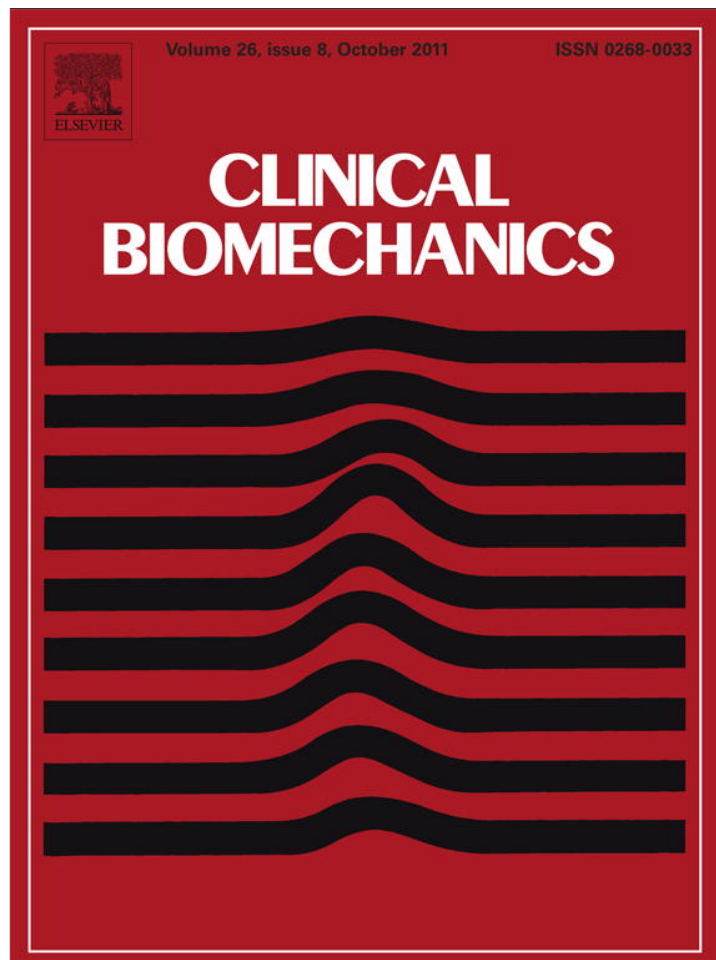


Provided for non-commercial research and education use.
Not for reproduction, distribution or commercial use.



This article appeared in a journal published by Elsevier. The attached copy is furnished to the author for internal non-commercial research and education use, including for instruction at the authors institution and sharing with colleagues.

Other uses, including reproduction and distribution, or selling or licensing copies, or posting to personal, institutional or third party websites are prohibited.

In most cases authors are permitted to post their version of the article (e.g. in Word or Tex form) to their personal website or institutional repository. Authors requiring further information regarding Elsevier's archiving and manuscript policies are encouraged to visit:

<http://www.elsevier.com/copyright>



Contents lists available at ScienceDirect

Clinical Biomechanics

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

Abdominal muscle activation increases lumbar spinal stability: Analysis of contributions of different muscle groups

Ian A.F. Stokes^{a,*}, Mack G. Gardner-Morse^a, Sharon M. Henry^b

^a Department of Orthopaedics and Rehabilitation, University of Vermont, Burlington, VT 05405, USA

^b Department of Rehabilitation and Movement Science, University of Vermont, Burlington, VT 05405, USA

ARTICLE INFO

Article history:

Received 20 February 2011

Accepted 12 April 2011

Keywords:

Abdominal muscles
Biomechanics
Stability
Rehabilitation

ABSTRACT

Background: Antagonistic activation of abdominal muscles and increased intra-abdominal pressure are associated with both spinal unloading and spinal stabilization. Rehabilitation regimens have been proposed to improve spinal stability via selective recruitment of certain trunk muscle groups. This biomechanical analytical study addressed whether lumbar spinal stability is increased by such selective activation.

Methods: The biomechanical model included anatomically realistic three-layers of curved abdominal musculature, rectus abdominis and 77 symmetrical pairs of dorsal muscles. The muscle activations were calculated with the model loaded with either flexion, extension, lateral bending or axial rotation moments up to 60 Nm, along with intra-abdominal pressure up to 5 or 10 kPa (37.5 or 75 mm Hg) and partial bodyweight. After solving for muscle forces, a buckling analysis quantified spinal stability. Subsequently, different patterns of muscle activation were studied by forcing activation of selected abdominal muscles to at least 10% or 20% of maximum.

Findings: Spinal stability increased by an average factor of 1.8 with doubling of intra-abdominal pressure. Forcing at least 10% activation of obliques or transversus abdominis muscles increased stability slightly for efforts other than flexion, but forcing at least 20% activation generally did not produce further increase in stability. Forced activation of rectus abdominis did not increase stability.

Interpretation: Based on analytical predictions, the degree of stability was not substantially influenced by selective forcing of muscle activation. This casts doubt on the supposed mechanism of action of specific abdominal muscle exercise regimens that have been proposed for low back pain rehabilitation.

© 2011 Elsevier Ltd. All rights reserved.

1. Introduction

Several differing concepts of lumbar spinal instability have been linked to causes of low back pain; here instability is defined as buckling of the spine. Spinal buckling provides a credible explanation for sudden onset back pain resulting from a 'self-injury' mechanism during sub-maximal efforts (Cholewicki and McGill, 1996). Such instability might occur if the paraspinal muscles are not successfully coordinated to prevent buckling episodes. It is known that the ligamentous spine is inherently unstable (Crisco et al., 1992), indicating that well coordinated muscle activity is required to maintain spinal stability. Spinal buckling has never been clearly documented in vivo, although cine-radiographic recording of an abnormally large intervertebral flexion associated with sudden onset pain in a weight-lifter, attributed to the subject starting in an awkward position was reported anecdotally by Cholewicki and McGill

(1992). Experiments in which spinal instability is deliberately produced and documented in living humans whose muscles are active are probably not feasible.

