ORIGINAL CONTRIBUTION

Increased Incidence and Severity of Somatic Dysfunction in Subjects With Chronic Low Back Pain

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Context: Patients with back pain make more than 14 million office visits per year to US physicians. Many of these patients have chronic low back pain (LBP) and are assumed to have more somatic dysfunction than those without chronic LBP.

Objective: To investigate incidence and severity of somatic dysfunction of four lumbar vertebral segments (L1-L4).

Methods: Sixteen subjects with chronic LBP and 47 subjects without chronic LBP were each evaluated by two blinded examiners using reliable osteopathic palpatory tests. The incidence and severity of somatic dysfunction for each test were then analyzed within and between the study groups.

Results: Resistance to anterior springing (P<.001) and tenderness (P=.002) were found at significantly greater incidence in the chronic LBP group than in the non-LBP group, but there were no significant differences between groups for incidence of tissue texture changes or static rotational asymmetry. Significantly greater severity of tissue texture changes (P=.006), static rotational asymmetry (P=.008), resistance to anterior springing (P<.001), and tenderness (P=.001) were observed in the chronic LBP group than in the non-LBP group.

Conclusion: When compared with non-LBP subjects, chronic LBP subjects had overall greater severity for each of the four elements of somatic dysfunction evaluated, as well as greater incidence of resistance to anterior springing and tenderness. Somatic dysfunction is more severe in individuals with chronic LBP than in individuals without chronic LBP.

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In 2005, more than 14 million office visits to US physicians were made by patients complaining of back pain.¹ Low back pain (LBP) has an overall prevalence of 60% to 80% in industrialized countries.^{2,3} In about 85% of these people, LBP is secondary to nonspecific or functional causes, meaning that no specific underlying anatomic etiologic condition can be identified.⁴

Patients with nonspecific LBP are frequently treated by osteopathic physicians (DOs) using osteopathic manipulative (OM) techniques directed at specific somatic dysfunctions diagnosed by palpation. Somatic dysfunction is defined in the osteopathic literature as "[i]mpaired or altered function of related components of the somatic (body framework) system: skeletal, arthrodial, and myofascial structures, and related vascular, lymphatic, and neural elements."⁵ In physical examinations of patients, somatic dysfunction may manifest as tissue texture changes, joint asymmetry, altered range of motion, or tenderness.⁵

In clinical practice, it is generally assumed that while people with and without LBP will demonstrate some level of somatic dysfunction, people with chronic LBP will have more somatic dysfunction of the lumbar spine than those without chronic LBP.⁶ This assumption, however, has not been delineated directly by previous studies. Therefore, using this assumption as a hypothesis, we examined the incidence and severity of somatic dysfunction in a group of subjects with chronic LBP versus a group of subjects without this condition. Further study of this relationship may contribute to reproducible clinical interventions that target this somatic dysfunction and lead to better medical management of chronic LBP.

Methods

Subject Recruitment

As part of a previous study,⁷ we conducted an analysis of somatic dysfunction in the lumbar spine of subjects aged 20 to 40 years regardless of LBP symptoms. All subjects were recruited from the faculty, students, and staff of Kirksville (Mo) College of Osteopathic Medicine-A.T. Still University (KCOM-ATSU) and the Northeast Regional Medical Center, also in Kirksville, as well as from the local community. Flyers and emails were the primary recruitment methods. All subjects were prescreened through telephone interviews and later rescreened in person to ensure study eligibility.

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Individuals were excluded from the study if they had any medical conditions that could potentially alter the structure of the lumbar vertebrae, such as congenital vertebral anomalies (eg, spina bifida), prior lumbar or low thoracic vertebral fractures, or surgical structural changes. Individuals who had undergone spinal manipulation within 8 weeks of study initiation were also excluded. Evaluation of exclusion criteria was based on individuals' self-reported medical histories.

A total of 63 subjects were recruited. After recruitment, subjects were divided into two groups: a group of 16 individuals with chronic LBP and a group of 47 individuals without chronic LBP. For purposes of the present study, chronic LBP was defined as pain in the small of the back lasting a minimum of 5 days per week for at least 3 months. Subjects in the non-LBP group may occasionally have had LBP in the past, but only on an intermittent basis and not exceeding 2 days per week.

