The lumbar multifidus : from anatomy to rehabilitation

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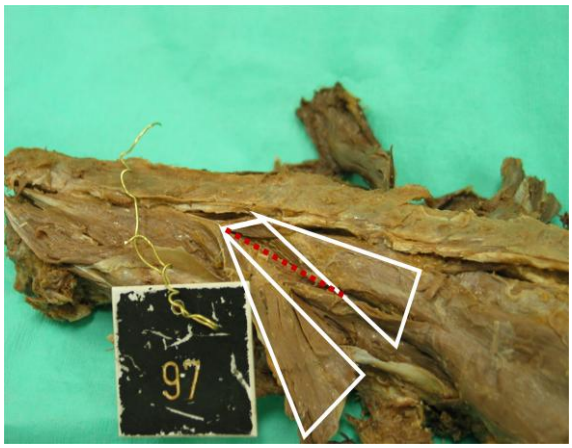
The provision of functional spinal stability involves a complex interaction between many muscles of the trunk and limb girdles. While some muscles perform and control the primary action, other muscles must work in synergy to balance any asymmetrical forces, control unwanted movements, and offer support to articular structures (Twomey & Taylor 1994).
The lumbar multifidus has an unique morphological capacity of providing lumbopelvic stability by controlling intervertebral and sacrovertebral motion.
The morphology of the lumbar multifidus is well described by different authors (Macintosh et al 1986, Twomey & Taylor 1994, Bogduk 1997, Kay 2000, Jemmett et al 2004).The lumbar multifidus muscle is the largest and most medial of the lumbar back muscles. The deepest fibers of the multifidus muscle in the lumbar spine (the laminar fibers) arise from the posteroinferior aspect of each vertebral lamina and articular capsule of the zygapophysial joint and insert into mamillary process of two levels below. The L5 laminar fibres have no mamillary process into which they can insert; instead they insert into an area on the sacrum just above the first dorsal sacral foramen.
The greater muscle mass is from the other five fascicles radiating from the lumbar spinous processes and a common tendon. At each segmental level, a fascicle arises from the base and caudolateral edge of the spinous process, and several fascicles arise, by way of a common tendon, from the caudal tip of the spinous process. Since neither Jemmet et al (n = 1) nor Bogduk (n = 3) studied a large number of cadavers, further work in this regard was warranted (Danneels 2007) .
Recent experiments at our department revealed a more complex architecture of each separate layer, which are partially in agreement with the findings of Jemmet et al (2004). First of all there was a lot of variation in the organisation of the deep laminar fibres. There were fibers crossing one, two and even three levels. The more superficial fibres originating from each separate level had a cone-shaped structure (Fig 1A). Each cone could be subdivided in medial, central and lateral fibers. The medial fibers make a connection with the spinous process of the level below. The central fibers go to the fascia. The lateral fibres go to the superior aspect of the facet joint three levels below. But it was very interesting to see that each cone also consisted of deep fibers that come together with the lateral fibers of the level below. This bi-pennate structure forms a common central tendon that goes to the facet joint (Fig 1B).
Further research on more specimens will be undertaken to evaluate the consistency of these findings and to elaborate on the functional and clinical implications of these connections.
Concerning the function of the MF, there can be stated that the primary role of the laminar fibers is to control intersegmental rotational and shear forces through the exertion of compressive force between segments. The superficial fibers have a combined function: exerting compressive loading of the spine to enhance its stiffness, and producing an effective moment arm for extension of the lumbar spine and control the lumbar lordosis (Danneels 2007). The results of many studies support both functions.
In addition to these functions, the multifidus also attaches to the deep laminae of the posterior thoracolumbar fascia. This occurs through a raphe separating the multifidus and the gluteus maximus (Willard 1997). The anterior border of the raphe is anchored to the SIJ capsule and the posterior border of the raphe becomes part of the thoracolumbar fascia. Tendinous slips of the multifidus pass between the superior band of the sacrotuberous ligament and the long dorsal sacroiliac ligament to join with the sacrotuberous ligament; these connections are thought to integrate the multifidus into the ligamentous support system of the SIJ (Willard 1997). In the pelvis, this muscle is contained

between the dorsal aspect of the sacrum and the deep layers of the thoracodorsal fascia (Vleeming et al 1995).