Since buckling instability of the spine cannot be investigated experimentally in living subjects, structural analyses have been employed to analyze possible buckling mechanisms. Spinal buckling and stability were analyzed quantitatively by Bergmark (1989) in an analysis of the lumbar spine that included short (local) and global (long) trunk muscles. Activation of muscles increases their stiffness, so antagonistic activation provides a way to increase stiffness and stability. Apparently antagonistic activation of abdominal muscles is observed in extension, lateral bending and axial rotation (Thelen et al., 1995). In addition to their presumed role in maintaining lumbar stability, activation of all of the abdominal wall muscles is associated with intra-abdominal pressurization, whose exact function is unclear but is thought to reduce spinal loading as well as to stabilize the spine (Arjmand and Shirazi-Adl, 2006; Cholewicki et al., 1999a).

The concept that abdominal wall muscle activation and associated rise in intra-abdominal pressure (IAP) increase lumbar spinal stability has been supported by experimental studies (Hodges et al., 2003) and analytical studies (Cholewicki et al., 1999a; Gardner-Morse and

* Corresponding author.

E-mail address: Ian.Stokes@uvm.edu (I.A.F. Stokes).

Stokes, 1998; Kavcic et al., 2004). Wearing a corset or belt evidently increases stability (Cholewicki et al., 1999b; Ivancic et al., 2002). However, prior analyses of the stabilizing effects of altered or augmented abdominal muscle activity have employed arbitrary values of abdominal muscle activation, and have not examined different patterns of activation or considered the relationship among abdominal wall forces and the internal pressure in a 'pressure vessel' analysis. Additionally, prior studies have not included anatomically realistic abdominal wall geometry and most have not included the transversus abdominis (TA) muscle, which is proposed to play a key role in maintaining lumbar stability (Hodges and Richardson, 1996). Since the tension in the abdominal wall is related deterministically to the intra-abdominal pressure, these two variables should be linked in any analysis, and the geometry should include curved abdominal muscle paths.

Clinically, a link has been sought between abnormal or impaired function of 'stabilizing' muscles and the onset and persistence of low back pain, and rehabilitation programs have been developed to target these muscles. Observations of a decrease in cross sectional area of the multifidus muscles (Barker et al., 2004; Hides et al., 2008; Hodges et al., 2006), altered activation of abdominal wall muscles during anticipatory postural adjustments (Hodges and Richardson, 1996; O'Sullivan et al., 1998) and delayed trunk muscular responses during automatic postural responses (Radebold et al., 2000, 2001; van Dieën et al., 2003) in people with low back pain have provided a rationale for the development of a specific lumbar stabilization exercise approach to encourage people with low back pain to activate deep abdominal and multifidus muscles preferentially. The selective training of the transversus abdominis (TA) muscle ('specific stabilization exercises') has been recommended (Costa et al., 2009; O'Sullivan, 2000; O'Sullivan et al., 1997; Richardson et al., 2004). The rationale for the targeted voluntary activation of the deep abdominals (namely the TA and internal oblique muscles) is based on a theory that purports: (1) these deep abdominal muscles play a unique role in contributing to lumbar stability, (2) motor control impairments of these muscles contribute to spinal instability and (3) stability and control of the spine are altered in people with low back pain. The goal of the specific stabilization exercise regimen is to ameliorate specific abdominal muscle impairments (e.g., delayed TA activation), restoring the ability of these muscles to provide lumbar joint protection and therefore lumbar stabilization (Richardson et al., 2004). Stabilization exercises have been criticized because it is unclear that certain muscles, such as the TA, are more important for stabilization of the spine than other muscles (Lederman, 2010; McNeill, 2010). Additionally, a mechanical analysis of spinal stability associated with 'brace' or 'hollow' abdominal muscle activation strategies (Grenier and McGill, 2007) did not support this exercise approach.

The purpose of the present study was to examine the effects of differing abdominal muscle activation patterns on spinal stability in an analytical model. The model was used to simulate the forced activation of each of the three components of the abdominal wall (the transversus, the internal and external obliques, and rectus abdominis, bilaterally) by setting their minimum activation to either 10% or 20% of maximum activation. The findings were used to test the hypothesis that the stability of the lumbar spinal column would be increased in each case by forcing muscle activation to exceed 10% or 20%.

2. Methods

A biomechanical model of the spine and its musculature (Stokes et al., 2010) was employed to analyze spinal stability during generation of moments (efforts) about the trunk with differing patterns of abdominal muscle activation. This biomechanical model included 111 symmetrical pairs of muscle 'slips' (77 pairs of dorsal muscle slips including psoas, 11 pairs each of internal oblique, external oblique and transversus abdominis, and one pair representing rectus abdominis),

and 5 lumbar vertebra (between the fixed pelvis and rigid thorax). Vertebrae were linked by flexible intervertebral joints whose stiffness in six degrees of freedom was specified (Gardner-Morse and Stokes, 2004). Published values of muscle cross-sectional areas and active and passive stiffness properties were assigned (Stokes et al., 2010). The diaphragm and pelvic floor were assumed to be rigid (isometric) and the diaphragm was attached to a rigid thorax, hence details of their structure and deformations were not included in the analyses (Fig. 1).

The model was loaded in turn with flexion, extension, lateral bending or axial rotation moments in increments of 20 Nm up to 60 Nm, along with IAP increasing in the same proportional steps up to 5 or 10 kPa (37.5 or 75 mm Hg) and partial bodyweight (340 N) (Schultz, 1990). These loading conditions corresponded to a person attempting to generate an effort opposing the externally applied moment, and with IAP increasing linearly with the magnitude of the external effort. Any complex quasi-static task involving the trunk can be considered as an effort generating forces and moments on the trunk that can be partitioned into three forces and three moments. Forces (other than superimposed bodyweight) were not analyzed here separately since their effect is generally small compared with that of the moments. The magnitudes of pure moments (up to 60 Nm) about each of the principal axes were equal to average maximum efforts reported for women making maximum voluntary efforts in axial rotation (other effort directions can generate higher moments, Stokes and Gardner-Morse, 1995). The muscle activations were calculated in an optimization scheme by which both muscle stress and stretch were minimized (Stokes and Gardner-Morse, 2001; Stokes et al., 2010) via a cost function. The optimization scheme respected that muscle tension was between zero and a physiological maximum corresponding to 0.46 MPa and was subjected to constraints on relative intervertebral displacements and muscle stresses (Stokes et al., 2010). In the Baseline analyses no other constraints were imposed on muscle activation.

