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Young individuals with a more ankle-steered proprioceptive control strategy may develop mild non-specific low back pain

Kurt Claeys [a,b,*], Wim Dankaerts [a], Lotte Janssens [a], Madelon Pijnenburg [a], Nina Goossens [a], Simon Brumagne [a]

[a] Research Group Musculoskeletal Rehabilitation, KU Leuven Department of Rehabilitation Sciences, KU Leuven, Leuven, Belgium
[b] Research Group Musculoskeletal Rehabilitation, KU Leuven Department of Rehabilitation Sciences, Campus Brugge, Brugge, Belgium

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ABSTRACT

Altered proprioceptive postural control has been demonstrated in people with non-specific low back pain (LBP). However, the cause-effect relation remains unclear. Therefore, more prospective studies are necessary.

Proprioceptive postural control of 104 subjects was evaluated at baseline using a force plate and with application of vibration stimulation on ankle and back muscles. Spinal postural angles were measured with digital photographs. Psychosocial variables and physical activity were registered using questionnaires. Ninety subjects were followed over two years concerning their LBP status, 14 were lost to follow-up.

Four distinct groups were determined after two years based on pain and disability scores: never LBP, no LBP at intake with future mild LBP, mild LBP at intake with no further LBP, LBP at intake with further episodes of mild LBP. Risk factors for developing or sustaining LBP were calculated using logistic regression analysis.

A more ankle-steered proprioceptive postural control strategy in upright standing increased the risk for developing or having recurrences of mild LBP within two years (Odds: 3.5; 95% CI: 1.1–10.8; $p < 0.05$). Increased postural sway, altered spinal postural angles, psychosocial and physical activity outcomes were not identified as risk factors for future mild LBP. These findings could contribute to improving the prevention and rehabilitation of LBP.

1. Introduction

Non-specific low back pain (LBP) is one of the most frequent musculoskeletal disorders with high rates of reoccurrence. Life time prevalence of LBP is very high and 11–12% of the people with LBP is disabled (Balague et al., 2012). As a result, LBP is a major health problem in the western society resulting in high economic costs (Carragee et al., 2005). Recently, studies on the causes and mechanisms for LBP were identified as a top primary care research priority for LBP research (Costa et al., 2013). Therefore, prospective studies need further consideration to determine potential risk factors for developing LBP.

Altered proprioceptive postural control (e.g. decreased use of lumbar proprioceptive inputs and/or increased use of ankle proprioceptive inputs during postural control) has been shown frequently in people with LBP (Brumagne et al., 2004; Claeys et al., 2011; della Volpe et al., 2006). However, a cause-result relation remains unclear because most studies were cross-sectional in design.

Indeed, until now, only few prospective studies investigated the cause-effect relation between this altered proprioceptive postural control and the development of LBP. Increased posterior pelvic tilt and larger lumbar repositioning errors during sitting were shown to increase the risk for developing LBP in nursing students (Mitchell et al., 2010). Moreover, delayed trunk muscle responses during sitting contributed to the development of LBP in college athletes (Cholewicki et al., 2005). Both prospective studies suggested proprioceptive deficits as an underlying mechanism, but a more direct evaluation of the proprioceptive system was not performed in these studies.

Besides postural control, were also psychosocial variables demonstrated to play a role in the development of LBP (Vlaeyen et al., 1995). Future serious LBP was predicted strongly by baseline

* Corresponding author at: Research Group Musculoskeletal Rehabilitation, KU Leuven Department of Rehabilitation Sciences, KU Leuven, Leuven, Belgium.

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psychosocial characteristics; i.e. fear and distress) in different working populations (Carragee et al., 2005; Hiebert et al., 2012). Moreover, in a systematic review, depression, psychological distress, passive coping and fear avoidance beliefs have also been demonstrated to contribute to the transition from acute to chronic LBP (Ramond et al., 2011). However, the role of psychosocial variables in combination with proprioceptive postural control characteristics as predictors for LBP episodes remains unclear.

In addition to psychosocial influences, the role of physical activity also remains obscure and ambiguous in the development of LBP (Heneweer et al., 2011). A U-shaped relation was found, which implies that people with moderate physical activity levels are less at risk for developing LBP compared to both people with extremely high or low physical activity levels (Heneweer et al., 2009). However, studies evaluating physical activity as a risk factor did not take postural control characteristics into account.

The aim of this prospective study was to investigate the role of specific proprioceptive use during postural control, psychosocial variables and physical activity in the development of LBP in a young population. Baseline measurements were performed to evaluate proprioceptive steering (i.e. ankle versus back proprioceptive use), postural sway, usual standing and usual sitting posture, pain, disability, physical activity and psychosocial characteristics. The LBP status was registered during a two year follow-up using questionnaires to evaluate pain and disability.

2. Materials and methods

2.1. Characteristics of the subjects

A young population of 104 university students participated voluntarily in this study. Test procedures were approved by the Medical Research Ethics Committee of KU Leuven with respect to the declaration of Helsinki (Ethical Principles for Medical Research Involving Human Subjects). All subjects gave their written informed consent. Participants were followed up over a period of two years and the incidence of LBP was registered every three months by filling out the Oswestry Disability Index (ODI-2) (Fairbank and Pynsent, 2000) and by rating their back pain on a Numerical Rating Scale (NRS) (Joos et al., 1991). During the follow-up period, subjects had to fill out four other questionnaires, every three months: a Physical Activity Index (PAI) questionnaire (Baecke et al., 1982), the Fear-Avoidance Beliefs Questionnaire (FABQ) (Waddell et al., 1993), the Four-Dimensional Symptom Questionnaire (4DSQ) (Terluin, 1998) and the Tampa Scale for Kinesiophobia (TSK) (Roelofs et al., 2004). For all questionnaires, subjects were asked to rate their average status during the last month. Table 1 gives an overview of the characteristics of the subjects at intake. During the follow-up, subjects were declared as having "no LBP" if the ODI-2 score was less than six and the NRS score was equal to zero at all of the follow-ups (Claeys et al., 2011). If any of the ODI-2 scores during the follow-up was higher than six or if the NRS score was higher than zero, subjects were classified as having "LBP". Ninety subjects completed the prospective study. Fourteen subjects decided to leave the study because they experienced filling out the questionnaires as too time-consuming. Fig. 1 shows an overview of the subjects with the drop-outs.

Table 1
Characteristics of the subjects at baseline.