The present study was planned as a two-factor nested design in which the between-subject factor was the group (chronic LBP or non-LBP) and the within-subject factor was the lumbar vertebral segment (L1-L4). The Institutional Review Board of KCOM-ATSU approved all aspects of the present study. All subjects completed informed consent forms before study enrollment. The study was conducted at KCOM-ATSU.

Palpatory Examination

After informed consent was obtained, each subject received a focused structural examination by two of three DO examiners. One examiner (B.F.D.) was an AOA board-certified specialist in neuromusculoskeletal medicine and osteopathic manipulative medicine (OMM). Another examiner (K.T.S.) was in her first year of practice and was eligible for AOA board certification in neuromusculoskeletal medicine and OMM. The third examiner (E.J.S.) was a resident in neuromusculoskeletal medicine and OMM. All three DOs rotated as needed in performing subject examinations to accommodate sometimes conflicting schedules. To ensure interexaminer reliability during this physical evaluation, the two examiners conducting the evaluation were blinded to the subject's LBP history, as well as to each other's physical findings for each subject.

In preparation for the present study, the three participating examiners evaluated the interexaminer reliability of 15 types of osteopathic palpatory tests commonly taught for diagnosis of somatic dysfunction at KCOM-ATSU.⁷ From these 15 palpatory tests, the four tests that demonstrated the greatest interexaminer reliability (texture changes, static rotational asymmetry, resistance to anterior springing, tenderness) in our previous study⁷ were further refined, and the three examiners designed a training protocol to promote consensus. The methods used for palpatory test evaluation and consensus training are described elsewhere.⁷

The examination of the L1-L4 vertebral segments were conducted individually with each subject lying in the prone

position while the examiner evaluated tissue texture changes on the left and right sides as well as static rotational asymmetry, resistance to anterior springing, and tenderness. The physical findings of each examiner were compared, and the subject was reexamined by both DOs together to establish consensus for any conflicting findings. Vertebral segment L5 was not evaluated because of the high frequency of occult (ie, soft tissue) congenital anomalies associated with it.⁸

The *Figure* summarizes the palpatory techniques used during structural examinations in the present study. Before each subject's examination, researchers calibrated pressure for assessment of tenderness by repeatedly applying pressure on an 11 lb (5 kg) food scale (Taylor Precision Products, Oak Brook, Ill) with the pad of the thumb until intraexaminer reliability was obtained within three pressure ranges (<2 kg/cm², 2-4 kg/cm²).

An individual vertebra was considered positive for somatic dysfunction in a specific palpatory test if the vertebra was rated as 2 (mild dysfunction) or 3 (moderate/severe dysfunction) on a severity scale of 1 to 3, with 1 indicating no dysfunction. The range for the number of positive findings (ie, incidence) for somatic dysfunction at each of the four vertebral segments (L1-L4) examined was 0 to 5, with 0 meaning there were no positive findings for any of the five palpatory tests (tissue texture changes on the left side, tissue texture changes on the right side, static rotational asymmetry, resistance to anterior springing, tenderness), and a number between 1 and 5 indicating there were minor to moderate/severe findings for one to five of the palpatory tests.

In addition, the somatic dysfunction severity score for each vertebral segment was defined as the sum of the severity ratings for each of the five palpatory tests conducted. For example, if a subject had no somatic dysfunction findings in any of the palpatory tests, the severity score would be a 5. If a subject demonstrated moderate/severe somatic dysfunction for all five tests, the severity score would be 15 (5 tests \times severity rating of 3). Therefore, the possible range of the somatic dysfunction severity score was 5 to 15 per vertebral segment.