Subsequently, different patterns of abdominal muscle activation were studied by forcing at least 10% or 20% of maximum activation of

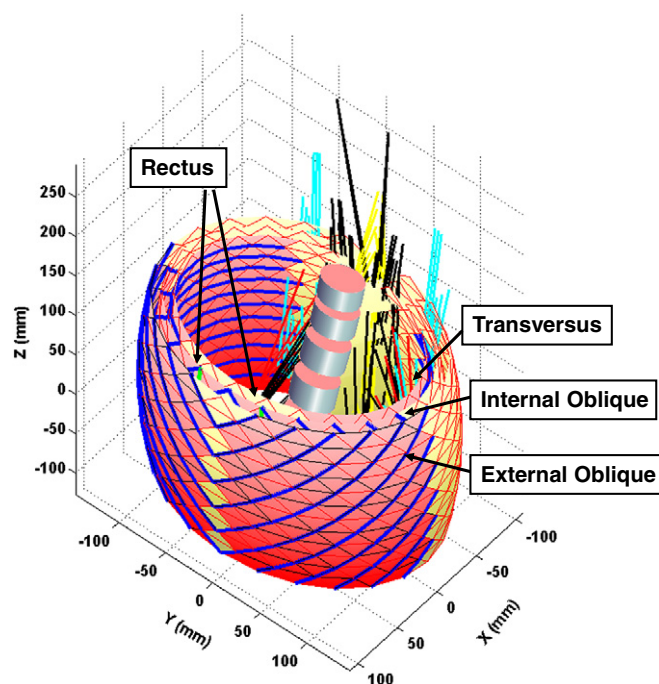


Fig. 1. Geometry of the model, showing the lumbar spine, dorsal muscles and three layers of abdominal muscles. The vertebrae are shown symbolically as cylinders to indicate their position only.

either (1) transversus abdominis or (2) the internal and external obliques, or (3) rectus abdominis, by setting the desired level of activation as a lower bound in the calculation of muscle forces while the activations of the other abdominal muscles were not constrained. In these analyses the intra-abdominal pressure was held at the same previously prescribed levels (increasing in steps proportional to the moment effort up to 5 or 10 kPa).

After solving for the muscle forces to achieve static equilibrium in each loading case, an analysis of spinal buckling was performed. The activation dependent stiffness of the 111 pairs of muscles was increased by a factor of four to represent 'short range stiffness', using $q = 4$ in the expression $stiffness = q \times muscle\ force / muscle\ length$ as proposed by Bergmark (1989), and as used by others (Crisco and Panjabi, 1991; Gardner-Morse et al., 1995) in stability analyses. Static condensation (McGuire and Gallagher, 1979; Paz, 1985) was used to limit the number of analyzed buckling modes to the 36 degrees of freedom of the spine (i.e. to exclude possible buckling of the abdominal wall). Potential buckling was determined by finding the eigenvalues of the combined elastic and geometric stiffness matrix of the model (Bergmark, 1989). The stiffness matrix was assembled by including the stiffness of the spinal motion segments and of the muscles (Gardner-Morse et al., 1995). The value of the smallest eigenvalue in this buckling analysis was used as a measure of the spine's stability. The spine would be unstable if the lowest eigenvalue was less than zero, metastable if the lowest eigenvalue equaled zero and stable if the lowest eigenvalue was greater than zero. In each loaded state, the buckling mode shape for each eigenvalue was specified by the corresponding eigenvector. The mode shape corresponding to the smallest eigenvalue (the most likely buckling mode) was examined in each loading state.

Sensitivity analyses were performed with altered geometrical and muscle activation-dependent stiffness parameters. The purpose of these analyses was to test the robustness of the analyses, rather than to investigate effects of differences in anatomy, etc., representative of variations in the human population. In the 'Baseline' model and models with forced muscle activation the model parameters were set to the presumed correct values. In the sensitivity analyses these parameters were changed systematically to evaluate the effects on the model stability. To test for sensitivity to abdominal wall geometry, the inclinations of the oblique muscles were changed to first to 37.5° and then to 53° relative to the horizontal (compared to 45.5° in the 'Baseline' model). The amount of abdominal wall bulge was set to 0 or 15 mm (compared to 10 mm in the Baseline model). The activation-dependent muscle stiffness parameter q in the expression $stiffness = q \times muscle\ force / muscle\ length$ was set to either 2 or 8 (it was 4 in the Baseline model).

3. Results

The 'Baseline' model predicted that the spine was increasingly stable with increased IAP (see Table 1). The spinal stability as measured by the smallest eigenvalue was on average 1.8 times greater with doubling of the IAP (from 5 kPa to 10 kPa at the 60 Nm effort, and pro-rated at lesser efforts), and was always stable at both pressurizations of the 'Baseline' model.

With forced minimum 10% activation of either the obliques or transversus muscles the spinal stability generally increased only slightly for efforts other than flexion, but forced activation of rectus reduced the stability in more cases than it increased stability. A further increase to minimum 20% activation did not produce further increase in stability (Table 1). In half of the 144 cases with forced activation of abdominal muscles listed in Table 1 there was an increase in stability relative to the 'Baseline' model. However, the mean change in the stability (as measured by the smallest eigenvalue) was generally negative (indicating a reduction in stability). The mean change was positive, indicating an increase in stability, for forced activation of the obliques to at least 20% of maximum activation, and at least 10% activation with the lower intra-abdominal pressure. With respect to the direction of the external effort, lateral bending and extension efforts were associated with increases in stability with forced muscle activation, while the opposite was observed for the flexion and axial rotation efforts.

The buckling modes observed for each of the loading conditions involved predominantly a single vertebra that was displaced and rotated, which is a buckling mode that might be associated with local tissue injury (e.g. Fig. 2).

In the 'Baseline' model, activation of the abdominal muscles was predicted in all four effort directions, with activation magnitudes comparable with reported values from electromyographic (EMG) studies (Grew, 1980; Mairiaux and Malchaire, 1988). The greatest activation at the 60 Nm effort magnitude was for the flexion effort with activation of 22% for external obliques and 34% for internal obliques although rectus and transversus muscles were not activated in this case (Table 2). Then, forcing activation of all the elements of respectively the obliques, transversus and rectus to at least 10% or 20%, produced an alteration in the distribution of muscle activation between these groups. With these forced selective activation of abdominal wall muscles there were generally small reductions in the activation of the other abdominal muscles. Averaged over the muscles whose activity was not forced, there was a mean reduction of percent activation of 0.01 for lateral bending effort of 60 Nm, 1.3 for extension effort of 60 Nm, -0.6 (i.e. a mean increase) for flexion effort of 60 Nm and 0.9 for axial rotation effort of 60 Nm (Table 2). The forcing of

Table 1

Model calculated values of spinal stability (as measured by the smallest eigenvalue; negative eigenvalue indicates instability) for four trunk effort directions (moments) of 20, 40 and 60 Nm, with intra-abdominal pressurization of 5 or 10 kPa at 60 Nm effort (pro-rated for lesser efforts). Values of the eigenvalues are given for the 'Baseline' condition and twelve conditions of forced minimum abdominal muscle activation. Note: the '+' symbol indicates an eigenvalue that is greater than the corresponding 'Baseline' value (i.e. more stable than 'Baseline'). The missing values in this table are for conditions with no plausible analytical solution, attributed to the abdominal wall activation required for spinal equilibrium in turn requiring abdominal pressurization greater than that specified.