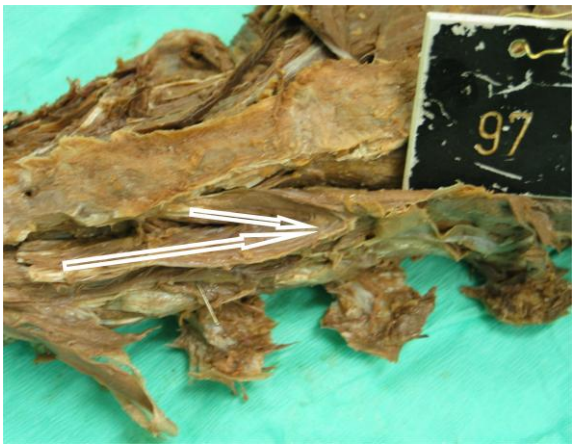


Figure 1A A posterolateral view of the cone shaped layers arising from two subsequent levels consisting of medial, lateral and deep fibers. The upper layer is turned over. The deep fibers that come together with the lateral fibers of the level below (dotted line).
Figure 1B A close-up of the bi-pennate structure that forms a common central tendon that goes to the facet joint.

Dysfunction of the lumbar multifidus

Trunk muscle dysfunction is being implicated as a contributory factor in the development or recurrence of subacute and chronic mechanical back complaints.
Although most studies provide data on gross muscle function, more specific information is required concerning the pattern and degree to which individual muscles contribute to the dysfunction. Last decade, researchers have found that the muscular response to back pain may not be uniform amongst all muscles of the back: it is mainly the action of the deep muscle system that is disturbed and inhibited in the presence of LBP (Hides et al 1994, Hides et al 1996, Hodges & Richardson 1996, Hodges & Richardson 1998, Danneels et al 2000, Barker et al 2004).

Several studies performed at our department investigated changes in muscle recruitment due to acute muscle pain, using experimental pain models. A specific approach to investigate LBP mechanisms is the use of experimentally induced LBP. Inducing pain in otherwise asymptomatic subjects allows investigation of recruitment strategies with and without LBP within one subject. Additionally, inducing deep muscle pain in the lumbar back muscles creates a specific subgroup of patients in which the location of the pain, as well as pain duration and intensity is known. Experimental pain models can help to elucidate some of the motor-control mechanisms which are affected by muscle pain (Graven-Nielsen et al 2000), as the cause-effect relationship of low back muscle pain and motor control changes can be investigated. (Graven-Nielsen 2006)
The effect of induced pain by intramuscular injection with hypertonic saline was investigated during *automatic contractions*, and both *low and high load voluntary contractions*.

The effect of pain on muscle recruitment during the voluntary trunk extension exercises (both low and high load) was investigated with muscle functional MRI (mfMRI). This technique enables to determine which muscles or regions within muscles were activated during a functional task (Dickx et al – in press). The results of the experiment including low load contractions indicate that unilateral

and unisegmental pain induction, inhibits the m. multifidus, m. erector spinae lumborum and m. psoas, bilateral and at multiple levels (Dickx et al 2008).
The same trunk extension exercise was investigated at a high load intensity. In contrast to the results of the low load trunk extension exercise, no significant changes in muscle recruitment caused by pain were found (Dickx et al – in press).
Both studies examined voluntary muscle recruitment, whereas, in a third experiment automatic recruitment during pain was evaluated. This was done by use of ultrasound since the mfMRI is not sensitive enough for low intensities reached during automatic muscle contractions. An arm lifting task was used to activate the dorsal muscle chain, including the m. multifidus. The results indicate that localized unilateral muscle pain induces an immediate inhibition of the m. multifidus, which was observed at both sides of the body and at multiple levels (Dickx et al 2010).

Based on these results, 3 different aspects will be further discussed: 1) inhibition of the deep muscle system due to pain, 2) task and load dependency of muscle changes and 3) selective or generalized pain mechanisms. In addition, potential clinical implications of our studies will be discussed.