Statistical Methods

The gender composition of the chronic LBP and non-LBP groups was compared using the Fisher exact test, while the age and body mass index (BMI) of the two groups were compared using Mann-Whitney *U* tests. For each of the five measured palpatory tests of somatic dysfunction, the chronic LBP and non-LBP groups were compared on the incidence of positive findings (ie, severity scale rating of 2 or 3) using logistic regression models and on the severity of findings using proportional odds models for ordinal outcome variables. Proportional odds models were also used to compare the two groups on the number of positive findings. To account for the hierarchical structure of the data resulting from the repeated measurements of somatic dysfunction made for each subject (ie, measurements of L1-L4), the generalized estimating equa-

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Palpatory Examination	Assessment Protocol	Indication of Positive Finding	Severity Scale
Tissue texture changes	Assessed by palpating subcutaneous tissues with pads of fingers directly posterior to inferior articular facets of L1-L4.	Localized edema and/or fibrotic changes, rated separately for right and left inferior articular facets of each vertebra.	 1 = No texture changes 2 = Mild texture changes 3 = Moderate/severe texture changes
Static rotational asymmetry	Assessed with simultaneous placement of thumbs on transverse processes of L1-L4. Anterior pressure applied until transverse processes could be palpated. No motion testing performed.	Based on static positioning of transverse processes of each vertebra. Direction of rotation defined by whether right or left transverse process demonstrated posterior prominence.	1 = No rotation 2 = Mild rotation 3 = Moderate/severe rotation
Resistance to anterior springing	Localized extension induced by springing anteriorly with hypothenar eminence on spinous processes of L1-L4. Each examiner could spring anteriorly as many as three times.	Resistance encountered to anterior springing, compared with vertebral segment above or below.	 1 = No motion restriction 2 = Mild motion restriction 3 = Moderate/severe motion restriction
Tenderness	Applied localized anterior thumb pressure directly over spinous processes of L1-L4.	Subject verbalized response to development of tenderness as elicited by anterior thumb pressure.	 1 = No tenderness with as much as 4 kg/cm² pressure 2 = Tenderness with 2-4 kg/cm² pressure 3 = Tenderness with <2 kg/cm² pressure
			5

Figure. Summary of palpatory examinations performed with subjects (N=63) in study of somatic dysfunction associated with chronic low back pain. The assessment protocols, indications of positive findings for somatic dysfunction, and three-point severity scales used for each palpatory test are shown. Abbreviations: L1-L4, lumbar vertebral segment 1 to lumbar vertebral segment 4.

tions approach was applied to fit the logistic regression and proportional odds models. A *P* value of less than or equal to .05 was deemed necessary to achieve statistical significance.

In a post hoc analysis that tested whether static rotational asymmetry to one side—either left or right—was more common than rotational asymmetry to the other side, the Wilcoxon signed rank test was used, comparing the number showing left rotational asymmetry with the number of vertebrae showing right rotational asymmetry. This comparison was conducted for both study groups together and each separately. In addition, the Mann-Whitney *U* test was used to compare the study groups on the number of vertebrae showing left rotational asymmetry.

Results

Of the 63 volunteers who were recruited for the present study, 16 (25%) were in the chronic LBP group and 47 (75%) were in the non-LBP group. The mean (SD) age of subjects was 30 (6) years, with an age range from 20 to 40 years. Forty-eight (76%) of the study subjects were women, with 10 women

(63%) in the chronic LBP group and 38 women (81%) in the non-LBP group. Participants' mean (SD) BMI was 26 (5). This value is slightly lower than the national average BMI of 27.3 for people in this age range.⁹ There was no statistically significant difference between the two study groups in terms of age, sex, or BMI.

Based on the 0-to-5-point scale used to evaluate somatic dysfunction incidence per vertebra, subjects with chronic LBP had more positive findings of somatic dysfunction (4.0 [0.8]; 95% confidence interval [CI], 3.7-4.4) than those without LBP (3.3 [1.0]; 95% CI, 3.0-3.4). This difference between the chronic LBP and non-LBP groups was statistically significant (P<.001). Based on the 5-to-15-point scale used to evaluate somatic dysfunction severity per vertebra, subjects with chronic LBP also had greater severity of somatic dysfunction (10.7 [1.3]; 95% CI, 10.1-11.2) than those without LBP (9.2 [1.7]; 95% CI, 8.9-9.5). This difference between the two groups was also statistically significant (P<.001). Summary statistics on the incidence (expressed as percentages of vertebral segments demonstrating individual components of somatic dysfunction

tion) and the severity (expressed as mean and 95% CIs on the 1-to-3-point severity scale) of somatic dysfunction for the chronic LBP and non-LBP groups are provided in *Table 1*.