Effort direction	Effort (Nm)	Baseline		Obl>10%		Obl>20%		Trans>10%		Trans>20%		Rectus>10%		Rectus>20%	
		5 kPa	10 kPa	5 kPa	10 kPa	5 kPa	10 kPa	5 kPa	10 kPa	5 kPa	10 kPa	5 kPa	10 kPa	5 kPa	10 kPa
Lateral bending	20	0.07	0.29	0.47+	0.58+	0.42+	0.63+	0.13+	0.34+	0.00	0.32+	0.33+	0.30+	0.13+	0.29
	40	0.21	0.48	0.57+	0.59+	0.50+	0.57+		0.58+	0.22+	0.61+	0.25+	0.48	0.31+	0.49+
	60	0.41	0.75	0.61+	0.70	0.59+	0.71	0.74+	0.82+	0.02	0.96+	0.51+	0.79+	0.58+	0.76+
Extension	20	0.26	0.41	0.59+	0.76+	0.59+	0.79+	0.32+	0.41	0.31+	0.49+	0.29+	0.42+	0.26	0.42+
	40	0.58	0.81	0.76+	0.85+	0.77+	1.04+	0.52	0.88+	0.48	0.68	0.44	0.77	0.69+	0.78
	60	0.74	1.02	0.65	0.92	0.83+	1.24+	0.64	1.16+	0.63	1.29+	0.57	0.92	0.67	1.09+
Flexion	20	0.52	0.60	0.18	0.16			0.45	0.63+	0.25	0.13	0.03	0.11	0.29	0.54
	40	0.61	1.36	0.19	0.76		0.36	0.63+	0.82		0.89		0.79	0.29	0.88
	60	0.60	0.80	0.57	0.88+		0.75	0.66+	0.68	0.35	0.80	0.12	1.25+	0.06	0.83+
Axial rot'n	20	0.56	0.80	0.71+	0.99+	0.00	0.94+	0.56	0.74	0.53	0.71	0.59	0.84+	0.60+	0.81+
	40	0.9	1.35	0.78	1.38+	0.92+	1.32	0.91+	1.23	0.89	1.19	0.85	1.37+	0.81	1.36+
	60	1.07	1.65	1.12+	1.63	1.09+	1.61+	1.08+	1.79+	1.17+	1.62	1.06	1.65	1.04	1.64
Mean difference from Baseline				0.06	-0.01	0.10	0.02	-0.04	-0.02	-0.14	-0.05	-0.08	-0.05	-0.07	-0.04

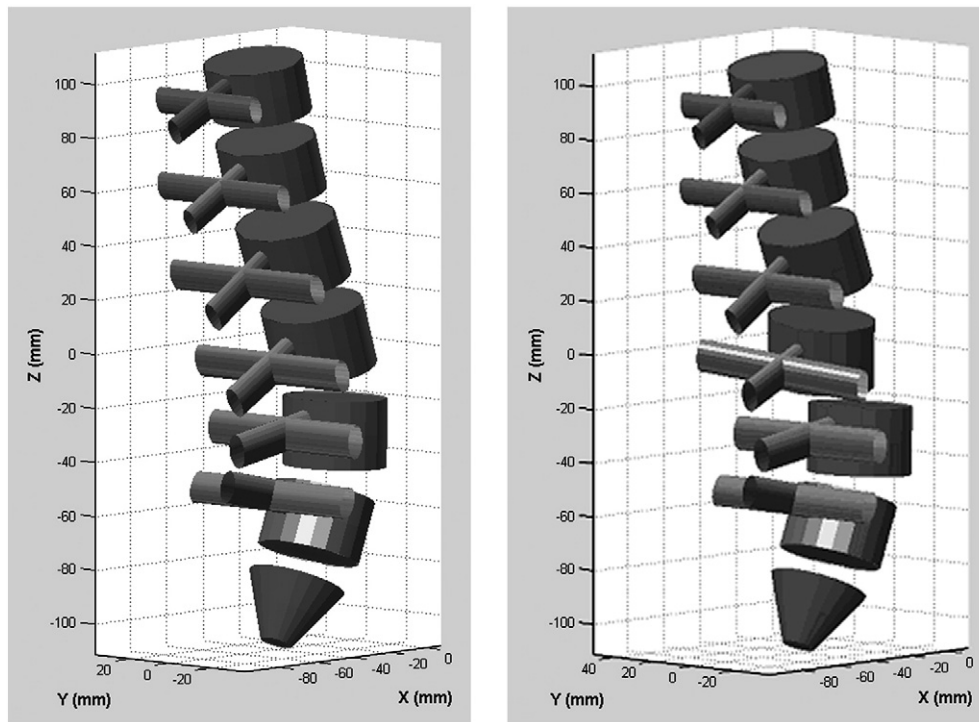


Fig. 2. Mode of buckling of the model for a typical loading example (extension effort, IAP = 5 kPa); left: initial undeformed geometry. Right: deformed (buckled) geometry. The vertebrae are shown symbolically to indicate where they have displaced to in the buckled mode relative to the initial geometry. The shape of the vertebrae and properties of ligaments and discs were held constant in the analyses, and the elastic properties of each motion segment (two vertebrae and intervening disc and ligaments) were represented by a stiffness matrix.

abdominal muscle activation was generally associated with a small (average 3%) increase in the spinal compression force (Table 2). This analytical model was previously reported (Stokes et al., 2010) to predict a reduction in spinal compressive force with increase in IAP from 5 to 10 kPa when abdominal muscles were not selectively forced to be active.