Variable	Subjects with LBP Mean + SD	Subjects without LBP Mean + SD	Significance
N	43 (10 M, 33 F)	61 (12 M, 49 F)	
Age (years)	19.1 ± 1.6	19.2 ± 3.7	0.85
Height (cm)	174.0 ± 8.1	170.4 ± 7.4	0.02
Weight (kg)	65.4 ± 8.8	63.3 ± 7.6	0.20
BMI (kg/m^2)	21.5 ± 2.2	21.8 ± 2.4	0.61
ODI-2 (%)	7.0 ± 4.5	1.8 ± 2.3	0.01
NRS	2.8 ± 2.2	0	0.01

LBP = non-specific low back pain, SD = standard deviation, *N* = number of subjects, M = Male, F = Female, cm = centimeters, kg = kilogram, BMI = Body Mass Index, m = meters, ODI-2 = Oswestry Disability Index, NRS = Numerical Rating Scale for pain.

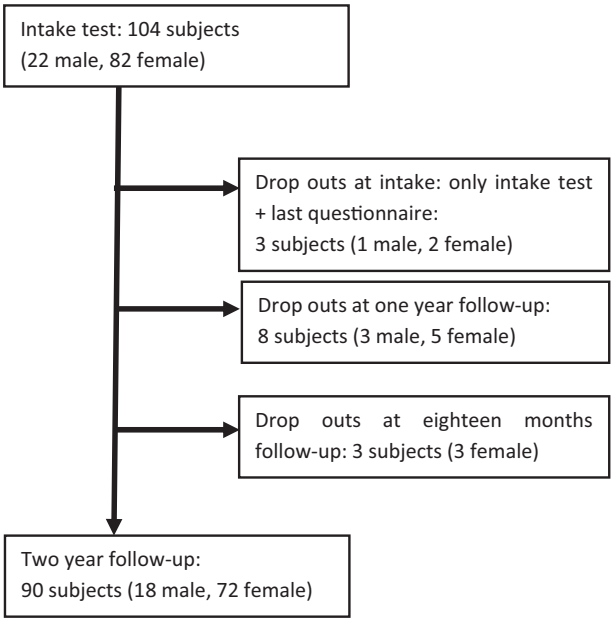

Fig. 1. Flowchart of the subjects participating in the study and drop-outs.

2.2. Postural sway, proprioceptive steering and relative proprioceptive weighting

2.2.1. Postural balance analysis

Postural sway characteristics were measured using a six channel strain gauge force plate (Bertec Corporation, Ohio, USA). Force plate data were sampled at 500 Hz using a micro 1401 data-acquisition system and Spike2 software (Cambridge Electronic Design, UK) and low pass filtered (a dual pass of a second order Butterworth filter, to create a zero-lag fourth order filter) with a cut-off frequency of 5 Hz before further data reduction and analysis.

2.2.2. Muscle vibration

Muscle vibration was used to examine the role of proprioception in postural control. Muscle vibration stimulates muscle spindles and creates a lengthening illusion of the muscles (Roll and Vedel, 1982; Vedel and Roll, 1982). Two muscle vibrators (self-manufactured with Maxon motors, Switzerland) were used. Vibration was applied bilaterally to the soleus muscles or to the lumbar multifidus muscles. These muscles represent the muscles used in an ankle-steered strategy or a multi-segmental strategy, respectively (Brumagne et al., 2008). Muscle vibration was initiated 15 s after the start of the trial for the duration of 15 s. Activation and deactivation of the vibrators were controlled manually. The frequency of the vibration was set at 60 Hz and the amplitude was approximately 0.5 mm. These characteristics of vibration were demonstrated to induce a significant muscle lengthening illusion in healthy individuals (Cordo et al., 2005). To avoid falling during muscle vibration, corrective displacements are made to compensate for the kinesthetic illusions. The amount

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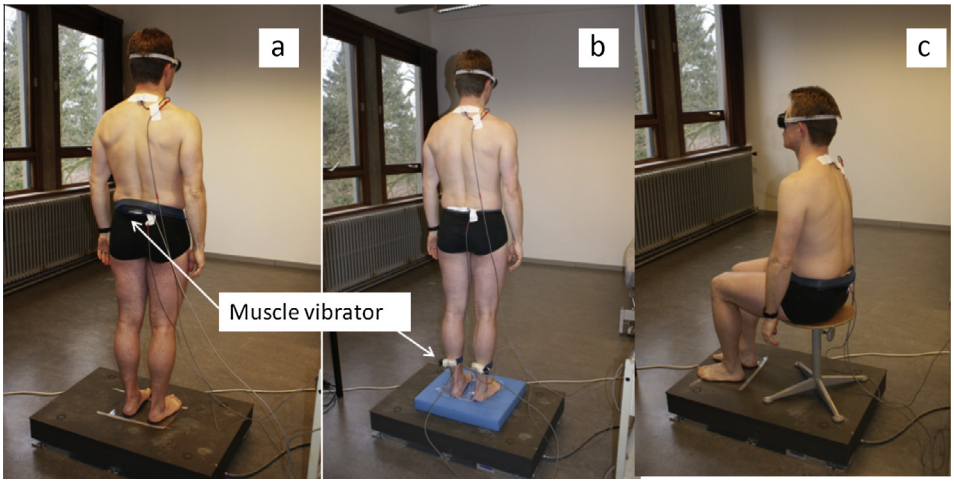

Fig. 2. Experimental set-up to investigate proprioceptive postural control: (a) standing on a stable support surface with vibration on lumbar multifidus muscles; (b) standing on an unstable support surface (foam pad) with vibration on soleus muscles; (c) sitting. All trials were performed without vision.

of directional corrective postural sway (i.e. center of pressure displacement) represented the extent to which the central nervous system used signals of the vibrated muscle for postural control. For instance, vibration of the soleus during standing can give the illusion of forward leaning and therefore the individual will compensate with a postural sway in a backward direction (i.e. dominant ankle muscles proprioceptive use) if the central nervous system mainly uses this proprioceptive afference for postural control (Brumagne et al., 2008). When vibration is applied to lumbar multifidus muscles during standing, a compensatory postural sway in a forward direction is expected (i.e. dominant back muscles proprioceptive use) if the central nervous system mainly uses this proprioceptive afference for postural control. (Brumagne et al., 2008).

