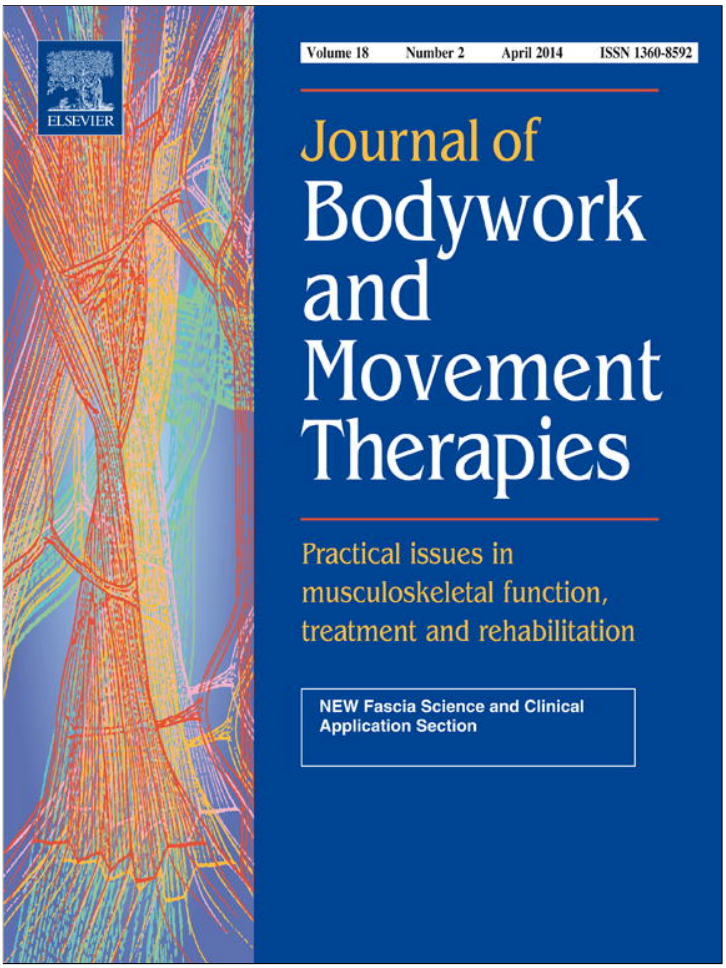

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CONFERENCE REPORT

Selected personal highlights of the 8th interdisciplinary world congress on low back and pelvic pain, Dubai, October 2013

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This was a highly successful conference with around 1000 delegates. An impressive array of speakers presented their work through either plenary or 10 min parallel sessions. With 196 posters, these were thankfully presented in electronic format.

Certain topics were popular with a number of related papers. I'd like to cover: the thoraco-lumbar fascia; pelvic girdle pain; the active straight leg raise test; the fascial system, neuroplasticity and motor control; subgroup classification – and breathing.

The thoraco lumbar fascia (TLF) (Stecco, 2013)

i. The fascial architecture of the back is complicated. Essentially there are 3 layers – superficial, intermediate and deep, all with connections to the head limbs thorax and abdomen. Among these layers there are 'lines of fusion' – well defined points where the muscles and fascia of one layer merge with muscles and fascia of an adjacent layer. These lines of fusion guarantee the co-ordination among the various muscle groups

The TLF is multilayered with multidirectional fibres in the line of muscle pulls.

The three layers must be free to slide between each other – enabled by loose connective tissue and hyaluronic acid lubrication between each layer. The nerves and vessels are usually found in the loose connective tissue. A normal 'sliding system' stimulates the mechanoreceptors providing valuable sensory CNS input.

The superficial layer of the deep fascia is a thin fibro-elastic layer with a dense innervation. It invests the trapezius, latissimus dorsi, and gluteus maximus and includes the posterior lamina of the TLF – a big retinaculum connecting the two halves of the body with the upper and lower limbs. The TLF provides a definite coupling between latissimus dorsi and the contralateral gluteus maximus – important in trunk rotation and stabilisation of the lumbo-pelvis – notably during contralateral limb movements in walking and running.

The middle layer is thicker with a primary mechanical function – and proprioceptive.

The deep layer surrounds the erector spinae and within this, multifidus and the interspinales have their own individual fascial layers.