In the sensitivity analyses that tested the ‘robustness’ of the analyses, the stability of the model was altered by small amounts relative to changes in the abdominal wall shape (abdominal bulge and helix angle of obliques) or to changes in the muscular activation-dependent (short range) stiffness. For the variations in abdominal wall geometry, the smallest eigenvalue (measure of spinal stability)

Table 2
Spinal compression forces (Newtons) and muscle activations (percent of maximum) for an effort of 60 Nm in each of the four principal effort directions (lateral bending, extension, flexion and axial rotation) with IAP = 10 kPa, for the four muscle activation strategies: Baseline, transversus > 10%; obliques > 10%; and rectus > 10%. Lt = left side muscles and Rt = right side muscles.

	Spinal compression force (N)	External obliques		Internal obliques		Transversus		Rectus		
		Lt (%)	Rt (%)	Lt (%)	Rt (%)	Lt (%)	Rt (%)	Lt (%)	Rt (%)	
<i>Lateral bending</i>										
Baseline	469	10.5	9.5	4	3.2	0	9	3.8	21.7	
Trans. > 10%	473	11.7	11.0	3.9	5.1	10.0	12.1	0.1	23.4	
Obl. > 10%	481	13.5	20.7	10.0	12.2	0.0	3.1	0.0	27.7	
Rectus > 10%	463	9.7	9.0	4.2	5.1	0.0	9.2	10.0	21.9	
<i>Extension</i>										
Baseline	952	14.5	13.0	2.0	2.0	3.5	3.8	20.3	17.4	
Trans. > 10%	977	12.1	12.3	1.1	1.1	10.0	10.0	13.9	13.9	
Obl. > 10%	997	19.8	20.0	11.0	10.8	3.9	3.9	17.0	18.3	
Rectus > 10%	971	10.9	14.4	1.4	2.1	2.0	4.4	10.2	12.2	
<i>Flexion</i>										
Baseline	537	21.9	22.6	34.1	33	0.0	0.0	0.0	0.0	
Trans. > 10%	559	24.4	23.2	32.5	33.2	10.1	10.1	0.0	0.0	
Obl. > 10%	632	36.2	36.9	53.0	52.0	1.2	0.0	0.0	0.0	
Rectus > 10%	579	27.8	26.9	34.5	34.6	0.8	0.0	10.0	10.0	
<i>Axial rotation</i>										
Baseline	459	6.2	3.5	1.4	3.9	0.0	6.8	1.8	3.1	
Trans. > 10%	478	6.4	2.8	0.9	4.6	10.0	10.1	0.0	0.1	
Obl. > 10%	477	10.4	10.1	10.0	10.0	0.0	3.6	0.0	0.0	
Rectus > 10%	417	5.7	2.6	1.7	4.2	0.0	6.0	10.0	10.0	

increased in 11 cases, and decreased in 21 cases in the 32 permutations that were tested (2 pressures and 4 loading directions; 2 variations of the bulge and 2 variations of the fiber angle of the obliques). The changes were less than 20% in 12 of 32 cases, and there were only 2 cases of predicted instability for a case that was stable in the Baseline condition (for flexion effort with 5 kPa IAP). When the activation dependent muscle stiffness was halved or doubled, the smallest eigenvalue generally changed less than 10% (in 92 of 112 loading direction, IAP, and forced muscle activation permutations examined). In these 112 permutations there were no cases of predicted instability with alteration of the muscle stiffness.

4. Discussion

In these analyses, spinal stability increased with increased abdominal pressurization. However, the degree of stability was not substantially influenced by forcing either transversus or obliques to be active, at either 10% or 20% of maximum activation. This casts doubt on the proposed mechanism of action of specific lumbar stabilization exercise regimens that have been proposed for low back pain rehabilitation. In fact, in some cases forced increased activation of abdominal muscles produced decreased lumbar stability. This key finding indicates that intra-abdominal pressure increases stability, but forcing component parts of the abdominal wall (transversus, obliques or rectus) to be preferentially active does not systematically increase stability. No patterns associating forced activations with either an increase or a decrease in stability were identified, and both increases and decreases were observed in approximately equal numbers.

The findings reported here are considered to be 'robust', because the sensitivity analyses showed that the calculated spinal stability was altered minimally compared to changes in the muscle stiffness (and model geometry parameters).

Since these findings were obtained from an analytical model, several limitations should be noted. While human activities involve an infinite number of permutations of forces and moments, the current study employed three pure moments (about each principal axis) of magnitudes up to 60 Nm to provide a representative and objective sample of realistic activities. The greatest effort about each axis that was analyzed (60 Nm) is equal to average maximum efforts reported for women making maximum voluntary efforts in axial rotation (other effort directions and male subjects can generate higher moments) (Stokes and Gardner-Morse, 1995), so it is considered representative of the range of efforts in every-day life.

The analysis used anthropometric data as compiled by Stokes and Gardner-Morse (1999). These values correspond to an averaged adult skeleton, and with muscle cross sectional areas taken from anatomical dissections and from the male and female 'Visible Humans'. No attempt was made to vary these values to represent people of different body types, and the values used may be quite representative since values the size and position of the muscles of people having differing body mass index vary by only about 10% (Wood et al., 1996). The magnitude of the intra-abdominal pressure expressed as a ratio of the moment (effort) was 0.6 kPa per Nm when the pressure was 10 kPa, which is a physiological ratio according to Stokes et al. (2010).

The diaphragm must also be activated to support any pressure differential between the abdomen and the thorax. In these analyses the diaphragm and pelvic floor muscles were considered to be rigid (i.e. isometric), so the stress in them and the relative roles of their activation and possible elastic strains associated with tissue stretching were not considered, since under isometric circumstances they would not affect the spinal stability.

The analytically derived muscle activations and recruitment patterns were based on hypothetical muscle stress and strain optimization employing a 'cost function' approach. These only represent one of a potentially huge number of individual activation patterns that might

be employed by humans, and coactivation patterns among the entire muscle set may vary considerably from these assumptions. The realism of biomechanical models can be improved in the 'EMG-assisted' approach that employs data from EMG studies to provide initial estimates of muscle activations, subsequently adjusted to ensure static equilibrium of net moments and forces at articulations. We did not use EMG data as inputs for the model because the complexity of the model (number of individually activated muscles included) makes EMG 'drive' impractical.