2.2.3. Test procedure

A prospective study was used to investigate if proprioceptive postural control characteristics could be identified as risk factors for developing or having recurrences of LBP. To examine postural sway characteristics and proprioceptive postural control, three postural conditions were performed on the force plate: quiet standing on a stable support surface, quiet standing on an unstable support surface (foam pad) and sitting on a stool with stool and feet on the force plate, respectively (Claeys et al., 2011). The foam condition should force the subjects to rely less on soleus proprioceptive inputs which can highlight potential back proprioceptive deficits (Brumagne et al., 2008; Claeys et al., 2011). In the two standing conditions, the subjects performed four trials: quiet standing, quiet standing with ballistic arm flexion, quiet standing with soleus muscles vibration and quiet standing with multifidus muscles vibration. The subjects had to stand barefoot with the arms hanging relaxed along the body. During sitting, subjects performed three trials: usual sitting, usual sitting with soleus muscles vibration and usual sitting with multifidus muscles vibration. The sitting condition was chosen as a condition in which subjects must rely more on back muscles proprioceptive inputs instead of soleus proprioceptive inputs to control postural balance (Claeys et al., 2011). The feet position (both heels 10 cm separated, both forefeet in a free splayed out position) was marked on a transparent sheet for standardization throughout all trials (standing and sitting). In all postural balance trials, vision was occluded by means of non-transparent goggles. However, subjects had to keep their eyes open with their gaze in a straight-ahead direction. In all trials, the subjects were asked to stand or sit in their usual standing or sitting position as immobile, but relaxed as possible. All trials had a duration of one minute and were carried out at intake. Fig. 2 shows the experimental set-up of the proprioceptive postural control trials on the force plate during standing and sitting. After the postural control trials, postural angles in usual standing and sitting were evaluated with digital photographs. Questionnaires were filled out after the experimental trials. These evaluations by questionnaires were repeated every three months during two years. Table 2 gives an overview of the proprioceptive postural control tests on the force plate.

Table 2
Overview of the experimental trials to evaluate postural sway and proprioceptive postural control.

Posture: Upright standing	
Condition 1: Stable support surface	
Trial 1	Quiet Standing
Trial 2	Quiet standing, ballistic shoulder flexion at 30s
Trial 3	Quiet standing, bilateral triceps surae muscles vibration
Trial 4	Quiet standing, bilateral lumbar multifidus muscles vibration
Condition 2: Unstable support surface (foam)	
Trial 5	Quiet standing
Trial 6	Quiet standing, ballistic shoulder flexion at 30s
Trial 7	Quiet standing, bilateral triceps surae muscles vibration
Trial 8	Quiet standing, bilateral lumbar multifidus muscles vibration
Posture: Sitting on stool	
Trial 9	Sitting
Trial 10	Sitting, bilateral triceps surae muscles vibration
Trial 11	Sitting, bilateral lumbar multifidus muscles vibration

2.2.4. Postural angles

To assess the spinal posture in the sagittal plane, an experienced physiotherapist positioned manually photo-reflective markers on six anatomical landmarks of the subjects using double-sided tape as follows: spinous process of cervical vertebra C7, spinous process of thoracic vertebra T12, spinous process of lumbar vertebra L3, spinous process of the sacrum S2, anterior superior iliac spine (right side), midpoint of the greater trochanter (right side). The spinal posture was evaluated in the sagittal plane from the right side during usual standing and usual sitting. Five sagittal spinal angles were evaluated: pelvic tilt, lumbar curve, lumbar angle, thoracic flexion and trunk angle (O'sullivan et al., 2002). Subjects were asked to keep their gaze forward during the photographic assessment. To measure the angles, 2-D lateral

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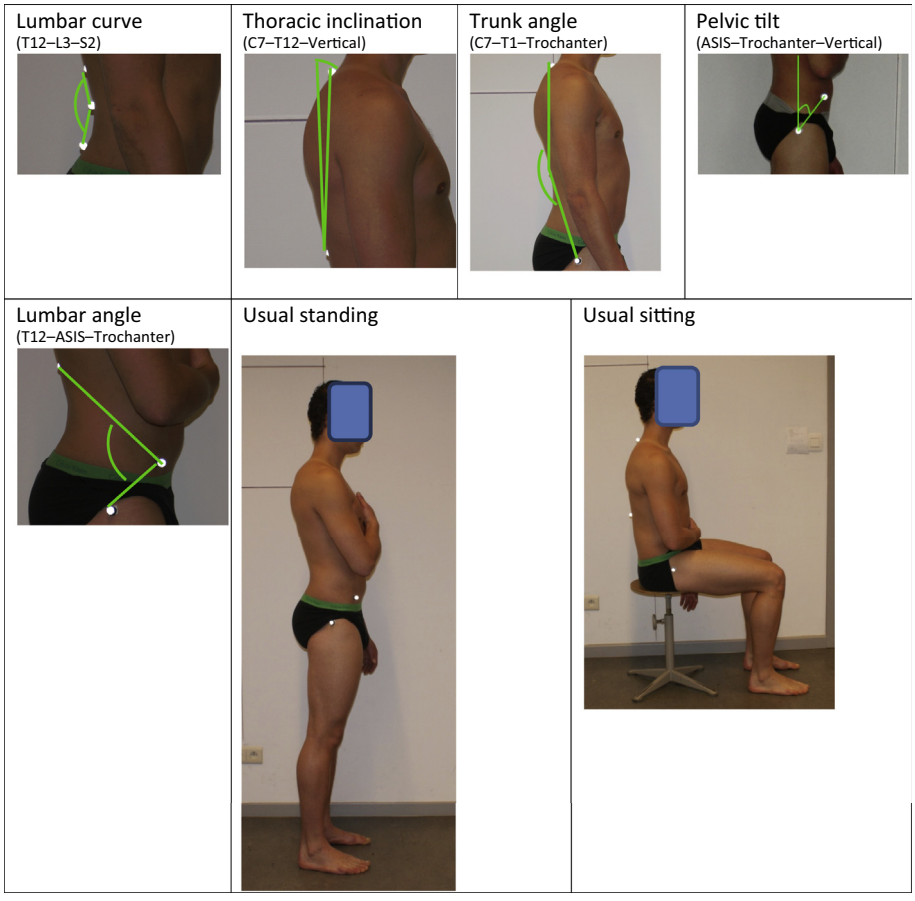

Fig. 3. Postures and the postural angles calculated in degrees.

photographs were taken with a digital photo camera (Sony A200 DSLR-A200K with lens DT18-70mm F3.5-5.6.), stabilized on a tripod with a height of 946 mm and positioned six meter away from the subject. The digital photographs were imported in an image-processing program (MatLab R2008a, MathWorks Inc., Massachusetts USA) to determine the 2D-co-ordinates (X- and Y-co-ordinates) of each bony landmark. Nine postural angles were calculated by MathCAD 14 (PTC, USA) using formulas of trigonometry. The use of these markers in combination with digital photographs has been demonstrated to be a reliable method for postural research (Cohen, 1988). The evaluated postural angles are illustrated in Fig. 3.