The anterior layer of the TLF can also be included in this layer and gives insertion to the transversus abdominis and internal oblique – thus contributing to 'core control' mechanisms.

Both deep and superficial layers have a midline adherence at the inter-spinous ligaments.

Increased viscosity of the connective tissues decreases layer 'slide' and causes mechanoreceptor hyper-stimulation and nociception (Figs. 1 and 2).

ii. There were 2 papers on the Lumbar Inter-Fascial Triangle (LIFT)
- The first described the anatomy of the lateral raphe of the TLF (Schuenke et al., 2013) – and more

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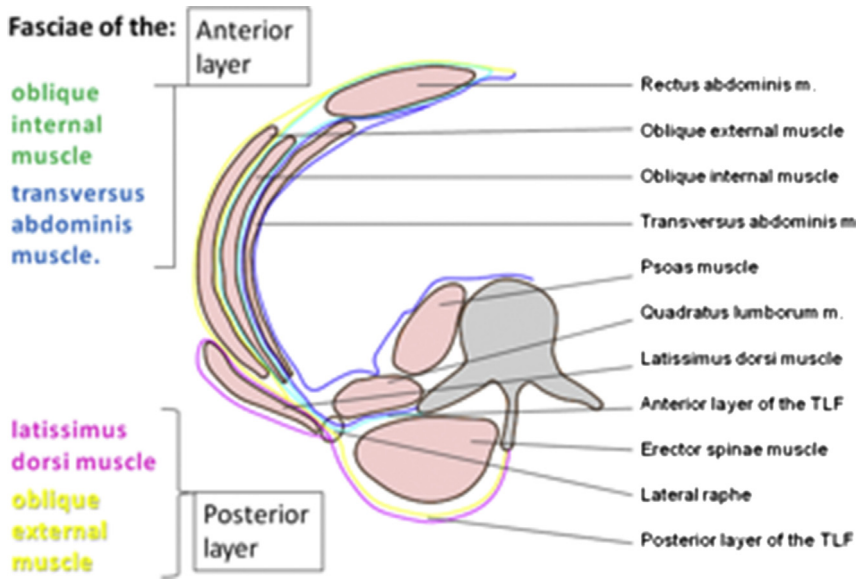

Figure 1 Components of the thoracolumbar fascia.

specifically, the interface between the aponeurosis of the deep abdominal muscles and the Paraspinal Retinacular Sheath (PRS) which splits into distinct anterior and posterior laminae. This junction forms the fat filled LIFT — the 'core' of the lateral raphe, which represents a dense fascial seam along the lateral border of the paraspinals, evident from the 12th rib to the iliac crest. The authors also confirmed the continuity of this fascial sheath from the spinous process to the transverse process — the PRS — thus forming a fibro-osseous tube. See Figs. 3 and 4.

- The second study, (Vleeming et al., 2013) simulated force transmission across the TLF, the hydraulic amplifier effect of the paraspinal fascial 'tube' and it's functional interplay with the 'abdominal myo-fascial ring/girdle' To do this they inflated the para muscuscular TLF container to simulate contraction of the multifidi and the erector muscles, combined with/out tensioning the common transversus tendon (CTrA). If only the extensors and multifidi work (inflation), especially the posterior layer of the TLF moves posteriorly and laterally. However, In conjunction with