The analyses were static in that they did not include dynamic inertial effects or time delays or variation in muscle activation 'sequencing'. Spinal buckling events may be more likely to occur under dynamic conditions. The possible variations in muscle activation over time in real-life activities were therefore not included. However, the cost-function approach does predict varying activation patterns (relative activation of different muscles) as the effort magnitude increases.

A limitation of these analyses is uncertainty about muscle stiffness properties and these are poorly understood, especially the stiffness of the abdominal wall muscles perpendicular to the lines of action of the muscle fibers and the value of 'short range' stiffness that is thought to be appropriate in buckling analyses. However, the findings concerning spinal stability were found to be relatively insensitive to the values of these parameters in the analyses.

The increase in lumbar stability with abdominal muscle activation was expected, based on findings in previous studies (Arjmand and Shirazi-Adl, 2006; Gardner-Morse and Stokes, 1998; Kavcic et al., 2004). These previous analyses have demonstrated analytically that spinal stability is generally increased with antagonistic activation of abdominal muscles, but have not been able to explore the interactions with IAP that added physiological realism to the present study. The prior models have not employed the representation of the abdominal muscles that permitted investigation of variations in the relative activation of the abdominal muscles while maintaining compatibility with the IAP, made possible here by considering the abdominal wall as a pressure vessel with curved muscles containing the pressure. More importantly, other models have not investigated relative contributions of different muscle groups as they relate to rehabilitation exercises. The stiffness of the muscle increases with its degree of activation and in these biomechanical analyses spinal stability depended on this activation-dependent muscle stiffness because the ligamentous spine is known to be unstable (Crisco et al., 1992).

There are no experiments in which spinal instability is deliberately produced and documented in living humans with muscle activity. Buckling of axially loaded fingers may be the nearest analogous case of instability in a multi-joint system. Therefore validation of our analyses against empirical data is not possible, and these kinds of analyses 'belong to a certain category of models in science for which there are no tools for model validation' (Cholewicki and McGill, 1996).

Because the abdominal muscles were modeled as curved structures they could contain a pressure within the abdomen and equally any tension in these muscles was necessarily associated with a rise in abdominal pressure. Thus all activities involving abdominal muscle activation required a rise in IAP in the model, and this probably explains why IAP is raised in most physiological efforts. In some modeled cases with forced activation of abdominal muscles there was no plausible analytical solution because the required muscle forces would result in a pressure higher than that specified. These cases are shown as missing values in Table 2.

The model employed here has previously been compared with physiological behavior with regard to statics (magnitudes of internal forces in a stable equilibrium condition) (Stokes et al., 2010). It was reported that the calculated spinal compression forces were in the range 250 N (with 5 kPa IAP and zero effort) to 1202 N (60 Nm extension effort) while in vivo spinal compression forces range from 500 N (passive standing) to 2000 N (lifting activity), based on intra-

discal pressure measurements. The magnitudes of abdominal muscle activation predicted by the model were also comparable with those reported in electromyographic studies (Arjmand and Shirazi-Adl, 2006; Cresswell et al., 1992; de Looze et al., 1999; McCook et al., 2009; Thelen et al., 1995).

The present study reports relative values of the smallest eigenvalue as a measure of spinal stability. Other analytical studies have used the 'stability index' (Howarth et al., 2004) as a measure of stability which is actually a product of 18 eigenvalues, and the relevance of this derived number to the most likely (i.e. smallest eigenvalue buckling mode) has been questioned (Gardner-Morse et al., 2006). The magnitude of the smallest eigenvalue provides a comparative measure of the stability of the system, and it is non-dimensional (has no physical units). Thus, the larger the eigenvalue the more stable a system is.

This study revisits the fundamental question: why raise IAP when this often requires antagonistic muscle activity? It is thought that IAP unloads and/or stabilizes the spine. Approximately, doubling the muscle activation doubles the pressure according to simplified statics. Thus, IAP and abdominal wall muscle activation are linked. Antagonistic muscle activity generally not only helps stabilize a joint, but also increases the joint loading. In the case of the lumbar spine, the associated IAP also generates an extension moment that serves to unload the spine (Stokes et al., 2010).

Given that the degree of lumbar stability was not substantially influenced by forcing either transversus abdominis or obliques to be active at 10% or 20% of maximum activation in the biomechanical model, doubt is cast upon the proposed mechanism of action of specific abdominal muscle exercise regimens (Richardson et al., 2004) that have been advocated for low back pain rehabilitation. Since the present work is based on a static analysis of spinal loading, it did not address the possible effects of delays in muscle recruitment that have been reported (Radebold et al., 2000, 2001; van Dieën et al., 2003) and shown analytically to influence spinal stability by Franklin et al. (2008). However, earlier observations of delayed anticipatory postural adjustments of the deep abdominal muscles in people with low back pain have not been consistently substantiated in subsequent studies (Gubler et al., 2010).

While the mechanism of action is important to understand, the clinically important question is whether or not a specific stabilization exercise regime is effective at increasing function and decreasing pain levels and recurrences in people with low back pain. Early clinical studies provided promise of the specific stabilization approach (Hides et al., 1996, 2001; O'Sullivan et al., 1997; Stuge et al., 2004a, 2004b) but recent studies have not substantiated these findings. For example, improvements in both activity and patients' impression of recovery were small in both the short and long terms following a motor control exercise regime in patients with chronic low back pain (Costa et al., 2009). Koumantakis et al. (2005) added specific stabilization exercises to general exercise for people with nonspecific low back pain and reported a greater reduction in disability immediately post-treatment in the general exercise group only. Recently, a trial comparing specific stabilization exercises (called 'motor control exercises' in the study), high-load sling exercises, or general exercises did not show any overall group effects in pain levels, disability, and fear-avoidance beliefs in people with chronic low back pain (Unsgaard-Tøndel et al., 2010) and demonstrated no added benefit of specific exercises over general exercises in this population. Thus, there is no clear clinical evidence that specific stabilization exercise regimens that target specific retraining of TA and multifidus muscles are better than other forms of exercises in people with chronic low back pain (Macedo et al., 2009; Rackwitz et al., 2006; Standaert et al., 2008). Our biomechanical analyses would suggest that preferential activation of the deep abdominal muscles does not provide additional lumbar stability and may provide insight as to why this clinical approach has not proven to provide superior patient outcomes.