No significant differences were found between the chronic LBP and non-LBP groups for the incidence of vertebrae testing positive for tissue texture changes (P=.54) or static rotational asymmetry (P=.21). However, the two groups were significantly different for the severity of somatic dysfunction detected as tissue texture changes (P=.006) and static rotational asymmetry (P=.008), with the chronic LBP group having greater severity than the non-LBP group.

Significant differences were found between the two study groups in the incidence of vertebrae testing positive for resistance to anterior springing (P<.001) and tenderness (P=.002), with both occurring more frequently in the chronic LBP group than in the non-LBP group. Furthermore, the severity of somatic dysfunction detected as resistance to anterior springing (P<.001) and tenderness (P=.001) was also significantly different between the two groups, with the chronic LBP group again having greater severity than the non-LBP group.

Because static rotational asymmetry could be measured either to the left or to the right, the incidence and severity of each possible rotation was evaluated, with summary statistics, including means and 95% CIs (*Table 2*). The chronic LBP group and non-LBP group each demonstrated a greater incidence of left rotation compared with right rotation (P<.001 for each group). In addition, each group had a significantly greater occurrence of moderate/severe left rotation when compared to moderate/severe right rotation (P<.001 for each group).

In comparisons between the chronic LBP and non-LBP groups, no significant differences were found in the incidence of either left (P=.31) or right (P=.70) rotation. The chronic LBP group had a greater occurrence of moderate/severe left rotation than the non-LBP group (P=.01), but there was no significant difference between the two groups regarding the occurrence of moderate/severe right rotation (P=.76).

Comment

The present study revealed that somatic dysfunction was more prevalent in subjects with chronic LBP than in subjects without chronic LBP. The subjects with chronic LBP had overall greater severity of each of the four elements of somatic dysfunction evaluated: tissue texture changes, static rotational asymmetry, resistance to anterior springing, and tenderness in L1 to L4. In addition, the present study found greater incidence of resistance to anterior springing and tenderness in the subjects with chronic LBP than in the subjects without chronic LBP. However, no statistically significant differences were found between the chronic LBP and non-LBP groups for the incidence of tissue texture changes or static rotational asymmetry. Despite the lack of statistical significance for the incidence of static rotational asymmetry between the two study groups, a distinct difference in the incidence of left rotation versus right rotation was observed between the groups. Moderate/severe left rotation was more likely to occur in subjects with chronic LBP than in subjects without chronic LBP.

Because the chronic LBP and non-LBP groups were not significantly different in terms of the potential confounding factors of age, sex, or BMI, these factors are not likely to be the reason for the observed differences between the groups in incidence and severity of somatic dysfunction. Based on our findings, we believe that palpatory assessment of somatic dysfunction can be a valid measure in physical examinations of patients with chronic LBP.

In previous studies,^{9,10} lumbar gross range of motion has been used to assess LBP. However, those measurements demonstrated poor correlation with back pain symptoms because gross range of motion can change throughout the day, affecting the reproducibility of findings.^{10,11} In a study by Leboeuf-Yde et al¹² that evaluated chiropractic motion-testing procedures similar to the OM motion-testing procedures used in the present study, no consistent association was found between subjects' lumbar motion palpatory findings and history of LBP. However, the study by Leboeuf-Yde et al¹² used student examiners who did not receive consensus training, whereas the present study used DO examiners who were trained extensively before study initiation to establish interexaminer reliability for each individual examination element. In addition, to establish consensus, we always used two DO examiners (from a potential pool of three) to independently examine each subject and then recheck each other's findings. As a result of this consensus training, the present study revealed reproducible symptom findings that occur more frequently and with greater severity in patients with chronic LBP than in those without that condition.