2.2.5. Data reduction and statistical analysis

Postural sway characteristics from the force plate readings were collected and calculated using Spike 2 (CED, Cambridge, UK) and Microsoft Excel software. Displacements of the center of pressure (COP) in anterior–posterior direction were estimated from the raw force data using the equation: COP = Mx/Fz. Root mean square (RMS) values of the COP displacements were calculated to evaluate postural sway and mean values of the COP displacements for the trials with muscle vibration were calculated to analyze the directional effect of muscle vibration on COP displacement. The COP displacements during the different trials were calculated relative to a baseline initial value calculated in usual standing or sitting, respectively. This position was a standard starting position for each subject measured separately for all trials on the force plate. During the muscle vibration trials, the COP displacements were analyzed over two periods: the 15 s preceding and the 15 s during muscle vibration. Forward COP displacement will give a positive value. Negative values correspond to backward COP displacement. To provide additional information about the proprioceptive dominance, a Relative Proprioceptive Weighting ratio (RPW) was calculated using the equation: RPW TS/LM = (absolute TS)/(absolute TS + absolute LM). In this equation, absolute TS is the absolute value of the mean COP displacement during soleus muscle vibration and absolute LM is the absolute value of the mean COP displacement during back muscle vibration. A RPW outcome of 1 corresponds to 100% reliance on soleus muscle afference. A RPW score of 0 indicates a 100% reliance on lumbar multifidus muscle afference (Brumagne et al., 2008; Claeys et al., 2011; Kiers et al., 2014).

Four different subgroups were determined after two years based on the NRS pain en ODI-2 scores described above: Group 1 consisted of people with no LBP both at intake and during the two year follow-up (NoLBP–NoLBP), Group 2 consisted of people with no LBP at intake and who developed minimum one episode of LBP during the follow-up period (NoLBP–LBP), Group 3 consisted of people with LBP at intake who had no further episode of LBP (LBP–NoLBP), Group 4 consisted of people with LBP at intake who had minimum one episode of LBP during the follow-up period (LBP–LBP).

Group differences in RMS and mean values of the COP displacements, RPW values, postural angles, PAI and psychosocial factors were analyzed using analysis of variance (ANOVA). When a significant difference was found, post hoc tests (Tukey's unequal N HSD) were performed to further analyze the detailed effects. All data are presented as mean ± standard deviation

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(SD). The level of statistical significance was set at $p < 0.05$. To determine the likelihood of developing or sustaining LBP after the intake test, logistic regression analysis was performed for the variables with statistically significant differences in the four group analysis. A Hosmer–Lemeshow goodness of fit test was performed to investigate if the model was appropriate for the variables investigated. Odds ratios and 95% confidence intervals (CI) were calculated. The statistical analysis was performed with SPSS Statistics 20 (IBM, USA).

3. Results

3.1. Characteristics of the subjects

The NoLBP–LBP group had a significantly smaller body height compared to the LBP–NoLBP group ($p = 0.05$). A significant difference on the 4DSQ Fear scale existed within the NoLBP group at intake: the NoLBP–LBP group had larger scores on the 4DSQ Fear scale compared to the NoLBP–NoLBP group ($p = 0.05$). Results of the post hoc tests also showed that the LBP–NoLBP group had significantly larger scores on the FABQ physical activity (FABQ-PA) scale compared to both the *NoLBP–NoLBP* ($p = 0.01$) and the NoLBP–LBP group ($p = 0.02$) The LBP–LBP group scored higher than the NoLBP–LBP group on the FABQ-PA scale (post hoc test: $p = 0.01$). Subject characteristics are shown in Table 3.

3.2. Postural sway, proprioceptive steering and relative proprioceptive weighting

First, no significant differences between the four groups were demonstrated in the RMS scores of the COP displacements in the stable and unstable standing conditions (with and without ballistic arm flexion. Table 4 shows the RMS scores of the COP displacements. Second, a significant difference between groups in the muscle vibration trials was shown: the NoLBP–LBP group showed more reliance on ankle proprioceptive inputs in the stable standing condition compared to the NoLBP–NoLBP group (post hoc test: $p = 0.03$). Table 5 shows the results of the proprioceptive steering trials. Third, significant differences in RPW were demonstrated between groups: during standing on the unstable support surface, the NoLBP–LBP group showed significantly higher RPWvalues compared to the NoLBP–No LBP group (pots hoc test: $p = 0.04$). In addition, in the sitting condition, the LBP–NoLBP showed significantly lower RPW values compared to the LBP–LBP group (post hoc test: $p = 0.03$). Fig. 4 shows the RPW-scores of the different groups.

3.3. Postural angles

No significant differences in postural angles could be identified between the different groups ($p > 0.05$). Table 6 shows the results of the postural angles during usual standing and sitting.

Table 3
Characteristics of the four groups at baseline.