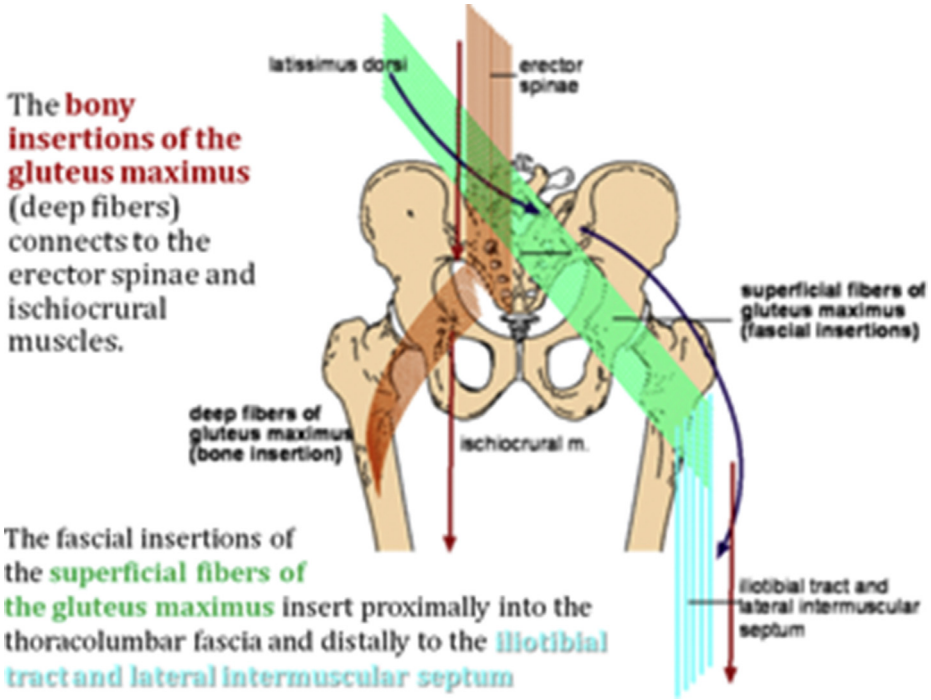

Figure 2 Mechanical transmission of forces between trunk and inferior limbs through fascial connections.