The somatic dysfunction components of static rotational asymmetry and resistance to anterior springing represent altered spinal positioning and vertebral mechanics. These components may be related to other symptoms frequently experienced by individuals with chronic LBP-particularly impaired proprioception and impaired muscular imbalance.¹³⁻¹⁸ These symptoms, in turn, are associated with aberrant neuromuscular firing patterns and difficulty maintaining or finding neutral spinal positioning.^{17,19-21} Asymmetric spinal positioning is common when patients are in the standing or seated position and may be apparent when patients are evaluated in a prone position, such as that used in the present study. Therefore, the somatic dysfunction components of static rotational asymmetry and resistance to anterior springing could result directly in muscular imbalance-or these components could be compensatory to a preexisting imbalance. In individuals with chronic LBP, the somatic dysfunction may be severe enough (indicating abnormal vertebral mechanics) that the body can no longer compensate for the altered biomechanical loading and, thus, becomes symptomatic.

Static rotational asymmetry of individual lumbar vertebrae was found in the present study to be common in both study groups. However, the subjects with chronic LBP had more

Vertebral Segments With Somatic Dysfunction* No. (%)			Severity Rating of Somatic Dysfunction [†] Mean (SD) 95% Confidence Interval			
Palpatory Examination	Chronic LBP (n=64)‡	Non-LBP (n=188)§	P Value ^{//}	Chronic LBP (n=64) [‡]	Non-LBP (n=188)§	P Value [¶]
Tissue texture changes	61 (95.3)	174 (92.6)	.54	2.5 (0.6) 2.3-2.7	2.3 (0.6) 2.2-2.4	.006
Static rotational asymmetr	y 61 (95.3)	166 (88.3)	.21	2.4 (0.6) 2.2-2.6	2.1 (0.6) 2.0-2.3	.008
Resistance to anterior springing	59 (92.2)	133 (70.7)	<.001	2.3 (0.6) 2.1-2.5	1.9 (0.7) 1.8-2.0	<.001
Tenderness	33 (51.6)	29 (15.4)	.002	1.7 (0.8) 1.5-2.0	1.2 (0.4) 1.0-1.3	.001

* Vertebral segments examined were lumbar vertebrae L1-L4.

+ Severity rating based on three-point scale: 1, no somatic dysfunction; 2, mild somatic dysfunction; 3, moderate/severe somatic dysfunction.

‡ Sample size shown is total number of lumbar vertebrae for the 16 subjects in chronic low back pain (LBP) group.

§ Sample size shown is total number of lumbar vertebrae for the 47 subjects in non-LBP group.

// P value represents between-group comparison based on logistic regression fit with generalized estimating equations.

¶ P value represents between-group comparison based on proportional odds model fit with generalized estimating equations.

severe somatic dysfunction, with left vertebral rotation being more frequent than right vertebral rotation. This finding is consistent with the common postural compensatory pattern reported by Zink and Lawson,²² as well as the most common idiopathic lumbar scoliotic curvature (ie, convex to the left or "sidebent" right with left rotation). Zink and Lawson²² reported that the human body can readily adapt to structural asymmetry through compensatory postural changes. The most common compensatory pattern found by Zink and Lawson²² was right pelvic rotation accompanied by compensatory left lumbar rotation, which changes to right thoracic rotation above the thoracolumbar junction and then changes again to left cervical rotation above the cervicothoracic junction.²¹ Each postural change thereby compensates for the previous asymmetry. Therefore, using this model of compensatory changes in the spine, the increased severity of the left rotational preference observed in the chronic LBP group may represent an exaggeration of common postural compensatory findings.

Flexion Somatic Dysfunction and Lumbar Lordosis

Resistance to anterior springing of the vertebrae as a general screening test used to locate somatic dysfunction has relevance for treatment modalities²⁴ and superior reliability to other tests of segmental motion.⁸ In the spine, resistance to anterior springing corresponds with resistance to local extension. Such a finding is consistent with a preference for flexion mechanics, which is known as flexion somatic dysfunction.⁷ However, because of the significant presence of rotational asymmetry observed in the present study, our findings indicate that resistance to anterior springing likely correlates with triplanar dysfunction rather than isolated sagittal plane dysfunction.