1. Inhibition of the deep muscle system due to pain

There is vast evidence of impairment of the m. multifidus in LBP patients. (Hides et al 1994, Mannion 1999, Zao et al 2000, Danneels et al 2000, Kader et al 2000, Macdonald et al 2009) Consequently, pain models predict that pain has a consequent effect on the deep spinal muscles, which manifests as inhibition. (Hodges and Moseley 2003, Hodges 2004) However, the evidence of the effect of pain on the m. multifidus is lacking as experimental studies could not consistently replicate the changes in the m. multifidus as observed in patients. (Hodges and Moseley 2003, Hodges et al 2003)
Our results show that experimentally provoked LBP can induce inhibition of the deep paraspinal muscles which underline the hypothesis that pain in itself can induce inhibition of the m. multifidus.
To date, attention has been focused on two deep stabilizing muscles: the m. multifidus and m. transversus abdominis. Nevertheless, it is apparent that also other muscles have similar functions.
There is increasing evidence that the m. psoas shares characteristics with the lumbar m. multifidus regarding spinal stabilization (Penning 2000, Comerford and Mottram 2001, Jemmet et al 2004 Gibbons 2005) and different studies demonstrated wasting of this muscle in LBP patients. (Cooper et al 1992, Dangaria and Naesh 1998, Kader et al 2000, Barker et al 2004) Nevertheless, the mechanism of impairment of the m. psoas remains unknown. Our study provides evidence that pain can induce inhibition of the m. psoas. To the best of our knowledge, we are the first to demonstrate the effect of pain on the m. psoas. Our finding is supported by a clinical LBP study which demonstrates a positive correlation between the percentage decrease in CSA of the m. psoas and the rating of pain in patients. (Barker et al 2004)
Another muscle which can labelled as a deep stabilizing muscle system is the m. erector spinae pars lumborum. (Macintosh and Bogduk 1987, Comerford and Mottram 2001, Bogduk 2005, Danneels et al 2007, Danneels 2007) In most studies no differentiation has been made between the lumbar and thoracic parts of the m. erector spinae, despite the evidence of differential anatomical and biomechanical characteristics of both parts. (Bogduk 2005, Danneels 2007) Our results demonstrate that pain can inhibit the lumbar part of the m. erector spinae. This may underline the importance to distinguish between the lumbar and thoracic part of the m. erector spinae and may, at least partly, explain the large variability in results in literature concerning the m. erector spinae. (Hodges and Moseley 2003, van Dieen et al 2003)
Taken together, our studies provide evidence for the hypothesis that pain can induce inhibition of the deep muscular system (Hodges and Moseley 2003) and indicate that the inhibition is not only limited to the m. multifidus but to all the muscles sharing characteristics of the deep muscular system such as the m. psoas and the m. erector spinae lumborum.
The pain model proposed by Hodges et al predicts not only decreased activity of the deep muscles, but also increased activity of at least one superficial muscle. (Hodges and Moseley 2003, Hodges

2004, Hodges 2003a) In our study, only deep muscles were investigated, therefore it is not known if pain also induced changes in the superficial muscles. Based on our results, especially these of the low load trunk extension exercise, it can be assumed that there are compensation mechanisms, as the output did not change i.e. also in the pain condition the task was performed. The possible compensation strategies are currently addressed in new experiments.

2. Task and load dependency of muscle changes

It has been argued that the effect of pain on muscle recruitment is task and load dependent. (Graven-Nielsen et al 2000, Hodges and Moseley 2003, van Dieen et al 2003, Falla et al 2007, Graven-Nielsen and Arendt-Nielsen 2008, Djupsjöbacka et al 2008) Therefore, in our experiments muscle activity was investigated during different tasks: voluntary low load trunk extension, voluntary high load trunk extension, and automatic recruitment during arm lifting.
Pain-induced changes in lumbar muscle recruitment were demonstrated during arm lifting and low load trunk extension, but not during high load trunk extension, which may suggest that muscle changes due to pain are different depending on the load.
This is in line with the current view on muscle changes due to musculoskeletal pain. It is hypothesized that pain causes a dynamic reorganization of muscle recruitment, which is task and load dependent. It is assumed that the underlying aim of the muscle reorganization is protection of the painful part, i.e. avoiding the use of the painful muscles or painful movement, without disruption of the demanded task. (Falla et al 2007, Hodges 2008) This may explain why there is decreased activity of the deep muscles in the low load but not in the high load trunk extension exercise. It is hypothesized that during a high load task all muscles are needed to perform the movement, leaving no option for a changed recruitment strategy.
This would implicate that the alterations in neuromuscular control would only become evident in low load tasks. This hypothesis is supported in literature (Commerford and Mottram 2001), but further research is necessary, as to date most studies have focused on low load tasks and coordination tasks e.g. gait (Arendt-Nielsen et al 1996, Hodges et al 2003, Moseley et al 2003, Lamoth et al 2004, Macdonald et al 2009), whereas studies investigating the effect of LBP on muscle recruitment during high load dynamic exercises are limited. (Danneels et al 2002)
A possible underlying mechanism to explain the changes under low load conditions, is that inhibition selectively affects the lower threshold motor units. (Hodges et al 2008) Low threshold motor units innervate slow twitch (type I) muscle fibers. (Henneman et al 1965, Mannion 1999) This could explain why there is a significant decrease in muscle activity during low load tasks, where in particular type I fibers are recruited, whereas in high load tasks the contribution of the slow twitch fibers relative to the fast twitch fibers may be negligible. It has been proposed before that type I muscle fibers are more susceptible to the adverse aspects of pain and immobilization than type II fibers. (Appell 1990) Additionally, there is evidence that atrophic changes in muscles are not uniform and are more likely to affect slow twitch muscle fibers. (Meyer et al 2005, Jull et al 2008)
Another element, which could have played a role in the differential changes during the low load and high load trunk extension, is the intensity of the perceived pain. In both experiments the pain intensity was comparable, however, it might be possible that during high load tasks, the pain intensity needs to be higher in order to influence the muscle recruitment. The influence of intensity of pain on muscle recruitment during different task should be investigated in further research.