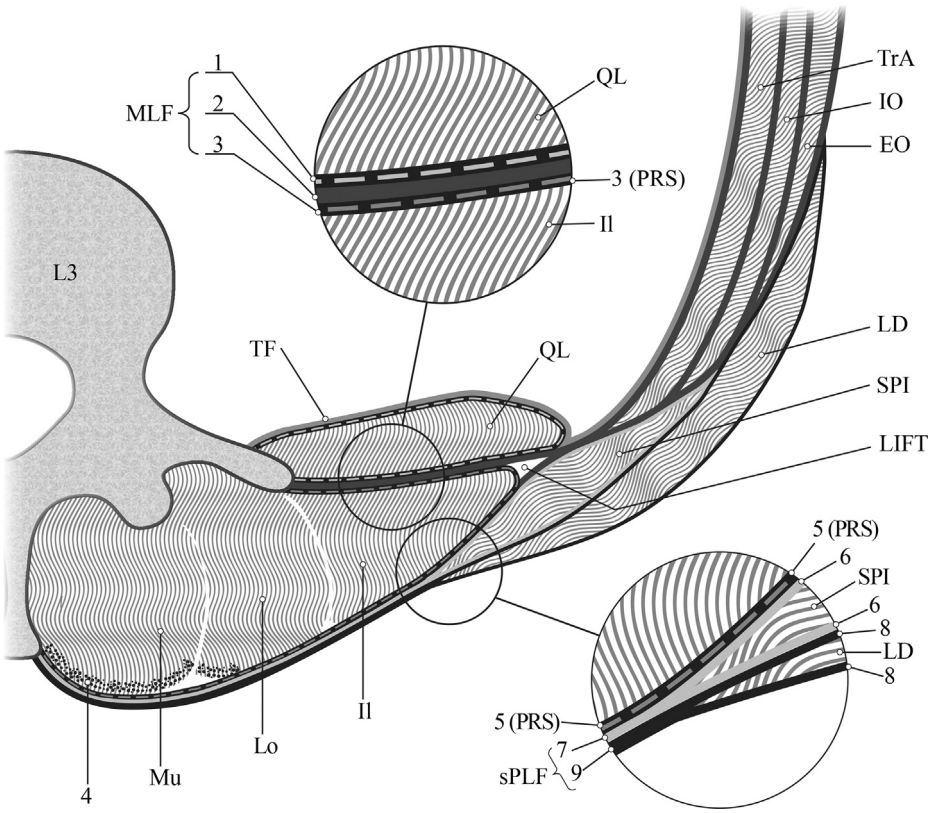

Figure 3 Modified with permission from Willard et al. (2012) Figure 9 in: The thoracolumbar fascia: Anatomy function and clinical considerations. This is a transverse section of the posterior (PLF) and middle layer (MLF) of the thoracolumbar fascia (TLF) and related muscles at the L3 level. Fascial structures are represented such that individual layers are visible, but not necessarily presented to scale. Please note that the serratus posterior inferior (SPI) often is not present caudal to the L3 level. The transversus abdominis (TrA) muscle is covered with a dashed line on the peritoneal surface illustrating the transversalis fascia (TF). This fascia continues medially covering the anterior side of the investing fascia of the QL, quadratus lumborum. Anteriorly and medially, the transversalis fascia (TF) also fuses with the psoas muscle fascia (not drawn). The Internal (IO) and External Oblique (EO) are seen external to TrA. SPI is highly variable in thickness and, more often than not, absent on the L4 level. Latissimus Dorsi (LD) forms the superficial lamina of the PLF together with the SPI, when present. The three paraspinal muscles, multifidus (Mu), longissimus (Lo) and iliocostalis (Il) are contained within the paraspinal retinacular sheath (PRS). The aponeurosis (tendon) of the paraspinal muscles (4) is indicated by stippling. Please note that the epimysium of the individual spinal muscles is very thin and follows the contours of each separate muscle within the PRS. The epimysium is not indicated in the present figure but lies anteriorly to the aponeurosis (4). The upper circle shows a magnified view of the different fascial layers contributing to the MLF. The picture shows that MLF is made up of three different structures: (1) This dashed line depicts the investing fascia of QL; (3) This dashed line represents the Paraspinal Retinacular Sheath also termed the deep lamina of the PLF encapsulating the paraspinal muscles; (2) The thick dark line between the two dashed lines 1 and 3, represents the aponeurosis of the abdominal muscles especially deriving from TrA. Numbers 1, 2 and 3 form the MLF. The lower circle shows a magnified view of the different fascial layers constituting the PLF. The picture shows that on the L3 level the PLF is also made up of three layers, since the fascia of SPI is normally present on this level. (5) This dashed line depicts the PRS or deep lamina of the PLF encapsulating the paraspinal muscles; (6) The investing fascia of SPI is seen blending medially into the grey line marked (7) and representing the aponeurosis of SPI- posteriorly to the PRS; (8) This dark line represents the investing fascia of LD blending medially into the black line representing the LD aponeurosis (9) posteriorly to the SPI aponeurosis. Numbers 5,7 and 9 form the PLF. Numbers 7 and 9 form the superficial lamina of the posterior layer (sPLF).

CTrA pull (simulation of Tr A /IAP) the posterior layer of the TLF moved medially and slightly less posteriorly.

This co-dependent mechanism between these structures relies upon pressure changes within the epiaxial TLF container and the hypaxial abdominal container and their balanced interplay. In fact, deep abdominal function, tensing the CTrA tendon is reciprocally linked to increased pressure of the TLF container during activation of the back muscles. Particularly this study shows that tension through the CTrA is mainly transferred through the posterior layer of the TLF (PLF) and not the middle layer (MLF). It is clinically apparent that when there is a functional mismatch between back and deep abdominal muscles, non-optimum control and altered fascial tension will ensue (Figs. 3 and 4).