Analytical studies provide support for the idea that the human lumbar spine may be at risk for buckling events responsible for sudden onset of certain forms of back pain. It is possible that individual variations in anatomy or muscle recruitment patterns could place certain individuals at higher risk of such events. The present study provides an explanation for the mechanism of the stabilizing effect of abdominal wall muscular activation associated with intra-abdominal pressurization, and additionally demonstrates analytically that forced activation of selected abdominal muscle layers would not necessarily provide additional lumbar stability. However, this remains a theoretic construct that would be difficult to validate scientifically. The predictions of analytical models might be tested for validity indirectly by comparison with epidemiological studies of human populations and clinical studies of the effects of treatments such as exercise regimens and muscle re-education programs.

5. Conclusion

Based on predictions from a buckling model analysis pressurization of the abdomen increased lumbar spinal stability, but the degree of spinal stability was not substantially influenced by selectively forcing either transversus abdominis or oblique muscles to be active. This supports the use of rehabilitation regimens that encourage abdominal activity pressurization by activation of abdominal wall muscles, but casts doubt on the supposed mechanism of action of specific abdominal muscle exercise regimens that have been proposed for low back pain rehabilitation.

Acknowledgment

This work was supported by the National Institutes of Health Grant NIH AR 40909.

References

- Arjmand, N., Shirazi-Adl, A., 2006. Role of intra-abdominal pressure in the unloading and stabilization of the human spine during static lifting tasks. *Eur. Spine J.* 15 (8), 1265–1275.
- Barker, K.L., Shambley, D.R., Jackson, D., 2004. Changes in the cross-sectional area of multifidus and psoas in patients with unilateral back pain: the relationship to pain and disability. *Spine* 29 (22), E515–E519.
- Bergmark, A., 1989. Stability of the lumbar spine. A study in mechanical engineering. *Acta Orthop. Scand. Suppl.* 230, 1–54.
- Cholewicki, J., McGill, S.M., 1992. Lumbar posterior ligament involvement during extremely heavy lifts estimated from fluoroscopic measurements. *J. Biomech.* 25 (1), 17–28.
- Cholewicki, J., McGill, S.M., 1996. Mechanical stability of the in vivo lumbar spine: implications for injury and chronic low back pain. *Clin. Biomech.* 11 (1), 1–15.
- Cholewicki, J., Juluru, K., McGill, S.M., 1999a. Intra-abdominal pressure mechanism for stabilizing the lumbar spine. *J. Biomech.* 32 (1), 13–17.
- Cholewicki, J., Juluru, K., Radebold, A., Panjabi, M.M., McGill, S.M., 1999b. Lumbar spine stability can be augmented with an abdominal belt and/or increased intra-abdominal pressure. *Eur. Spine J.* 8 (5), 388–395.
- Costa, L.O., Maher, C.G., Latimer, J., Hodges, P.W., Herbert, R.D., Refshauge, K.M., et al., 2009. Motor control exercise for chronic low backpain: a randomized placebo-controlled trial. *Phys. Ther.* 89, 1275–1286.
- Cresswell, A.G., Grundström, H., Thorstensson, A., 1992. Observations on intra-abdominal pressure and patterns of abdominal intra-muscular activity in man. *Acta Physiol. Scand.* 144 (4), 409–418.
- Crisco III, J.J., Panjabi, M.M., 1991. The intersegmental and multisegmental muscles of the lumbar spine. A biomechanical model comparing lateral stabilizing potential. *Spine* 16 (7), 793–799.
- Crisco, J.J., Panjabi, M.M., Yamamoto, I., Oxland, T.R., 1992. Euler stability of the human ligamentous lumbar spine. Part 2: experimental. *Clin. Biomech.* 7, 27–32.
- de Looze, M.P., Groen, H., Horemans, H., Kingma, I., van Dieën, J.H., 1999. Abdominal muscles contribute in a minor way to peak spinal compression in lifting. *J. Biomech.* 32 (7), 655–662.
- Franklin, T.C., Granata, K.P., Madigan, M.L., Hendricks, S.L., 2008. Linear time delay methods and stability analyses of the human spine. Effects of neuromuscular reflex response. *IEEE Trans. Neural Syst. Rehabil. Eng.* 16 (4), 353–359.
- Gardner-Morse, M.G., Stokes, I.A.F., 1998. The effects of abdominal muscle co-activation on lumbar spine stability. *Spine* 23 (1), 86–92.
- Gardner-Morse, M.G., Stokes, I.A.F., 2004. Structural behavior of human lumbar spinal motion segments. *J. Biomech.* 37 (2), 205–212.
- Gardner-Morse, M., Stokes, I.A.F., Laible, J.P., 1995. Role of muscles in lumbar spine stability in maximum extension efforts. *J. Orthop. Res.* 13 (5), 802–808.