	No LBP – No LBP $n = 22$ (M = 7, F = 15)		No LBP – LBP $n = 30$ (M = 3, F = 27)		LBP – No LBP $n = 9$ (M = 2, F = 7)		LBP – LBP $n = 29$ (M = 6, F = 23)		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Significance
Age	20.5	3.8	20.5	2.0	21.0	1.9	19.9	0.9	A: 1.00; B: 0.95; C:0.92; D: 0.96; E: 0.93; F: 1.00
Height (cm)	172.5	6.3	168.7	7.4	176.0	10.6	172.5	6.8	A: 0.25; B: 0.62; C:1.00; **D: 0.05**; E: 0.19; F: 0.60
Weight (kg)	62.3	6.5	63.8	8.1	65.7	11.8	64.9	7.4	A: 0.91; B: 0.72; C:0.67; D: 0.93; E: 0.95; F: 0.99
BMI (kg/m^2)	20.9	1.8	22.4	2.3	21.1	2.4	21.8	2.0	A: 0.06; B: 1.00; C:0.45; D: 0.34; E: 0.66; F: 0.81
ODI-2 (%)	1.0	1.4	2.3	2.8	6.8	3.6	7.6	4.8	A: 0.54; **B: 0.00; C:0.00; D: 0.01; E: 0.00**; F: 0.93
NRS NSLBP	0.0	0.0	0.0	0.0	3.6	2.2	2.7	2.3	A: 1.00; **B: 0.00; C:0.00; D: 0.00; E: 0.00**; F: 0.47
4DSQ Distress	4.4	3.3	7.0	6.6	5.2	3.9	8.3	5.0	A: 0.29; B: 0.98; **C:0.05**; D: 0.80; E: 0.78; F: 0.41
4DSQ Depression	0.1	0.2	0.6	1.0	0.0	0.0	0.3	0.8	A: 0.10; B: 1.00; C:0.55; D: 0.21; E: 0.68; F: 0.64
4DSQ Fear	0.6	1.2	2.9	4.4	1.8	1.2	2.0	2.1	A: **0.05**; B: 0.75; C:0.34; D: 0.77; E: 0.70; F: 1.00
4DSQ Somatisation	4.2	3.6	5.7	4.7	4.6	4.4	6.6	4.2	A: 0.63; B: 1.00; C:0.22; D: 0.90; E: 0.84; F: 0.60
FABQ Physical activity	6.9	5.7	4.3	4.5	10.1	5.4	9.4	5.0	A: 0.29; B: 0.40; C:0.32; **D: 0.02; E: 0.00**; F: 0.99
FABQ Work	2.2	5.9	2.6	5.2	4.7	8.5	5.3	6.7	A: 1.00; B: 0.76; C:0.32; D: 0.82; E: 0.35; F: 0.99
TSK	32.2	4.8	32.0	4.8	34.8	4.4	34.4	5.4	A: 1.00; B: 0.57; C:0.41; D: 0.47; E: 0.25; F: 1.00
PAI Work	2.1	0.4	2.1	0.4	2.0	0.3	1.9	0.5	A: 1.00; B: 0.97; C:0.54; D: 0.95; E: 0.42; F: 0.95
PAI Sports	3.3	0.6	3.4	0.5	2.6	1.2	3.1	0.6	A: 0.96; B: 0.10; C:0.75; D: 0.30; E: 0.35; F: 0.33
PAI Leisure Time	2.8	0.4	3.1	0.6	2.4	1.0	2.8	0.5	A: 0.39; B: 0.47; C:1.00; D: 0.36; E: 0.44; F: 0.33
PAI Total Score	7.7	2.0	8.5	0.8	7.1	2.1	7.8	1.0	A: 0.22; B: 0.62; C:1.00; **D: 0.04**; E: 0.22; F: 0.52

LBP = non-specific low back pain, n = number, SD = standard deviation, cm = centimeters, kg = kilogram, BMI = Body Mass Index, m = meters, NRS = Numerical Rating Scale for pain, 4 DSQ = Four-dimensional symptom questionnaire, FABQ = Fear Avoidance Beliefs Questionnaire, TSK = Tampa Scale for Kinesiophobia, PAI = Physical Activity Index (Baecke), 'Significance' shows the results of the post hoc test: A: No LBP – No LBP vs. No LBP – LBP, B: No LBP – No LBP vs. LBP – No LBP, C: No LBP – No LBP vs. LBP – LBP, D: No LBP – LBP vs. LBP – No LBP, E: No LBP –LBP vs. LBP – LBP, F: LBP – No LBP vs. LBP – LBP.
Bold values in the right column are the significance values.

Table 4
Root Mean Square (RMS) values of the center of pressure displacement (in meter) of the postural robustness trials at baseline.

	No LBP – No LBP $n = 22$ (M = 7, F = 15)		No LBP – LBP $n = 30$ (M = 3, F = 27)		LBP – No LBP $n = 9$ (M = 2, F = 7)		LBP – LBP $n = 29$ (M = 6, F = 23)		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Significance
RMS US	0.014	0.005	0.013	0.004	0.017	0.003	0.013	0.004	A: 0.97; B: 0.30; C: 0.93; D: 0.14; E: 1.00; F: 0.11
RMS US with ballistic arm flexion	0.015	0.006	0.015	0.004	0.019	0.007	0.015	0.004	A: 0.98; B: 0.13; C: 0.99; D: 0.20; E: 1.00; F: 0.16
RMS US foam	0.035	0.007	0.034	0.007	0.037	0.008	0.033	0.007	A: 0.96; B: 0.91; C: 0.85; D: 0.70; E: 0.99; F: 0.57
RMS US with ballistic arm flexion foam	0.031	0.005	0.031	0.006	0.034	0.007	0.030	0.007	A: 1.00; B: 0.69; C: 0.76; D: 0.72; E: 0.63; F: 0.25

LBP = non-specific low back pain, n = number, SD = standard deviation, RMS = Root Mean Square of the center of pressure displacement, US = usual standing, 'Significance' shows the results of the post hoc test: A: No LBP – No LBP vs. No LBP – LBP, B: No LBP – No LBP vs. LBP – No LBP, C: No LBP – No LBP vs. LBP – LBP, D: No LBP – LBP vs. LBP – No LBP, E: No LBP –LBP vs. LBP – LBP, F: LBP – No LBP vs. LBP – LBP.

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Table 5
Mean values of the center of pressure displacement (in meter) during the muscle vibration trials at baseline.

	No LBP – No LBP n = 22 (M = 7, F = 15)		No LBP – LBP n = 30 (M = 3, F = 27)		LBP – No LBP n = 9 (M = 2, F = 7)		LBP – LBP n = 29 (M = 6, F = 23)		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Significance
US MV soleus	−0.085	0.051	−0.121	0.043	−0.112	0.036	−0.106	0.046	**A: 0.03**; B: 0.43; C: 0.37; D: 0.96; E: 0.58; F: 0.98
US MV multifidus	0.027	0.019	0.036	0.021	0.045	0.036	0.031	0.025	A: 0.51; B: 0.24; C: 0.91; D: 0.78; E: 0.87; F: 0.46
US MV soleus foam	−0.039	0.022	−0.057	0.035	−0.041	0.017	−0.050	0.030	A: 0.13; B: 1.00; C: 0.54; D: 0.47; E: 0.79; F: 0.85
US MV multifidus foam	0.050	0.025	0.045	0.028	0.042	0.021	0.046	0.028	A: 0.91; B: 0.85; C: 0.92; D: 0.98; E: 1.00; F: 0.98
USit MV soleus	0.001	0.002	0.001	0.004	0.002	0.004	0.000	0.003	A: 0.86; B: 0.95; C: 0.78; D: 0.65; E: 1.00; F: 0.58
USit MV multifidus	−0.016	0.009	−0.017	0.010	−0.018	0.005	−0.016	0.012	A: 0.97; B: 0.98; C: 1.00; D: 1.00; E: 0.99; F: 0.99

LBP = non-specific low back pain, n = number, SD = standard deviation, RMS = Root Mean Square of the center of pressure displacement, US = usual standing, Usit = usual sitting, MV = muscle vibration, 'Significance' shows the results of the post hoc test: A: No LBP – No LBP vs. No LBP – LBP, B: No LBP – No LBP vs. LBP – No LBP, C: No LBP – No LBP vs. LBP – LBP, D: No LBP – LBP vs. LBP – No LBP, E: No LBP –LBP vs. LBP – LBP, F: LBP – No LBP vs. LBP – LBP.
Bold values in the right column are the significance values.