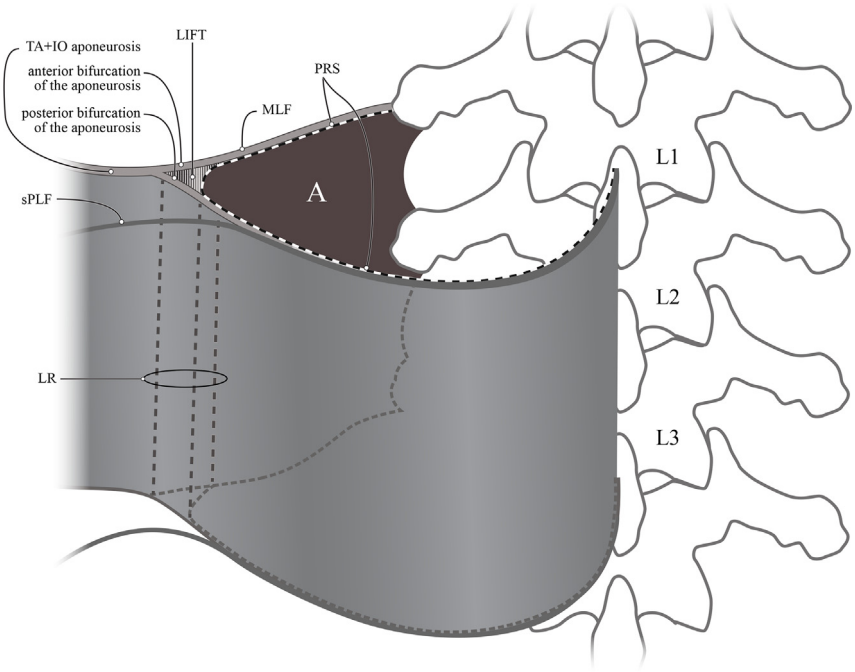

Figure 4 With permission from: Willard et al. (2012). A schematic and simplified view of the bifurcation of the TA and IO aponeurosis and the paraspinal retinacular sheath, creating the lumbar interfascial triangle (LIFT). A represents the empty space normally occupied by the paraspinal muscles and enclosed by the paraspinal retinacular sheath (PRS). The aponeurosis of the transversus abdominis (TA) and internal oblique (IO) bifurcates into anterior and posterior laminae. The anterior lamina contributes to the middle layer of the thoracolumbar fascia (MLF). The posterior lamina contributes to the deep lamina of the posterior layer of the thoracolumbar fascia (PLF). The lateral raphe (LR) represents a thickened complex of dense connective tissue at the lateral border of the PRS, from the iliac crest caudally to the 12th rib cranially. The junction of the transversus abdominis aponeuroses with the PRS creates the lumbar interfascial triangle (LIFT), which is at the core of the LR. Thus, the raphe is formed at the location where abdominal myofascial structures join the PRS surrounding the paraspinal muscles. *sPLF* superficial lamina of PLF.

Pelvic Girdle Pain (PGP)

i. The association between PGP and Pelvic Floor Muscle (PFM) function (Fitzgerald and Mallinson, 2013): There is an association between PGP and deep (levator ani and obturator internus) but not superficial PFM tenderness in pregnancy. However, there was no difference between the 2 groups (PGP and non PGP) in PFM voluntary contraction, involuntary contraction or relaxation — or in PFM strength Those with PGP were more likely to have a history of LBP or PGP
ii. The association between PGP and PFM function *(Stuge)*. There was no difference between PGP and controls in *voluntary* PFM function measured by palpation, manometry or ultrasound. Women with PGP had a statistically significant smaller levator hiatus and a tendency for higher vaginal resting pressure compared with controls. This may indicate increased PFM activity — They found no evidence to recommend PFM strengthening exercises for PGP

Active Straight Leg Raise test (ASLR)

i. The automatic PFM response to the ASLR in PGP (Stuge et al., 2013a,b) there was no difference in the *automatic* PFM response during ASLR in people with PGP and matched controls measured by trans-perineal ultrasound. A significant PFM contraction occurred during ASLR in both groups (this is counter to O'Sullivan's 2002 study which found PFM depression on ASLR). There was a strong positive correlation between voluntary and automatic PFM contractions. The PFM shortens more in ASLR than with voluntary contraction no doubt due to abdominal synergy and IAP generation. Reduction of levator hiatus or muscle length from rest to involuntary/automatic contraction was much the same in both groups. However, women with PGP had a significantly smaller levator hiatus at rest, during voluntary contraction and automatic contraction with ASLR than in controls. Significantly, manual compression of the pelvis *reduced* the automatic PFM contraction by 62—66%! This study further shows no evidence to recommend strengthening the PFM — rather, they need to relax! Overactive PFM will pull the sacrum into counter-nutation — a provocative position for the SIJ — and common clinically
ii. Muscle function and pelvic stability in the ASLR and gait (Hai Hua et al., 2013 — presented by van Dieën). In an EMG study of the ASLR, they found a significant main effect of side: dominant ipsilateral activity of Iliacus, adductor longus (not mentioned in the proceedings), rectus femoris transversus, internal oblique — and of the contralateral biceps femoris. Weighted ASLR increased the response and the asymmetry. Applying a pelvic belt *reduced* the activity

in transversus, both obliques, rectus abdominis and Iliacus — yet *increased* the activity in rectus femoris, gluteus maximus and biceps femoris. An earlier study showed that psoas is bilaterally involved in frontal plane stabilisation

These two ASLR studies have enormous clinical implications — overload the system and the patient is forced to further recruit globally dominant synergies. Using manual pressure or pelvic belts reduces the important contribution from deep system control. Failure of the deep abdominal muscles to contract, diminishes the necessary anterior pelvic compression and hence other muscles have to compensate to recreate sufficient anterior compression of the pelvis.