3. Selective or generalized pain mechanisms?

As mentioned before, there is vast evidence of impairment of the m. multifidus in LBP. Changes in muscle morphology and in muscle function have been identified in acute, (Hides et al 1994, Hides et al 1996) recurrent (Macdonald et al 2009) as well as in chronic LBP (Danneels et al 2000, Danneels et al 2002) patients. Despite this broad evidence of muscle impairment, some important questions remain unresolved.

First, it is not known if LBP affects the m. multifidus selectively, i.e. at the side and level of symptoms or more widespread, as there is evidence for both propositions. Second, it remains unknown whether the observed structural muscle changes are a result of functional adaptations, or in contrast, precede the functional impairment. And third, it is not yet clarified to what extent the observed muscular changes in the acute phase of LBP may play a role in the pathophysiology of recurrent and chronic LBP.

Our experimental studies (Dickx et al 2008 – Dickx et al 2010) indicate that acute unilateral pain can induce widespread changes in muscle recruitment. Inhibition was demonstrated bilateral and at multiple segments in all investigated muscles during respectively, low load voluntary contraction and automatic contraction.
In contrast to our study, Hides et al (1994) demonstrated segmental and unilateral changes in the m. multifidus in acute unilateral LBP patients. These seemingly conflicting findings are probably not that contradictory, but may provide new insights within the different mechanisms that play a role in LBP.
Hodges et al (2006) set up a study to elucidate the mechanism of fast onset of selective reduction in CSA of the m. multifidus in acute unilateral LBP. In a laboratory study, experimental disc and nerve root lesions were made in anesthetized pigs, and the effect on the m. multifidus CSA was investigated with ultrasound. In addition, muscle biopsies from the m. multifidus were taken to investigate the effect of injury on histo-chemical parameters i.e. muscle water content, lactate concentration, and fat distribution.
The results showed a rapid (within 3 days), selective reduction in CSA of the m. multifidus at the level and side of the disc injury. These data confirm the possibility for rapid atrophy to occur in LBP at a single segment.
The underlying mechanisms for these selective changes, however, could not be elucidated. The changes following disc injury cannot be explained by denervation, as the distribution of atrophy is different following nerve dissection. Following nerve dissection (section of the medial branch of the dorsal ramus, which innervates the m. multifidus), the CSA was not reduced over 1 but over 3 lumbar segments.
Histo-chemical analysis of the muscle biopsies revealed reduced water content and lactate concentration, along with increased adipocytes. However, these changes cannot explain the acute selective changes in CSA, as these histo-chemical changes were present at multiple levels and at both sides of the m. multifidus.
To explain the rapid, selective reduction in CSA following disc injury, it was hypothesized that the neural drive to the m. multifidus may be reduced by an inhibitory process, such as reflex inhibition. (Hodges et al 2006)
Using this injury-model, the role of pain remains unknown. Our studies, using a pain-model, provide support that pain does not induce selective, but rather induces widespread changes in muscle function. This is supported by other pain studies in diverse muscles, which found that the effect of nociceptor stimulation is not localized, but has a rather broad effect on muscles. (Hodges et al 2008, Falla et al 2007a)
These findings lead to the hypothesis that pain and injury may differentially affect the m. multifidus. In short term, pain may lead to more generalized changes in muscle function, while injury (with disc or nerve root involvement) may lead to more specific changes of the m. multifidus. Importantly, in LBP, both injury and pain mechanisms are likely to occur concurrently. This supposition can explain the seemingly contradictory findings in acute LBP and can provide a link to chronic LBP. The hypothesis is represented in diagram form, in figure 2.