Furthermore, because the decrease in local extension alters the normal lordotic curvature and the weight-bearing mechanics of the lumbar spine, flexion somatic dysfunction is of particular importance in the clinical treatment of patients with LBP. Loss of lumbar lordosis is an independent risk factor for LBP.²⁴ When alterations are made to the mechanical loading of vertebral discs, either through congenital anomalies or surgery, degenerative changes develop in time. For example, lumbosacral transitional vertebrae alter lumbar mechanical loading via fusion of the L5 transverse processes to the sacrum, resulting in increased degeneration in the intervertebral disc of L4 and L5.²⁵ This degeneration is also seen after lumbarfusion surgeries.

All types of spinal fusions alter the local mechanical loading of the unfused vertebral segments, leading to profound changes in the biomechanics of the facet joints and intervertebral discs.²⁶ Oda et al²⁷ reported that loss of lumbar lordosis secondary to a kyphotic (flexed) posterolateral lumbar fusion of L3 through L5 in sheep resulted in substantially more degenerative changes in the vertebral segments above the fusion than in the in situ fusion group. In humans, flattening of the lordotic curvature of the lumbar spine after spinal fusion has been correlated with early degenerative changes, spinal stenosis, and LBP.^{28,29}

Because individuals who are asymptomatic for LBP frequently demonstrate incidental degenerative findings in magnetic resonance imaging and radiographic evaluations, finding somatic dysfunction in both the chronic LBP and non-LBP study groups was not surprising. However, the presence of chronic LBP increases with the incidence and severity of degenerative findings.^{30,31} Likewise, because flexion somatic dysfunction, as detected by resistance to ante-

Table 2 Incidence of Right Versus Left Static Rotational Asymmetry, by Severity Rating, for Chronic Low Back Pain and Non–Low Back Pain Groups (N=63)								
Static Rotational Asymmetry		Vertebral Segments Per Subject With Somatic Dysfe No., Mean (SD) 95% Confidence Interval						
Severity Rating ⁺	Direction of Rotation	Chronic LBP (n=16)	Non-LBP (n=47)	P Value‡				
2 or 3	Right	0.4 (1.0) -0.2 to 0.9	0.4 (0.9) 0.2 to 0.7	.70				
	Left	3.4 (1.2) 2.8 to 4.1	3.1 (1.4) 2.7 to 3.5	.31				
	<i>P</i> Value§	<.001	<.001					
3	Right	0.1 (0.3) -0.1 to 0.2	0.1 (0.3) 0 to 0.2	.76				
	Left	1.8 (1.2) 1.2 to 2.4	1.0 (1.1) 0.6 to 1.3	.01				
	P Value [§]	<.001	<.001					

+ Severity rating based on three-point scale: 1, no somatic dysfunction; 2, mild somatic dysfunction; 3, moderate/severe somatic dysfunction. Rows designated by severity rating of 2 or 3 include data when static rotational asymmetry was present either as mild or moderate/severe somatic dysfunction. Rows designated by severity rating of 3 include data when static rotational asymmetry was present only as moderate/severe somatic dysfunction.

‡ P value based on Mann-Whitney U test comparing chronic low back pain (LBP) group with non-LBP group.

§ P value based on Wilcoxon signed rank test comparing right rotation with left rotation.

rior springing, ultimately alters the lordotic curvature of the lumbar spine, it may predispose vertebral segments to degenerative changes.

Limitations

The present study found statistically significant differences in somatic dysfunction between the chronic LBP and non-LBP groups, but certain study limitations must be considered when interpreting our results. Although the number of subjects in the chronic LBP group was small (n=16), four observations were made for each subject in the group (L1-L4), resulting in a large pool of data. Even with an imbalance in sample size between the two groups (the ratio of subjects in the chronic LBP group to subjects in the non-LBP group was approximately 1:3), a large number of observations resulted in narrow CIs. This result allowed small differences between the groups to look compelling, though these differences may not be detectable in clinical settings by DOs who have not undergone the stringent training used by investigators in this study. Therefore, the data must be interpreted conservatively until more subjects have been studied by a larger cohort of DO examiners. We are currently working to reproduce these findings using a much larger sample size at multiple research sites.