iii. The relationship between tissue sensitivity and the outcome of clinical tests in lumbopelvic pain (Palsson and Graven-Nielsen, 2013). In a healthy cohort, hypertonic saline was injected into the long posterior sacroiliac ligament with measurement before and after of tissue sensitivity; SIJ pain provocation tests and the ASLR. Significantly more SIJ provocation tests became positive 'during pain' which correlated significantly with lowered pressure pain threshold (PPT) values at S2 and the overall levels of pain. With the ASLR, all subjects demonstrated a significant *increase* in muscle activity as well as an increased feeling of 'difficulty' — both significantly correlated to the level of pain. The increased muscle activity involved a more global activation of the trunk muscles or 'bracing' compared to the more unilateral pattern seen in controls. A further study involved pregnant women with PGP grouped into low and high disability. All had significantly lowered PPTs in both the lumbo-pelvic area and extremities yet, only the high disability group rated ASLR more difficult.

iv. Sonographic characteristics of the abdominal muscles and bladder in individuals with lumbopelvic pain at rest and during ASLR (Whittaker et al., 2013) The lumbo-pelvic pain cohort (LPP) cohort had less total abdominal muscle thickness, a wider inter-recti distance and a significantly thinner rectus abdominis. During ASLR, the LPP group demonstrated smaller increases in transversus thickness and a greater inferior bladder base position during the task. The percent change in transversus had a fair correlation with pain intensity and duration and disability score

Some further insights into fascia

Excellent comprehensive review papers were given by Findlay, 2013 and Schleip, 2013.

i. Fascia is a tensegrity system on a macro and micro level. (Fonseca, 2013) To act as a tension network capable of distributing stresses the musculo-skeletal system needs to be *prestressed* (springs analogy). This allows for greater stability and responsiveness. Without prestress, the system cannot generate tension — and is likely to collapse when a force is applied. Muscles act in combination, hence considering the stabilisation actions of isolated muscles is unrealistic

ii. Mechanical loading of human tendon (Kjaer, 2013) results in increased protein synthesis in both muscle fibres and the connective tissues. Exercise stimulated increased collagen formation lasts for about 3 days and is associated with stimulation of collagen degradation. Connective tissues are less intensity dependent than muscle in stimulating protein synthesis — the same rise occurs between light and heavy exercise. In tendinopathy, signalling for collagen synthesis and degradation is upregulated. Only controlled loading (strength training) results in any manifest rise in collagen synthesis and tendon remodelling

iii. Sonoelastography (Dennenmoser et al., 2013) is actually able to reveal the physical qualities of fascial tissue — stiffness/hardness due to pathology and its consequent softening following 'release' with 3—5 min of manual therapy

iv. The anatomical and functional relation between gluteus maximus and fascia lata (Stecco et al., 2013) Opinions diverge as to the distal insertion of gluteus maximus. (GM). Dissection of 8 cadavers revealed: "GM presented a major insertion into the fascia lata, so large that the iliotibial tract could be considered a tendon of insertion of the GM". The iliotibial band is a reinforcement of the fascia lata and cannot be separated from it. Its inner side is in continuity with the lateral intermuscular septum which divides the quadriceps from the hamstring. The fascial insertion of GM could explain the transmission of forces from the TLF to the knee (Fig. 5).

Neuroplasticity and motor control in chronic lumbo-pelvic pain syndromes

The brain is certainly "in" at the moment! — neuroplasticity of the sensorimotor system. Current motor control research is extensive and exciting. Some tit bits:

I. There is evidence that spinal reflexes are delayed and reduced in subjects with chronic low back pain (Wagner et al., 2013)

II. Precision of trunk movement in LBP patients (Willigenburg et al., 2013 — presented by van Dieën). Difficulty sustaining an upright sitting posture without postural drift is associated with proprioceptive deficit. When the support is unstable the subject downplays proprioception and vestibular and visual control is more evident. These 'higher order' processes slow the system response. Proprioception is needed for trunk alignment. We need fast feedback for this. It is proprioceptive deficit more than motor impairment which impairs trunk control in LBP. These people move their backs less. Visual compensation is possible. Control loss is situation dependent due to sensory reweighting. Proprioception can't be retrained with balancing exercises on a ball! — but may be helped with sensory discrimination training and/or motor control retraining

