normal and pathological gait. J. Bone Joint Surg. Am. 35, 543–558.
Schache, A.G., Bennell, K.L., Blanch, P.D., Wrigley, T.V., 1999. The coordinated movement of the lumbo-pelvic-hip complex during running: a literature review. Gait Posture 10 (1), 30–47.
Schache, A.G., Blanch, P., Rath, D., Wrogley, T., Bennell, K., 2002a. Three-dimensional angular kinematics of the lumbar spine and pelvis during running. Hum. Mov. Sci. 21, 273–293.
Schache, A.G., Blanch, P.D., Rath, D.A., Wrigley, T.V., Starr, R., Bennell, K.L., 2002b. Intra-subject repeatability of the three dimensional angular kinematics within the lumbo-pelvic-hip complex during running. Gait Posture 15, 136–145.
Seay, J.F., Van Emmerik, R.E., Hamill, J., 2011a. Influence of low back pain status on pelvis–trunk coordination during walking and running. Spine 36 (16), E1070–E1079.
Seay, J.F., van Emmerik, R.E., Hamill, J., 2011b. Low back pain status affects pelvis–trunk coordination and variability during walking and running. Clin. Biomech. 26 (6), 572–578.
Selles, R.W., Wagenaar, R.C., Smith, T.H., Wuisman, P.I., 2001. Disorders in trunk rotation during walking in patients with low back pain: a dynamical systems approach. Clin. Biomech. 16 (3), 175–181.
Steele, J., Bruce-Low, S., Smith, D., Jessop, D., Osborne, N., 2013a. A randomised controlled trial of limited range of motion lumbar extension exercise in chronic low back pain. Spine 38 (15), 1245–1252.
Steele, J., Bruce-Low, S., Smith, D., 2013b. A review of the specificity of exercises designed for conditioning the lumbar extensors. Br. J. Sports Med. (Online First).
Steele, J., Bruce-Low, S., Smith, D., 2014. A reappraisal of the deconditioning hypothesis in low back pain: a review of evidence from a triumvirate of research methods on specific lumbar extensor deconditioning. Curr. Med. Res. Opin. 30 (5), 865–911.
Stewart, W.F., Ricci, J.A., Chee, E., Morganstein, D., Lipton, R., 2003. Lost productive time and cost due to common pain condition in the US workforce. JAMA 290 (18), 2443–2454.
Stokes, V.P., Andersson, C., Forssberg, H., 1989. Rotational and translational movement features of the pelvis and thorax during adult human locomotion. J. Biomech. 22 (1), 43–50.
Syczewska, M., Oberg, T., Karlsson, D., 1999. Segmental movements of the spine during treadmill walking with normal speed. Clin. Biomech. 14, 384–388.
Thorstensson, A., Carlson, H., Zomlefer, M.R., Nilsson, J., 1982. Lumbar back muscle activity in relation to trunk movements during locomotion in man. Acta Physiol. Scand. 116, 13–20.
Thurston, A.J., Harris, J.D., 1983. Normal kinematics of the lumbar spine and pelvis. Spine 8 (2), 199–205.
Tsao, H., Hodges, P.W., 2008. Persistence of improvements in postural strategies following motor control training in people with recurrent low back pain. J. Electromyogr. Kinesiol. 18 (4), 559–567.
van Der Hoorn, W., Bruijn, S.M., Meijer, O.G., Hodges, P.W., van Dieen, J.H., 2012. Mechanical coupling between transverse plane pelvis and thorax rotations during gait is higher in people with low back pain. J. Biomech. 45, 342–347.
van Der Hulst, M., Vollenbroek-Hutten, M.M., Rietman, J.S., Schaake, L., Groothuis-Oudshoorn, K.G., Hermens, H.G., 2010a. Back muscle activation patterns in chronic low back pain during walking: a "guarding" hypothesis. Clin. J. Pain 26 (1), 30–37.
van Der Hulst, M., Vollenbroek-Hutten, M.M., Rietman, J.S., Hermens, H.J., 2010b. Lumbar and abdominal muscle activity during walking in subjects with chronic low back pain: support of the "guarding" hypothesis? J. Electromyogr. Kinesiol. 20 (1), 31–38.
van Der Hulst, M., Vollenbroek-Hutten, M.M., Schreurs, K.M., Rietman, J.S., Hermens, H.J., 2010c. Relationships between coping strategies and lumbar muscle activity in subjects with chronic low back pain. Eur. J. Pain 14 (6), 640–647.
van Der Krogt, M.M., Delp, S.L., Schwartz, M.H., 2012. How robust is human gait to muscle weakness? Gait Posture 36 (1), 113–119.
van Tulder, M.W., Koes, B.W., Bouter, L.M., 1995. A cost-of-illness study of back pain in The Netherlands. Pain 62 (2), 233–240.
Vincent, H.K., Vincent, K.R., Seay, A.N., Conrad, B.P., Hurley, R.W., George, S.Z., 2013. Back strength predicts walking improvement in obese, older adults with chronic low back pain. PM R 6 (5), 418–426.
Vogt, L., Banzer, W., 1999. Measurement of lumbar spine kinematics in incline treadmill walking. Gait Posture 9 (1), 18–23.
Vogt, L., Pfeifer, K., Portscher, M., Banzer, W., 2001. Influences of nonspecific low back pain on three-dimensional lumbar spine kinematics in locomotion. Spine 26 (17), 1910–1919.
Vogt, L., Pfeifer, K., Banzer, W., 2003. Neuromuscular control of walking with chronic low back pain. Man. Ther. 8 (1), 21–28.
Waddell, G., Burton, A.K., 2000. Occupational health guidelines for the management of low back pain at work: evidence review. Occup. Med. 51, 126–135.
Waddell, G., Feder, G., Lewis, M., 1997. Systematic reviews of bed rest and advice to stay active for acute low back pain. Br. J. Gen. Pract. 47, 647–652.
Waddell, G., Aylward, M., Sawney, P., 2002. Back Pain, Incapacity for Work and Social Security Benefits: An International Literature Review and Analysis. The Royal Society of Medicine Press Ltd, Glasgow.
Walker, B.F., 2000. The prevalence of low back pain: a systematic review of the literature from 1966 to 1998. J. Spinal Disord. 13 (3), 205–217.
White, A.A., Gordon, S.L., 1982. Synopsis: workshop on idiopathic low back pain. Spine 7, 141–149.
Whittle, M.W., Levine, D., 1999. Three-dimensional relationships between the movements of the pelvis and lumbar spine during normal gait. Hum. Mov. Sci. 18, 681–692.
Winter, D.A., 1983. Biomechanical motor patterns in normal walking. J. Mot. Behav. 15 (4), 302–330.
Winter, D.A., MacKinnon, C.D., Ruder, G.K., Wieman, C., 1993. An integrated EMG/biomechanical model of upper body balance and posture during human gait. Prog. Brain Res. 97, 359–367.
World Health Organisation, 1998. The World Health Report 1998: Life in the 21st Century: A Vision for all. Office of Publications, World Health Organisation, Geneva.