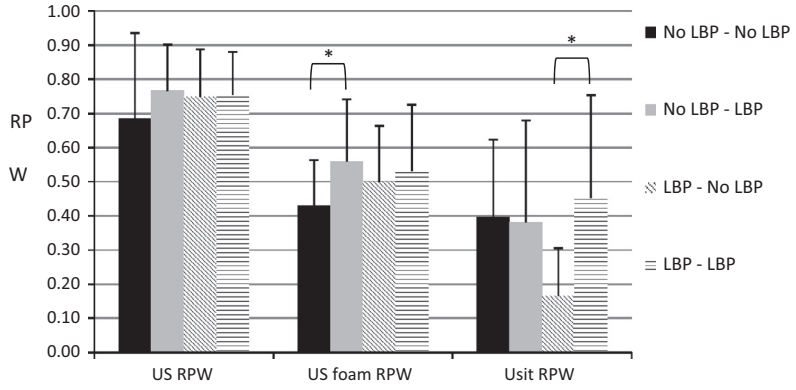

Fig. 4. Relative proprioceptive weighting scores of the subjects at intake. RPW = Relative Proprioceptive Weighting, No LBP – No LBP = people with no LBP both at intake and during the two year follow-up, No LBP – LBP = people with no LBP at intake and who developed minimum one episode of LBP during the follow-up period, LBP – No LBP = people with LBP at intake who had no further episode of LBP, LBP – LBP = people with LBP at intake who had minimum one episode of LBP during the follow-up period.

Table 6
Postural angles at baseline (in degrees).

	No LBP – No LBP n = 22 (M = 7, F = 15)		No LBP – LBP n = 30 (M = 3, F = 27)		LBP – No LBP n = 9 (M = 2, F = 7)		LBP – LBP n = 29 (M = 6, F = 23)		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Significance
Lumbar Curve US	155.6	18.2	149.7	15.0	156.8	5.8	156.5	9.5	A: 0.56; B: 1.00; C: 1.00; D: 0.54; E: 0.37; F: 1.00
Thoracic Flexion US	8.2	15.5	7.0	11.9	3.7	1.9	6.9	11.5	A: 0.99; B: 0.83; C: 1.00; D: 0.91; E: 1.00; F: 0.92
Trunk Angle US	202.5	5.2	206.9	6.0	202.4	8.4	209.7	17.4	A: 0.68; B: 1.00; C:0.25; D: 0.76; E: 0.85; F: 0.37
Pelvic Tilt US	37.0	9.4	38.7	7.9	38.4	3.3	35.6	12.2	A: 0.94; B: 0.98; C: 0.96; D: 1.00; E: 0.66; F: 0.87
Lumbar Angle US	97.3	13.0	92.7	15.5	94.4	3.8	95.6	18.4	A: 0.76; B: 0.97; C: 0.98; D: 0.99; E: 0.91; F: 1.00
Lumbar Curve Usit	170.6	13.5	165.5	36.4	167.8	19.3	172.3	5.3	A: 0.93; B: 1.00; C: 1.00; D: 1.00; E: 0.81; F: 0.97
Thoracic Flexion Usit	16.2	16.5	15.6	25.0	15.3	13.4	14.4	11.5	A: 1.00; B: 1.00; C: 0.99; D: 1.00; E: 1.00; F: 1.00
Trunk Angle Usit	225.5	5.8	228.5	4.8	222.5	20.4	225.8	14.0	A: 0.88; B: 0.93; C: 1.00; D: 0.57; E: 0.87; F: 0.89
Pelvic Tilt Usit	20.9	15.8	18.9	13.7	18.2	22.2	20.0	13.5	A: 0.98; B: 0.97; C: 1.00; D: 1.00; E: 0.99; F: 1.00
Lumbar Angle USit	125.2	13.2	119.5	10.1	123.0	13.4	113.4	31.9	A: 0.86; B: 1.00; C: 0.34; D: 0.98; E: 0.76; F: 0.66

LBP = non-specific low back pain, n = number, SD = standard deviation, US = usual standing, Usit = usual sitting, 'Significance' shows the results of the post hoc test: A: No LBP – No LBP vs. No LBP – LBP, B: No LBP – No LBP vs. LBP – No LBP, C: No LBP – No LBP vs. LBP – LBP, D: No LBP – LBP vs. LBP – No LBP, E: No LBP –LBP vs. LBP – LBP, F: LBP – No LBP vs. LBP – LBP.

3.4. Predictors of future LBP

The logistic regression model contained the nine variables with statistically significant difference in the four group analysis. This model classified 78.4% of the individuals correctly with a sensitivity (true positives) of 91.5% and a specificity (true negatives) of 51.7%. The Hosmer–Lemeshow goodness of fit test ($p > 0.05$) showed that the model was able to predict future LBP. The only significant predictor was 'more reliance on ankle muscle afference in stable standing' ($p < 0.05$; Odds 3.5; 95% CI: 1.1–10.8). Results are shown in Table 7.

4. Discussion

The main finding of this study was that people who show larger COP displacements during muscle vibration on soleus muscles in a stable standing condition are at risk for developing or sustaining mild LBP within two years. In contrast, increased postural sway, postural differences in usual standing and sitting, psychological variables and physical activity level were not demonstrated as risk factors for LBP in this young population.

No different scores in RMS-values of the COP during the stable and unstable standing trials were observed between the four

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Table 7
Logistic regression predicting NSLBP during follow-up.