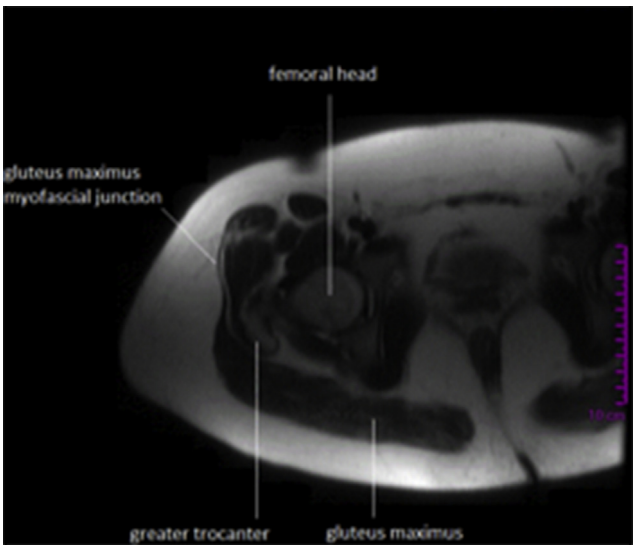

Figure 5 MRI T1 weight of the pelvic area, transverse section at the level of the femoral head. (A.Stecco).

III. Changes in the structural and functional properties of lumbar muscles in recurrent LBP (Danneels et al., 2013): There is a strong relationship between recurrent LBP and changed muscle function. Remission studies found structural change — the actual size is not reduced but there is decreased fibre density and increased fatty infiltration of multifidus, erector spinae and psoas. Experimental pain studies have shown decreased activity of the same muscles. During voluntary static/dynamic sagittal trunk movement in sitting, the LBP cohort showed increased multifidus activity. Rapid onset movements found higher co-contraction of the superficial muscles — a stiffening high load strategy for a low load task. Antagonistic extensor activity was higher in flexion, while agonistic flexor activity was lower. Similarly, EMG activity was higher for individual extensor muscles and lower for individual flexor muscles. Cortical reorganisation is apparent where representation of multifidus and ES shows a degree of overlap which may underpin loss of differential activation

IV. Motor control: a crucial factor for optimal function of the different structures; and Plasticity in the motor system and driving change with motor interventions (Hodges, 2013a,b) both big subjects — his research IS wide reaching. Salient points:
- Optimum control requires orchestrated function between the deep and superficial muscle systems to create the right balance between movement and stiffness. The contribution of the deep muscles is commonly compromised in PGP and LBP. Suboptimal movement changes the mechanical forces tissues are subjected to.
- Changed posture changes the way you breathe. 'Cavity' pressure changes affect fascial tension and have a direct relationship to lumbo-pelvic control, breathing and continence — which along with IAP must be matched to demand. Subjects with breathing problems and SUI demonstrate increased activity of EO in postural tasks. This is associated with compromised medio-lateral balance. Other Studies have found diaphragm fatigueability which may further compromise postural control. Interestingly, Hodges is now looking at glottal control and its relationship in controlling intrathoracic pressures — no doubt important in axial postural control.
- Neuroplasticity can underpin both negative and positive changes in sensorimotor aspects of lumbo-pelvic control. It opens the possibility for innovative new LBP and PGP treatments to change the nervous system and 'prime' or prepare it for change. Functional MRI and transcranial magnetic stimulation (TMS) have enabled researchers to learn more about the excitability of the motor cortex — particularly the excitability and organisation of the primary motor cortex. They have found larger motor evoked potentials for ES but smaller for Transversus — consistent with the changed co-ordination between these muscles seen clinically. This redistribution of activity within and between muscles varies from subtle to manifest changes.
- 'Cortical smudging' is predictive of certain typical gross motor behaviours. In LBP, transversus' cortical representation has been shown to shift posterolaterally. Also evident is a change in the paraspinal cortical representation from two to one. Recent evidence seems to show that the separate areas are involved in the activation of discrete muscles with separate areas for deep short muscles (multifidus) and longer more superficial muscles such as longissimus. He says… "it is tempting to speculate that the shift in cortical representation from multiple sites to a single site may relate to the propensity for many with low back pain to not express the differential activation of the deep and superficial muscles" (Proc.). Discrete control of movement is lost and replaced by loss of complexity of control and a more gross response. Increased trunk muscle co-contraction or 'splinting' may predispose to injury.
- Biopsychosocial influences will impinge on motor responses — amplification beyond necessary, inappropriate responses — and for longer than necessary.