- Gardner-Morse, M.G., Stokes, I.A., Huston, D.R., 2006. On the implications of interpreting the stability index: a spine example. *J. Biomech.* 39 (2), 391–392 (comment on: *J. Biomech.* 2004, 37(8):1147–54).
- Grenier, S.G., McGill, S.M., 2007. Quantification of lumbar stability by using 2 different abdominal activation strategies. *Arch. Phys. Med. Rehabil.* 88 (1), 54–62.
- Grew, N.D., 1980. Intraabdominal pressure response to loads applied to the torso in normal subjects. *Spine* 5 (2), 149–154.
- Gubler, D., Mannion, A.F., Schenk, P., Gorelick, M., Helbling, D., Gerber, H., et al., 2010. Ultrasound tissue Doppler imaging reveals no delay in abdominal muscle feed-forward activity during rapid arm movements in patients with chronic low back pain. *Spine* 35 (16), 1506–1513.
- Hides, J.A., Richardson, C.A., Jull, G.A., 1996. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. *Spine* 21, 2763–2769.
- Hides, J.A., Jull, G.A., Richardson, C.A., 2001. Longterm effects of specific stabilizing exercises for first-episode low back pain. *Spine* 26, E243–E248.
- Hides, J., Gilmore, C., Stanton, W., Bohlscheid, E., 2008. Multifidus size and symmetry among chronic LBP and healthy asymptomatic subjects. *Man. Ther.* 13 (1), 43–49.
- Hodges, P.W., Richardson, C.A., 1996. Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. *Spine* 21 (22), 2640–2650 (15).
- Hodges, P., Kaigle-Holm, A., Holm, S., Ekström, L., Cresswell, A., Hansson, T., et al., 2003. Intervertebral stiffness of the spine is increased by evoked contraction of transversus abdominis and the diaphragm: in vivo porcine studies. *Spine* 28 (23), 2594–2601.
- Hodges, P., Holm, A.K., Hansson, T., Holm, S., 2006. Rapid atrophy of the lumbar multifidus follows experimental disc or nerve root injury. *Spine* 31 (25), 2926–2933.
- Howarth, S.J., Allison, A.E., Grenier, S.G., Cholewicki, J., McGill, S.M., 2004. On the implications of interpreting the stability index: a spine example. *J. Biomech.* 37 (8), 1147–1154.
- Ivancic, P.C., Cholewicki, J., Radebold, A., 2002. Effects of the abdominal belt on muscle-generated spinal stability and L4/L5 joint compression force. *Ergonomics* 45 (7), 501–513.
- Kavcic, N., Grenier, S., McGill, S.M., 2004. Determining the stabilizing role of individual torso muscles during rehabilitation exercises. *Spine* 29 (11), 1254–1265.
- Koumantakis, G.A., Watson, P.J., Oldham, J.A., 2005. Trunk muscle stabilization training plus general exercise versus general exercise only: randomized controlled trial of patients with recurrent low back pain. *Phys. Ther.* 85, 209–225.
- Lederman, E., 2010. The myth of core stability. *J. Bodyw. Mov. Ther.* 14 (1), 84–98.
- Macedo, L.G., Maher, C.G., Latimer, J., McAuley, J.H., 2009. Motor control exercise for persistent, nonspecific low back pain: a systematic review. *Phys. Ther.* 89, 9–25.
- Mairiaux, P., Malchaire, J., 1988. Relation between intra-abdominal pressure and lumbar stress: effect of trunk posture. *Ergonomics* 31 (9), 1331–1342.
- McCook, D.T., Vicenzino, B., Hodges, P.W., 2009. Activity of deep abdominal muscles increases during submaximal flexion and extension efforts but antagonist co-contraction remains unchanged. *J. Electromyogr. Kinesiol.* 19 (5), 754–762.
- McGuire, W., Gallagher, R.H., 1979. *Matrix Structural Analysis*. John Wiley & Sons, New York, pp. 329–330.
- McNeill, W., 2010. Core stability is a subset of motor control. *J. Bodyw. Mov. Ther.* 14 (1), 80–83.
- O'Sullivan, P., 2000. Lumbar segmental “instability”: clinical presentation and specific stabilizing exercise management. *Man. Ther.* 5 (1), 2–12.
- O'Sullivan, P.B., Phytty, G.D., Twomey, L.T., Allison, G.T., 1997. Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine* 22, 2959–2967.
- O'Sullivan, P.B., Twomey, L., Allison, G.T., 1998. Altered abdominal muscle recruitment in patients with chronic back pain following a specific exercise intervention. *J. Orthop. Sports Phys. Ther.* 27 (2), 114–124.
- Paz, M., 1985. *Structural dynamics, Theory and Computation*, 2nd ed. Van Nostrand Reinhold, New York, pp. 248–249.
- Rackwitz, B., de Bie, R., Limm, H., von Garnier, K., Ewert, T., Stucki, G., 2006. Segmental stabilizing exercises and low back pain, what is the evidence: a systematic review of randomized controlled trials. *Clin. Rehabil.* 20, 553–567.
- Radebold, A., Cholewicki, J., Panjabi, M.M., Patel, T.C., 2000. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. *Spine* 25 (8), 947–954.
- Radebold, A., Cholewicki, J., Polzhofer, G.K., Greene, H.S., 2001. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. *Spine* 26 (7), 724–730.
- Richardson, C., Jull, G., Hodges, P., Hides, J., 2004. *Therapeutic Exercise for Spinal Segmental Stabilization in Low Back Pain*, 2nd ed. Churchill Livingstone, Philadelphia, PA.
- Schultz, A.B., 1990. Biomechanical analyses of loads on the lumbar spine. In: Weinstein, J.N., Wiesel, S.W. (Eds.), *The Lumbar Spine*. W.B. Saunders, Philadelphia, pp. 160–171.
- Standaert, C.J., Weinstein, S.M., Rumpeltes, J., 2008. Evidence-informed management of chronic low back pain with lumbar stabilization exercises. *Spine J.* 8, 114–120.
- Stokes, I.A., Gardner-Morse, M., 1995. Lumbar spine maximum efforts and muscle recruitment patterns predicted by a model with multijoint muscles and joints with stiffness. *J. Biomech.* 28 (2), 173–186.
- Stokes, I.A.F., Gardner-Morse, M., 1999. Quantitative anatomy of the lumbar musculature. *J. Biomech.* 32, 311–316.
- Stokes, I.A., Gardner-Morse, M., 2001. Lumbar spinal muscle activation synergies predicted by multi-criteria cost function. *J. Biomech.* 34 (6), 733–740.
- Stokes, I.A., Gardner-Morse, M.G., Henry, S.M., 2010. Intra-abdominal pressure and abdominal wall muscular function: spinal unloading mechanism. *Clin. Biomech.* 25 (9), 859–866.
- Stuge, B., Laerum, E., Kirkesola, G., Vøllestad, N., 2004a. The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy: a randomized controlled trial. *Spine* 29, 351–359.
- Stuge, B., Veierød, M.B., Laerum, E., Vøllestad, N., 2004b. The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy: a two-year follow-up of a randomized clinical trial. *Spine* 29, E197–E203.
- Thelen, D.G., Schultz, A.B., Ashton-Miller, J.A., 1995. Co-contraction of lumbar muscles during the development of time-varying triaxial moments. *J. Orthop. Res.* 13 (3), 390–398.
- Unsgaard-Tøndel, M., Fladmark, A.M., Salvesen, Ø., Vasseljen, O., 2010. Motor control exercises, sling exercises, and general exercises for patients with chronic low back pain: a randomized controlled trial with 1-year follow-up. *Phys. Ther.* 90 (10), 1–14.
- van Dieën, J.H., Cholewicki, J., Radebold, A., 2003. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. *Spine* 28 (8), 834–841.
- Wood, S., Pearsall, D.J., Ross, R., Reid, J.G., 1996. Trunk muscle parameters determined from MRI for lean to obese males. *Clin. Biomech.* 11 (3), 139–144.