In chronic LBP patients, the majority of studies indicate bilateral and multilevel atrophy of the m. multifidus. (Cooper et al 1992, Parkkola et al 1993, Kader et al 2000, Kamaz et al 2007) The contradiction between acute selective changes and chronic generalized changes has not been elucidated in literature. Our hypothesis may provide an explanation, as we found that in the acute stage pain may provoke widespread functional changes, which may lead to more generalized

adaptations in the long term which may be structural and/or functional. This hypothesis should be further investigated.

	Acute LBP	*Long- term*	**Recurrent - Chronic LBP**
Injury	Selective changes Structure/Function	⇨	Selective changes Structure/Function
Pain	Widespread changes Function		Widespread changes Structure/Function

Figure 2: Schematic representation of muscle adaptations in LBP. It is hypothesized that pain and injury have differential effects on the m. multifidus. On long term, changes in function may lead to structural adaptations and visa versa.

In summary, our experimental pain studies lead to a hypothesis which may provide new insights with regard to the underlying physiological mechanisms in LBP.
First, it is suggested that the findings of rapid selective inhibition in the m. multifidus in acute unilateral LBP patients does not have to be contradictory to our findings of more generalized changes in the m. multifidus due to induced unilateral LBP. It is assumed that selective and more generalized mechanisms, related to injury and pain respectively can occur concurrently. However, this has to be elucidated in further research.
Second, we propose that the immediate onset of changes in structure as well as function in acute LBP may result in long term changes. Consequently, with regard to the question of the cause-effect relationship of structural and functional changes in LBP, we suppose that both mechanisms may play a role; structural changes in LBP can be a result of long-term functional changes and visa versa.
Third, we put forward that immediate generalized inhibition of the m. multifidus due to pain (as observed in our studies) may precede the bilateral and multilevel atrophy as observed in chronic LBP patients, and therefore, might be a factor in the development of recurrent and chronic LBP.
Our hypotheses underline the complexity of mechanisms and multiple interactions within the LBP pathology. Moreover, only physiological mechanisms are discussed above, while also psychological mechanisms interact, especially in patients, making the LBP pathology even more complex. The multifariousness of mechanisms may explain why it remains so difficult to unravel the complex puzzle of LBP.

Implications for rehabilitation

Although the results of our experimental studies can not be directly translated to the LBP population, our results may be of clinical relevance, as they underlie some theoretical key points for therapeutic exercise selection for the rehabilitation of muscle impairment in LBP patients.
The results of our studies indicate that in patients, pain may induce inhibition of the deep stabilizing muscle system. As explained in the introduction the deep stabilizing system is very important for the fine tuning of stabilization. Loss of fine tuning may compromise optimal spinal function and lead to overload and injury. (Panjabi 1992, Hodges 2004a) Therefore, from a clinical perspective, our studies support current rehabilitation strategies which use specific stabilizing exercises (Hides et al 2001, O'Sullivan 2000) to restore normal muscle function.
Most rehabilitation strategies focused on the m. multifidus and m. transversus abdominis, whereas our results demonstrate that attention should also be given to the evaluation and rehabilitation of

the m. psoas and the lumbar portion of the m. erector spinae. Further research is required to investigate the clinical relevance of addressing these muscles in patients.

Progressive exercise program

As discussed above, structural as well as functional changes in the lumbar back muscles have been identified in LBP. Therefore, therapeutic exercises should address both structural and central neuromuscular adaptations in patients. (Jull et al 2008) Possibly, low level activation of the impaired muscles is not sufficient to reverse structural adaptations e.g. atrophy in the lumbar muscles.
The efficacy of low load stabilization exercises on restoring motor control and selective changes in the m. multifidus has been demonstrated in clinical trials with acute LBP patients. (Hides et al 2009)
However, high load exercises may be necessary to restore the structural changes in chronic LBP patients. Danneels et al (2001) demonstrated that in chronic LBP patients, low level activation is not sufficient to reverse m. multifidus atrophy; only a stabilization training program in combination with a strengthening program was efficient to restore it's size.
Also on the basis of the long-term pain and disability level, the combination of stabilization exercises and intensive lumbar resistance training was advised (Danneels 2008). Therefore progressive exercise training with increasing load may be advised to restore normal muscle function and structure. Importantly, the type, load, and frequency of the exercises should be tailored towards the individual patient.
Based on the results of our recent studies, which demonstrate inhibition of the deep paraspinal muscles during pain in the low load but not in the high load exercise, it is tempting to postulate that in patients it may be beneficial to work at high load levels, to facilitate normal recruitment. However, two remarks have to be made. First, in LBP patients, there are more mechanisms which have to be taken into account, for example the possibility of (re-)injury. Second, high load activity exercises may be beneficial to reverse atrophy in chronic LBP patients and to have long-term effects on pain and disability, as demonstrated by Danneels et al (2001, 2008, 2008a), but it is questionable if high load activity facilitates normal low load tonic recruitment of the deep muscle system, which is a prerequisite for healthy spinal function. Therefore the authors want to stress the importance of the combination and integration of strengthening training. Moreover there can no doubt about the statement that the treatment needs to be individual tailored based on a well performed examination that screens for all the relevant aspects.