	B	S.E.	Wald	df	Sig.	Odds ratio	95% C.I. for odds ratio	
							Lower	Upper
Height	−.989	.565	3.065	1	.08	.4	.123	1.125
PAI sports	−.116	.616	.035	1	.85	.9	.267	2.978
PAI leisure time	.468	.616	.577	1	.45	1.6	.477	5.346
PAI total score	.691	.650	1.128	1	.29	2.0	.558	7.139
4DSQ fear	.843	.579	2.123	1	.15	2.3	.747	7.227
FABQ physical activity	−.011	.554	.000	1	.98	1.0	.334	2.930
US MV soleus	**1.246**	**.578**	**4.647**	**1**	**.03**	**3.5**	**1.120**	**10.791**
US foam RPW	.787	.523	2.264	1	.13	2.2	.788	6.121
Usit RPW	.857	.573	2.239	1	.14	2.4	.767	7.246

PAI = Physical Activity Index, 4DSQ = Four-Dimensional Symptom Questionnaire, FABQ = Fear Avoidance Beliefs Questionnaire, US = usual standing, MV = muscle vibration, RPW = relative proprioceptive weighting, Usit = usual sitting.

groups, which is in contrast with previous studies (Brumagne et al., 2008; della Volpe et al., 2006; Mok et al., 2004). The low pain and disability scores of the current study population may explain these findings. Subjects were classified as having LBP if the NRS-pain score was more than zero or when the score on the ODI-2 was more than six percent. Consequently, the pain and disability scores of the participants may be clinically too mild to alter the postural sway. Previous studies demonstrating an increased postural sway in people with LBP included patients with higher pain and/or disability scores than in the current study (della Volpe et al., 2006; Mok et al., 2004; Popa et al., 2007). Moreover, the postural tasks used in the current study might not have been challenging enough to appraise postural sway differences between groups.

Although no differences were found in RMS-scores of the COP in the standing postural trials between the four groups, some noteworthy differences in proprioceptive use during postural control need further attention. The NoLBP–LBP group showed larger COP displacements during soleus muscles vibration while standing on a stable support surface compared to the NoLBP–NoLBP group. This finding indicates that the NoLBP–LBP group relied more on soleus muscle proprioceptive afference for stable standing postural control compared to the NoLBP–NoLBP group. Moreover, during standing on an unstable surface, higher RPW values were demonstrated in the NoLBP–LBP group compared to the NoLBP–NoLBP group. The RPW ratio is the proportion of the COP-displacements during soleus muscle vibration and multifidus muscle vibration, respectively. As a result, the NoLBP–LBP group showed an increased reliance on ankle proprioceptive signals compared to back muscle proprioceptive signals for standing postural control compared to the NoLBP–NoLBP group. This is in agreement with the results of previous cross-sectional studies which demonstrated a decreased reliance on back muscle proprioceptive inputs for standing postural control in people with LBP (Brumagne et al., 2008).

In contrast to these previous cross-sectional studies, the current study was a prospective analysis that identified differences at baseline within the proprioceptive system between healthy people developing LBP and healthy people remaining LBP-free. Indeed, people with a clearly dominant ankle proprioceptive use in stable standing showed an almost four times higher OR to develop LBP in the future. This indicates that, in this young population with mild LBP, LBP may be caused by a different proprioceptive weighting and that the observed differences in proprioceptive postural control were not only the result of pain which was frequently suggested in earlier cross-sectional studies (Brumagne et al., 2004; Brumagne et al., 2008; della Volpe et al., 2006). High reliance on ankle muscle proprioceptive inputs and the reduced ability to adapt this proprioceptive use when necessary (e.g. on foam) in people who develop LBP may result in a less fine-tuned spinal control during postural tasks. This may increase the mechanical stress on the lumbar spine which could lead to spinal injury and pain (Cholewicki et al., 2005).

These findings could be confirmed by the study of Marshall et al. (2009) who state that delayed trunk muscle reflexes in people with functional ankle instability predispose these patients for developing LBP. As a result, neural or neuromuscular adaptations (with possible proprioceptive alterations as underlying mechanism) at the trunk may not only be associated with spinal problems but may also play a role in other musculoskeletal disorders such as functional ankle instability.

The LBP–NoLBP group showed significantly lower RPW values during sitting compared to the LBP–LBP group. This indicates that people with LBP who use more back muscle proprioceptive signals for sitting postural control are more likely to become LBP-free in the near future (two years) compared to those who use less back muscle proprioceptive afference during sitting. However, it must be noticed that the LBP–NoLBP group is very small (nine subjects) compared to the other groups and thus these findings must be interpreted with caution.

Despite the clearly demonstrated proprioceptive postural control differences during standing and sitting, postural differences between groups were not demonstrated in the current study. These findings are in agreement with previous studies which could not demonstrate postural differences in usual standing and sitting between people who develop LBP and healthy controls (Mitchell et al., 2010; Mitchell et al., 2009). Symptoms in the current study may be too mild to be associated with postural differences between groups. Moreover, the studies that have been demonstrating postural differences subclassified their patients based on aggravating movements and postures (Dankaerts et al., 2006).

Fear and fear-avoidance beliefs may be ruled out as a risk factor in the development of mild LBP in this young population based on the current results. Patients with scores lower than 14 on the FABQ-PA were not demonstrated to be significantly more at risk for developing LBP in the first six months (George et al., 2008). Accordingly, the fairly low fear-avoidance beliefs in the current study may not result in future LBP, since the FABQ-PA scores were not higher than 10.1.

Some studies already demonstrated larger body height as a risk factor for future LBP (Coeuret-Pellicer et al., 2010; Hershkovich et al., 2013). This could not be confirmed by the current study since the variable height was not significant in the regression analysis. Moreover, in the current study, the largest body height was shown in the LBP–NoLBP group, which should indicate that a smaller body height would be a risk for developing LBP.

Altogether, a different proprioceptive control as an underlying mechanism in the development of mild LBP in this young population becomes more explicit. Despite the evaluation of psychosocial factors, physical activity level, postural angles and postural sway

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variables, only a strategy with dominant use of ankle proprioceptive signals during postural control could be identified as a clear risk factor for developing LBP during two year follow-up in this young population. Moreover, the fact that the level of LBP in the current study was very mild further emphasizes the role of the proprioceptive system as an underlying mechanism; already very mild symptoms could be predicted by evaluation of proprioceptive control during standing. These results may clarify the findings from Nelson-Wong and Callaghan (2014). They showed that transient LBP during a prolonged standing task in asymptomatic people predicts future clinical LBP. Neuromuscular changes are hypothesized by these authors as a possible underlying mechanism. In our opinion, it may be plausible that these previously asymptomatic people have proprioceptive impairments during standing (similar to the group of LBP-developers of the current study) which predispose them fur future back pain.