Another limitation of the present study was the use of palpatory assessment and verbal cues from subjects to determine tenderness. Dolorimeters are often used in research to objectify palpatory pressure needed to elicit tenderness.³² To

make our results more equivalent to the clinical setting, we chose not to use a dolorimeter.

Conclusion

Based on the results of the present study, it appears that somatic dysfunction is more apparent in individuals with chronic LBP than in individuals without chronic LBP. If this finding is clinically accurate, the palpatory tests used in this study (tissue texture changes, static rotational asymmetry, resistance to anterior springing, and tenderness in L1 through L4) to evaluate subjects with LBP may be valid diagnostic tools in the clinical setting.

By performing quick, simple, and cost-effective palpatory examinations of patients with LBP, osteopathic physicians will be able to better evaluate their patients. However, future research needs to investigate whether osteopathic manipulative treatment reduces the severity of somatic dysfunction found with these palpatory tests, and if there are objective, beneficial correlations among reducing the severity of somatic dysfunction, reducing symptoms, and improving function.

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ORIGINAL CONTRIBUTION

References

1. Cherry DK, Woodwell DA, Rechtsteiner EA. National Ambulatory Medical Care Survey: 2005 Summary. Hyattsville, Md: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics; June 29, 2007. DHHS Publication No. (PHS) 2007-1250. Available at: http://www.cdc.gov/nchs/data/ad/ad387.pdf. Accessed July 25, 2008.

2. Borenstein DG. Epidemiology, etiology, diagnostic evaluation, and treatment of low back pain [review]. *Curr Opin Rheumatol*. 2001;13:128-134.

3. Walker BF. The prevalence of low back pain: a systematic review of the literature from 1966 to 1998. J Spinal Disord. 2000;13:205-217.

 Vuori IM. Dose-response of physical activity and low back pain, osteoarthritis, and osteoporosis [review]. *Med Sci Sports Exerc*. 2001;33(6 suppl):S551-S586.

5. Glossary Review Committee, for the Educational Council on Osteopathic Principles and the American Association of Colleges of Osteopathic Medicine. *Glossary of Osteopathic Terminology*. July 2006. Available at: https://www.doonline.org/pdf/sir_collegegloss.pdf. Accessed July 25, 2008.

6. DiGiovanna EL, Schiowitz S, Dowling DJ. An Osteopathic Approach to Diagnosis and Treatment. 3rd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2005:241-242,275.

7. Degenhardt BF, Snider KT, Snider EJ, Johnson JC. Interobserver reliability of osteopathic palpatory diagnostic tests of the lumbar spine: improvements from consensus training. *J Am Osteopath Assoc.* 2005;105:465-473. Available at: http://www.jaoa.org/cgi/content/full/105/10/465. Accessed July 25, 2008.

8. Stevens JM, Rich PM. The spine: methods of examination. In: Grainger RG, Allison DJ, Adam A, Dixon AK, eds. *Grainger & Allison's Diagnostic Radiology: A Textbook of Medical Imaging*. Vol 3. 4th ed. Oxford, England: Churchill Livingstone; 2001:2412.

9. Ogden CL, Fryar CD, Carroll MD, Flegal KM. *Mean Body Weight, Height, and Body Mass Index, United States 1960-2002*. Hyattsville, Md: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics; October 27, 2004. DHHS Publication No. (PHS) 2005-1250. Available at: http://www.cdc.gov/nchs/data/ad/ad347.pdf. Accessed July 25, 2008.

10. Ensink FB, Saur PM, Frese K, Seeger D, Hildebrandt J. Lumbar range of motion: influence of time of day and individual factors on measurements. *Spine*. 1996;21:1339-1343.

11. Parks KA, Crichton KS, Goldford RJ, McGill SM. A comparison of lumbar range of motion and functional ability scores in patients with low back pain: assessment for range of motion validity. *Spine*. 2003;28:380-384.