LBP is a complex disorder in which muscle dysfunction seems to play an important role. Currently, multifidus dysfunction is being increasingly implicated as a contributory factor in the development or recurrence of subacute and chronic mechanical back complaints.
The current contribution wants to give insight into the anatomical structure of the multifidus and its response to LBP as crucial components for the development of accurate preventive and intervention strategies for LBP patients.

Literature

1. Appell HJ. Muscular atrophy following immobilisation. A review. *Sports medicine (Auckland, N.Z* 1990;10:42-58.
2. Arendt-Nielsen L, Graven-Nielsen T, Svarrer H, et al. The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. *Pain* 1996;64:231-40.
3. Barker KL, Shamley DR, Jackson D. Changes in cross-sectional area of multifidus and psoas in patients with unilateral back pain. The relation to pain and disability. Spine 2004;29:E515-E519.
4. Bogduk N. Clinical anatomy of the lumbar spine and sacrum. Edinburgh, Churchill Livingstone,1997.
5. Bogduk N. The lumbar muscles and their fasciae. In Bogduk N ed. *Clinical Anatomy of the Lumbar Spine and Sacrum*. Fourth Edition ed: Elsevier - Chirchil Livingstone, 2005:97-121.
6. Comerford MJ, Mottram SL. Movement and stability dysfunction--contemporary developments. *Manual therapy* 2001;6:15-26.