To the best of our knowledge, the current prospective study was the first to reveal proprioceptive deficits during a postural control task that were associated with the development of mild LBP in the near future. Risk factors for the development of LBP were already demonstrated in other studies, but these studies focused on the motor output of a postural task instead of the proprioceptive inputs during the postural task. Cholewicki et al. (2005) identified delayed trunk muscle responses as a risk factor in the development of LBP and Mitchell et al. (2010) showed a greater posterior pelvic rotation during slump sitting as a risk factor. In the current study, altered proprioceptive input during postural tasks was identified as a risk factor. Moreover, differences in the proprioceptive system were specifically evaluated by means of muscle vibration and not solely hypothesized as in most other studies (Mok et al., 2007; Popa et al., 2007). This evaluation method was already used to identify proprioceptive differences during standing and sitting in people with LBP in cross-sectional studies and was recently demonstrated as reliable method to evaluate the role of the proprioceptive system during postural control (Brumagne et al., 2004; Brumagne et al., 2008; Claeys et al., 2011; Kiers et al., 2014). The current study used the same methodology to identify proprioceptive differences in healthy people developing LBP in the near future.

Consequently, the results of this study have an important relevance for the rehabilitation and prevention of LBP. Motor output (i.e. postural sway in different postural positions) and postures (i.e. sagittal postural spinal curvature) are readily available and easy to evaluate by both clinicians and researchers. Therefore, therapists are often inclined to direct the examination and treatment solely to motor output and postures. Unfortunately, the sensory input and processing of this afference, which may lead to differences in motor output and postures, are often neglected. However, the results of this study suggested that differences in proprioceptive processing may already occurred without obvious differences in motor output (e.g. postural sway) and postures, but do increase the risk for mild LBP. Possibly, the altered proprioceptive use must be present long enough to result in visible motor output differences. Thus, in addition to motor control exercises and postural rehabilitation, addressing the sensory component may be a fruitful supplement in the prevention and rehabilitation of LBP.

5. Limitations and future directions

In spite of the demonstrated proprioceptive difference between people who develop mild LBP compared to healthy controls, the total number of subjects may be an important limitation of the current study. Only 90 subjects completed the two year follow-up. One of the subgroups (LBP–NoLBP) consisted of only nine subjects. It must be noticed that equal group size may result in more statistically significant differences such as the RPW values in the regression analysis. Therefore, larger prospective studies are necessary to further underpin the novel findings of this study.

Identifying risk factors is crucial to reduce the high frequency of LBP. A dominant ankle proprioceptive use during postural control was identified as a potential risk factor by means of muscle vibration. However, this evaluation method is not entirely feasible in a clinical setting. Developing tests to identify proprioceptive steering in a more clinical setting may be a crucial step in the evaluation, prevention and more optimal rehabilitation of LBP (Brumagne et al., 2013).

Despite the specific evaluation of the proprioceptive system by means of muscle vibration, it remains unclear whether these proprioceptive postural control differences are based on different peripheral inputs (at muscle spindle level) or different sensory processing (e.g. reweighting, at brain level) or a combination of both. Future research using muscle vibration in combination with brain imaging (e.g. fMRI, NIRS) during postural control tasks may help to clarify this research question (Pijnenburg et al., 2014)

Finally, one statistical concern needs to be discussed. Because of the small sample size, the logistical regression model used in this study was not tested on an independent sample of subjects. This may reduce the ability to predict future LBP based on these results. Also the sensitivity and specificity measurements were determined by applying the regression model to the same data set that was used in its development. Future prospective studies need a larger number of test subjects to have the ability to test the regression model on an independent sample of subjects.

6. Conclusion

Increased reliance on ankle muscle proprioceptive inputs during standing on a stable support surface increases the risk to develop mild LBP in young individuals. In contrast, postural sway, postural angles, psychosocial variables and physical activity level were not associated with the development or recurrence of LBP in this young population. Therefore, addressing proprioceptive input and processing impairments may prove fruitful in the prevention and rehabilitation of LBP.

Conflict of interest

None.

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Kurt Claeys, PhD, PT, MT graduated as a physiotherapist from the University of Ghent, Belgium (1993) and received his postgraduate as an orthopedic manual therapist at the IRSK-WINGS institute Ieper (Belgium) in 2005. He completed his PhD at the Catholic University of Leuven (KU Leuven) in 2013. Dr. Claeys is a doctor-assistant at the Catholic University of Leuven (campus Brugge) Belgium. His main interest of research is into the relation between altered proprioceptive postural control and the development/maintenance/reoccurrence of non-specific low back pain. Besides he works in a private practice in Zerkegem (Belgium) as Musculoskeletal Physiotherapist.

Wim Dankaerts, PhD, PT, MT graduated as a physiotherapist from the Catholic University of Leuven (KU Leuven), Belgium (1990) and received his Post Graduate Diploma in Manipulative Physiotherapy, from Curtin University, Perth, WA (1995). He completed his PhD at Curtin University (2005). Prof. Dankaerts is an Associate Professor in Musculoskeletal Physiotherapy at the Catholic University Leuven (KU Leuven), Belgium. He also works part-time in a private practice in Tienen (Belgium) as a Musculoskeletal Physiotherapist. His main interest of research is into mechanism-based classification for Chronic Low Back Pain. He has published several papers and presented his research findings at many international conferences. He has presented many clinical workshops on the evaluations and management of chronic low back pain internationally.

Lotte Janssens, PhD, PT, graduated as a physiotherapist from the Catholic University Leuven (KU Leuven), Belgium (2008). She completed her PhD at the Catholic University of Leuven (KU Leuven) in 2013. Dr. Janssens is a doctor-assistant in Musculoskeletal Physiotherapy at the Catholic University of Leuven (KU Leuven), Belgium. Her main interests of research are inspiratory muscles fatigue, proprioceptive postural control and non-specific low back pain.

Madelon Pijnenburg, PT, graduated as a physiotherapist from the Catholic University Leuven (KU Leuven), Belgium (2010). Currently, she is completing her PhD entitled: The neural basis of lumbosacral proprioceptive impairment in recurrent low back pain.

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Nina Goossens, PT, graduated as a physiotherapist from the Catholic University Leuven (KU Leuven), Belgium (2013). Currently, she is completing her PhD entitled: Proprioceptive postural control in people with respiratory disorders and low back pain.

Simon Brumagne, PhD, PT, MT received a BS (1991), MS (1993), and PhD (1999) in Rehabilitation Sciences and Physical Therapy at the University of Leuven, Belgium. From 1999 to 2001, he was a post-doctoral fellow of the Research Council at the University of Leuven and Neurological Sciences Institute, Oregon Health & Science University, Portland, U.S.A.
He is currently Professor of Physical Therapy in the Department of Rehabilitation Sciences at the University of Leuven. In addition, he is seeing patients with spinal impairments at the outpatient department, University Hospitals Leuven, Belgium. His research interests include peripheral and central mechanisms of proprioceptive postural control in people with spinal pain, inspiratory muscle loading and therapeutic exercise.

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