12. Leboeuf-Yde C, van Dijk J, Franz C, Hustad SA, Olsen D, Pihl T, et al. Motion palpation findings and self-reported low back pain in a population-based study sample. *J Manipulative Physiol Ther.* 2002;25:80-87.

13. Brumagne S, Cordo P, Lysens R, Verschueren S, Swinnen S. The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. *Spine*. 2000;25:989-994.

14. Danneels LA, Vanderstraeten GG, Cambier DC, Witvrouw EE, De Cuyper HJ. CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. *Eur Spine J.* 2000;9:266-272.

15. Hodges PW, Richardson CA. Inefficient muscular stabilization of the lumbar spine associated with low back pain: a motor control evaluation of transversus abdominis. *Spine*. 1996;21:2640-2650.

16. Leinonen V, Maatta S, Taimela S, Herno A, Kankaanpaa M, Partanen J, et al. Impaired lumbar movement perception in association with postural stability and motor- and somatosensory-evoked potentials in lumbar spinal stenosis. *Spine*. 2002;27:975-983.

17. Ng JK, Richardson CA, Parnianpour M, Kippers V. Fatigue-related changes in torque output and electromyographic parameters of trunk muscles during isometric axial rotation exertion: an investigation in patients with back pain and in healthy subjects. *Spine*. 2002;27:637-646.

18. Radebold A, Cholewicki J, Polzhofer GK, Greene HS. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. *Spine*. 2001;26:724-730.

19. Finneran MT, Mazanec D, Marsolais ME, Marsolais EB, Pease WS. Largearray surface electromyography in low back pain: a pilot study. *Spine*. 2003;28:1447-1454.

20. Newcomer KL, Laskowski ER, Yu B, Johnson JC, An KN. Differences in repositioning error among patients with low back pain compared with control subjects. *Spine*. 2000;25:2488-2493.

21. O'Sullivan PB, Burnett A, Floyd AN, Gadsdon K, Logiudice J, Miller D, et al. Lumbar repositioning deficit in a specific low back pain population. *Spine*. 2003;28:1074-1079.

22. Zink JG, Lawson WB. An osteopathic structural examination and functional interpretation of the soma. *Osteopathic Annals*. 1979;7:433-440.

23. Kuchera WA. Lumbar region. In: Ward RC, ed. Foundations for Osteopathic Medicine. 2nd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2003:727-750.

24. Adams MA, Mannion AF, Dolan P. Personal risk factors for first-time low back pain. *Spine*. 1999;24:2497-2505.

25. Luoma K, Vehmas T, Raininko R, Luukkonen R, Riihimäki H. Lumbosacral transitional vertebra: relation to disc degeneration and low back pain. *Spine*. 2004;29:200-205.

26. Lee CK, Langrana NA. Lumbosacral spinal fusion: a biomechanical study. Spine. 1984;9:574-581.

27. Oda I, Cunningham BW, Buckley RA, Goebel MJ, Haggerty CJ, Orbegoso CM, et al. Does spinal kyphotic deformity influence the biomechanical characteristics of the adjacent motion segments? An in vivo animal model. *Spine*. 1999;24:2139-2146.

28. Jackson RP, McManus AC. Radiographic analysis of sagittal plane alignment and balance in standing volunteers and patients with low back pain matched for age, sex, and size. A prospective controlled clinical study. *Spine*. 1994;19:1611-1618.

29. Tribus CB, Belanger TA, Zdeblick TA. The effect of operative position and short-segment fusion on maintenance of sagittal alignment of the lumbar spine. *Spine*. 1999;24:58-61.

30. Luoma K, Riihimaki H, Luukkonen R, Raininko R, Viikari-Juntura E, Lamminen A. Low back pain in relation to lumbar disc degeneration. *Spine*. 2000;25:487-492.

31. Peterson CK, Bolton JE, Wood AR. A cross-sectional study correlating lumbar spine degeneration with disability and pain. *Spine*. 2000;25:218-223.

32. Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. Report of the Multicenter Criteria Committee. *Arthritis Rheum.* 1990;33:160-172.