7. Cooper RG, St Clair Forbes W, Jayson MI. Radiographic demonstration of paraspinal muscle wasting in patients with chronic low back pain. *British journal of rheumatology* 1992;31:389-94.
8. Dangaria TR, Naesh O. Changes in cross-sectional area of psoas major muscle in unilateral sciatica caused by disc herniation. *Spine* 1998;23:928-31.
9. Danneels LA, Vanderstraeten GG, Cambier DC, Witvrouw EE. De Cuyper HJ. Clinical science award 2000: CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. Eur. Spine J 2000; 9:266-72.
10. Danneels LA, Vanderstraeten GG, Cambier DC, Witvrouw EE, Stevens VK, De Cuyper HJ. A functional subdivision of hip, abdominal, and back muscles during asymmetric lifting. Spine 2001;26:E114-E121.
11. Danneels LA, Vanderstraeten GG, Cambier DC, et al. Effects of three different training modalities on the cross sectional area of the lumbar multifidus muscle in patients with chronic low back pain. *British journal of sports medicine* 2001;35:186-91.
12. Danneels LA, Coorevits PL, Cools AM, Vanderstraeten GG, Cambier DC, Witvrouw EE, De Cuyper HJ. Differences in multifidus and iliocostalis lumborum activity between healthy subjects and patients with subacute and chronic low back pain. Eur Spine J 2002;11:13-19.
13. Danneels L. Clinical anatomy of the multifidus. In Vleeming & Stoeckaert (eds) : Movement, Stability and Low Back.Pain, Elsevier, 2007: 85-94.
14. Danneels L, Vanderstraeten G, De Cuyper HJ. Functional Spinal Stability: The Role of the Back Muscles. In Lewandrowski K-U, Yaszemski MJ, Kalfas IH, et al. eds. *Spinal Reconstruction. Clinical Examples of Applied Basic Science, Biomechanics and Engineering.* New York: Informa Healthcare 2007:91-108.
15. Danneels L. Stabilisation exercises with and without trunk muscle reconditioning : a five years follow-up. In proceedings IFOMT 2008a Rotterdam, abstract 0061A.
16. Dickx N., Cagnie B., Achten E., Vandemaele P., Parlevliet T., Danneels L. Changes in lumbar muscle activity because of induced muscle pain evaluated by muscle functional magnetic resonance imaging. Spine 2008;33(26):E983-989.
17. Dickx N., Lavens A., Cagnie B., Parlevliet T., Danneels L. The effect of unilateral muscle pain on recruitment of the lumbar multifidus during automatic contraction. An experimental pain study. Man Ther 2010 (doi:10.1016/j.math.2010.02.002).
18. Dickx N., D'hooge R., Cagnie B., Deschepper E., Verstraete K., Danneels L. Magnetic resonance imaging and electromyography to measure lumbar back muscle activity. Accepted for publication in Spine.
19. Djupsjöbacka M, Falla D, Farina D, et al. Part III Effects of Muscle Pain on Motor Function. In Graven-Nielsen T, Arendt-Nielsen L, Mense S eds. *Fundamentals of Musculoskeletal Pain* Seattle: IASP Press, 2008:369-475.
20. Falla D, Farina D, Dahl MK, et al. Muscle pain induces task-dependent changes in cervical agonist/antagonist activity. *Journal of applied physiology (Bethesda, Md.* 2007;102:601-9.
21. Falla D, Farina D, Graven-Nielsen T. Experimental muscle pain results in reorganization of coordination among trapezius muscle subdivisions during repetitive shoulder flexion. *Exp Brain Res* 2007a;178:385-93.
22. Gibbons S. The integration of the psoas major and the deep sacral gluteus maximus muscles into the lumbar cylinder model. World Congress on Manual Therapy. Rome, Italy, 2005.
23. Graven-Nielsen T, Svensson P, Arendt-Nielsen L. Effect of Muscle Pain on Motor Control: A Human Experimental Approach. Advances in Physiotherapy 2000;2:26-38.
24. Graven-Nielsen T. Fundamentals of muscle pain, referred pain, and deep tissue hyperalgesia. Scand J Rheumatol Suppl 2006:1-43.
25. Graven-Nielsen T, Arendt-Nielsen L. Impact of clinical and experimental pain on muscle strength and activity. *Curr Rheumatol Rep* 2008;10:475-81.
26. Henneman E, Somjen G, Carpenter DO. Functional Significance of Cell Size in Spinal Motoneurons. *J Neurophysiol* 1965;28:560-80.
27. Hides J, Stokes M, Saide M, Jull G, Cooper D. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine 1994;19:165-72.
28. Hides J, Richardson C, Jull G. Multifidus recovery is not automatic following resolution of acute first episode of low back pain. Spine 1996;21:2763-9.
29. Hides JA, Jull GA, Richardson CA. Long-term effects of specific stabilizing exercises for first-episode low back pain. *Spine* 2001;26:E243-8.
30. Hides JA, Stanton WR, Wilson SJ, et al. Retraining motor control of abdominal muscles among elite cricketers with low back pain. *Scand J Med Sci Sports* 2009.
31. Hodges P, Richardson C. Inefficient muscular stabilization of the lumbar spine associated with low back pain: A motor control evaluation of transversus abdominis. Spine 1996;21:2640-50.
32. Hodges P, Richardson C. Delayed postural contraction of the transversus abdominis in low back pain associated with movement of the lower limb. J Spinal Disord 1998;11:46-56.
33. Hodges PW, Moseley GL. Pain and motor control of the lumbopelvic region: effect and possible mechanisms. *J Electromyogr Kinesiol* 2003;13:361-70.
34. Hodges PW. Core stability exercise in chronic low back pain. *Orthop Clin North Am* 2003a;34:245-54
35. Hodges P. Pain Models. In Richardson CA, Hodges P, Hides J eds. *Therapeutic Exercise for Lumbopelvic Stabilization. A Motor Control Approach for the Treatment and Prevention of Low Back Pain.* Second edition ed: Churchill Livingstone, 2004:129-37.
36. Hodges P. Lumbopelvic stability: a functional model of the biomechanics and motor control. In Richardson CA, Hodges P, Hides J eds. *Therapeutic Exercise for Lumbopelvic Stabilization*. Second ed: Churchill Livingstone, 2004a:13-28.
37. Hodges PW, Moseley GL, Gabrielsson A, et al. Experimental muscle pain changes feedforward postural responses of the trunk muscles. *Exp Brain Res* 2003;151:262-71.
38. Hodges P. Changes in Sensorimotor Control in Low Back Pain In Graven-Nielsen T, Arendt-Nielsen L, Mense S eds. *Fundamenstals of Musculoskeletal Pain*. Seattle: IASP Press, 2008:445-59.
39. Hodges PW, Ervilha UF, Graven-Nielsen T. Changes in motor unit firing rate in synergist muscles cannot explain the maintenance of force during constant force painful contractions. *The journal of pain* 2008;9:1169-74.
40. Hodges P, Holm AK, Hansson T, et al. Rapid atrophy of the lumbar multifidus follows experimental disc or nerve root injury. *Spine* 2006;31:2926-33.
41. Jemmett RS, MacDonald DA, Agur AMR. Anatomical relationships between selected segmental muscles of the lumbar spine in the context of multi-planar segmental motion: a preliminary investigation. Manual Therapy 2004;9:203-210.