"Why you get the pain may not be the same as why you continue to get it".

V. "Priming the brain with neuromodulatory techniques" (Schabrun, 2013) is a new concept to promote neuroplasticity whereby TMS, and/or peripheral electrical stimulation can be used to decrease or increase neural excitability and strengthen synaptic connections, making the brain more receptive to learning and change. Normally our brain excitability goes up when we 'learn'/train. This can be maintained for 4 weeks. 'Priming' attempts to artificially 'force the brain up' which may make it easier for patients to learn! Peripheral electrical stimulation induces

cortical reorganisation; reduces motor and sensory cortical hyper-excitability; targets mechanisms of central sensitisation and is a repeatable form of afferent input. Afferent input is one of the most powerful drivers of neuroplasticity. Brains learn better with repeated short applications. Watch out for 'brain subgrouping' — as our individual capacities vary!

VI. "Plasticity in the sensorimotor systems and implications for treatment" (Wand et al., 2013) This was an epic comprehensive review. There was so much information that I don't feel I can do him justice in a summary. CLBP is characterised by a range of structural, functional and neurochemical, changes in the brain. Understanding these better may help better treatment outcomes. I plan to do a separate review sometime as there was a lot of valuable information for the clinician.

Clinical subgrouping classification in CNSLBP

With seven papers around this subject the concept would appear to have traction — yet in many respects it still seems to be finding its way as there is little consensus about the composition and characteristics of subgroups — and what the intervention is tested against. Three main approaches are apparent (Hartvigsen, 2013): diagnostic subgroups (pathology, presenting symptoms and signs); treatment effect modifier subgroups (identifying groups of people likely to respond to particular treatments), and prognostic factor subgroups (identifying those at risk of chronicity etc). One paper was a review of the subject offering 'accessible guidance' to appraising subgroup research (Steven et al 2013 — presented by Koes). Another paper dealt with the clinical presentation of high risk patients identified by the STaRT back tool and results of treatment (Hill, 2013).

While CNSLBP is a complex condition, in the first instance it is a physical problem and I believe that until we get better at understanding and recognising 'what's wrong' with the 'bio' we can't hope to apply effective treatments. Hence a subgrouping system based on a true bio-psycho-social approach seems de riguer.

Four RCTs were presented on the outcomes resulting from a classification based treatment approach (Fersum, 2013; Ford et al., 2013; Lehtola et al., 2013; Van Dillen et al., 2013). All showed variably positive treatment effects during the intervention and at follow up. I believe that the most impressive presentation was from *Fersum* reporting on the results of the first RCT to test the effectiveness of a "Cognitive Functional Therapy" approach as proposed by O'Sullivan. Subjects had a mean number of 7.7 treatments over a 12 week period and achieved both highly impressive clinical and statistically significant change which was maintained at 3 year follow up! Why were the results so good? The classification system is based upon a comprehensive bio-psycho-social construct which has been carefully developed and tested in a stepwise progression. Well done Peter O'Sullivan et al.

Breathing — last but not least. There was only one paper on this subject!

"Inspiratory muscle training improves proprioceptive postural control in individuals with recurrent nonspecific low back pain "(Janssens et al., 2013) was one of the many gems in the 10 min parallel sessions.

NSCLBP subjects show a suboptimal, more ankle steered proprioceptive postural control (PPC) strategy ('healthy' control is multisegmental — particularly the pelvis) NSCLBP is also strongly related to respiratory disorders and PPC seems to be impaired in individuals with compromised respiratory function. Loading of the inspiratory muscles impairs postural control by decreasing lumbar proprioceptive sensitivity. Individuals with LBP are known to have a greater diaphragm fatigability compared with healthy controls.

The intervention consisted of breathing through an inspiratory resistance of 60% of their maximal inspiratory pressure (controls breathed at 10% mip) — 30 times, twice daily for 8 weeks. Compared with the control group, the inspiratory muscle training group demonstrated a more multi-segmental postural control strategy, showed an increase in inspiratory muscle strength and reported a decrease of LBP severity. Addressing the trunk stabilisation and support role of the diaphragm is important!

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