42. Jull G, Sterling M, Falla D, et al. Alterations in Cervical Muscle Function in Neck Pain *Whiplash, Headache, and Neck Pain. Research-based directions for physical therapies.*: Chruchill Livingstone, 2008:41-58.
43. Kader DF, Wardlaw D, Smith FW. Correlation between the MRI changes in the lumbar multifidus muscles and leg pain. *Clinical Radiology* 2000;55:145-9.
44. Kamaz M, Kiresi D, Oguz H, et al. CT measurement of trunk muscle areas in patients with chronic low back pain. *Diagn Interv Radiol* 2007;13:144-8.
45. Kay AG. An extensive literature review of the lumbar multifidus: anatomy. Journal of manual and manipulative therapy 2000;8:102-114.
46. Lamoth CJ, Daffertshofer A, Meijer OG, et al. Effects of experimentally induced pain and fear of pain on trunk coordination and back muscle activity during walking. *Clin Biomech (Bristol, Avon)* 2004;19:551-63.
47. Macdonald D, Moseley GL, Hodges PW. Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. *Pain* 2009.
48. Macintosh J, Bogduk N. Volvo Award in Basic Science: The morphology of the lumbar erector spinae. Spine 1987;12:658-68.
49. Macintosh J, Valencia F, Bogduk N, Munro R. The morphology of the human lumbar multifidus. Clin Biomech 1986;1:196-204.
50. Mannion AF. Fibre type characteristics and function of the human paraspinal muscles: normal values and changes in association with low back pain. *Journal of electromyography and kinesiology* 1999;9:363-77.
51. Meyer DC, Pirkl C, Pfirrmann CW, et al. Asymmetric atrophy of the supraspinatus muscle following tendon tear. *J Orthop Res* 2005;23:254-8.
52. Moseley GL, Hodges PW, Gandevia SC. External perturbation of the trunk in standing humans differentially activates components of the medial back muscles. *J Physiol* 2003;547:581-7.
53. O'Sullivan PB. Lumbar segmental 'instability': clinical presentation and specific stabilizing exercise management. *Man Ther* 2000;5:2-12.
54. Panjabi M. The stabilising system of the spine: Part I, Function, dysfunction, adaptation and enhancement. Part II, Neutral zone and instability hypothesis. J Spinal Disord 1992;5:383-97.
55. Parkkola R, Rytokoski U, Kormano M. Magnetic resonance imaging of the discs and trunk muscles in patients with chronic low back pain and healthy control subjects. *Spine* 1993;18:830-6.
56. Penning L. Psoas muscle and lumbar spine stability: a concept uniting existing controversies. Critical review and hypothesis. *Eur Spine J* 2000;9:577-85.
57. Twomey L, Taylor J (Eds) : Physical Therapy of the low back. (2nd ed.) New York, Churchill Livingstone, 1994.
58. van Dieen JH, Selen LP, Cholewicki J. Trunk muscle activation in low-back pain patients, an analysis of the literature. *Journal of electromyography and kinesiology* 2003;13:333-51.
59. Vleeming A, Pool-Goudzwaard A, Stoeckart R, van Wingerden J, Snijders C. Posterior layer of the thoracolumbar fascia: its function in load transfer from spine to legs;Spine 1995;20:753.
60. Willard FH. The muscular, ligamentous and neural structure of the low back and its relation to back pain. In: Vleeming A, Mooney V, Dorman T, Snijders C, Stoeckart R (eds) Movement, stability and low back pain. Churchill Livingstone, Edinburgh, 1997:3.
61. Zhao WP, Kawaguchi Y, Matsui H, et al. Histochemistry and morphology of the multifidus muscle in lumbar disc herniation: comparative study between diseased and normal sides. *Spine* 2000;25:2191-9.