

The fall of the postural-structural-biomechanical model in manual and physical therapies: Exemplified by lower back pain

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KEYWORDS

Biomechanics, lower back pain, pathomechanics, posture, spine, structural asymmetries.

SUMMARY Manual and physical therapists often use a postural-structural-biomechanical (PSB) model to ascertain the causes of various musculoskeletal conditions. It is believed that postural deviations, body asymmetries and pathomechanics are the predisposing/maintaining factors for many musculoskeletal conditions. The PSB model also plays an important role in clinical assessment and management, including the choice of manual techniques and the exercise prescribed. However, this model has been eroded by research in the last two decades introducing profound challenges to the practice of manual and physical therapy. This article will examine how the sciences are challenging the PSB model, using lower back pain (LBP) as an example.

Introduction

There is a basic belief among manual and physical therapists that structural imbalances and asymmetry in the body can result in painful musculoskeletal conditions. In this model, the imbalances and asymmetries increase the abnormal mechanical/physical stresses imposed on the musculoskeletal system. This may lead to recurrent injury or the development of chronic conditions through a gradual process of wear-and-tear. This conceptual model manifests clinically in the form of postural, structural and biomechanical (PSB) assessments, manual treatments and exercise that aim to correct these structural factors. The PSB model is frequently used in clinic to manage patients suffering from low back pain (LBP).

The PSB evaluation/assessment often includes a static postural examination, observing the shape of the back, whether there are any increases in spinal curves such as scoliosis, kyphosis or lordosis. The assessment may also include measuring pelvic angles in the coronal plane, pelvic nutation/counternutation angles, the relative position of the sacrum to the ilia and leg length differences. It is believed that such misalignments impose excessive stress on the spine leading to degeneration/damage or dysfunction and

eventually to painful back conditions. These static observations are often followed by a dynamic assessment during standing in which the spine is examined in all planes of motion. Observed regional and segmental movement losses/stiffness is often used to determine the severity of the spinal condition and is also used to explain the cause of the condition. The PSB examination may sometimes include an assessment of the feet. The rationale here is that any problems in the physical foundations upon which the body rests will have repercussions on structures further up the mechanical chain, such as the knees or the lower back.

A palpatory examination is often incorporated into the PSB assessment during standing or lying on the treatment table. Information is gathered about abnormal tissue textures, unusual muscle stiffness or abnormal relationships between body masses, muscle bulks or position of vertebral landmarks. Although these findings are often used to estimate the location of damage or tissue-causing symptoms they are also used to identify predisposing local segmental PSB factors.

The structural model also includes beliefs about imbalances and misalignments in specific body subsystems or tissues as the causes of spinal and other pain conditions. These include adverse neural ten-

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sion, specific muscles such as multifidus or shortened hamstrings, muscle chains, kinematic chains and the fascia system.

Every few years this model shifts in focus to other body systems. In the last decade, this biomechanical model has infiltrated the neuromuscular dimension. Mechanical and computer code ideals are applied to motor control to explain musculoskeletal conditions. These include looking at minute timing changes between muscle groups, an emphasis on singling out particular muscles for rehabilitation or the identification of “weak muscle” or muscle imbalances. Core stability and spinal stabilization approaches are examples of this neuro-mechanistic model.

This PSB belief system also permeates other forms of manual therapy. In visceral osteopathy it manifests as a focus on the movement of the organs and their anatomical-mechanical relationship. In cranial approaches it appears as a focus on the position and movement of cranial structures including the articulations between cranial bones and tension in dural membranes. In many manual therapy disciplines there is a belief that spinal misalignments (subluxations) can cause visceral conditions as well as other health issues beyond the spine (Mirtz et al., 2009). In these disciplines, PSB factors are used to make links between skeletal and non-skeletal body

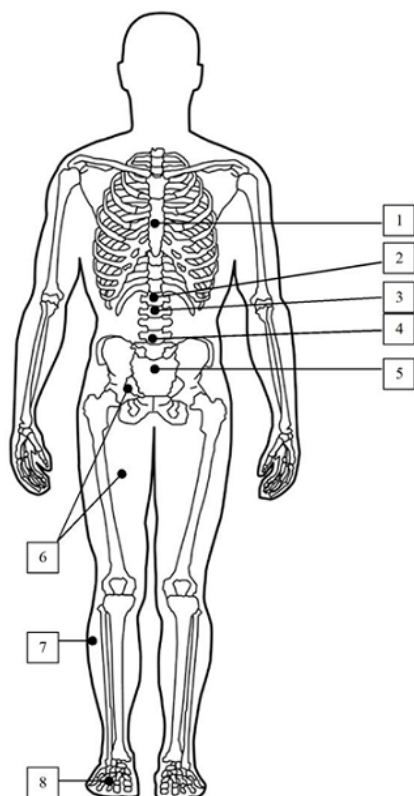
systems to explain the predisposing and maintaining factors for the condition.

The outcome of these PSB examinations is an appreciation of the individual’s PSB status. This information is then used to explain why the patient is suffering from back pain. It also forms the rationale for the treatment which may aim to mechanically/physically correct/change the observed misalignments or improve range. This is achieved through the use of various manual therapy procedures (e.g., manipulation, muscle energy techniques, stretching and articulation/mobilization) or specific exercises (e.g., McKenzie back exercise, core stability, Yoga, Pilates). The basic premise is that an existing condition will improve and future recurrences or chronicity can be prevented by correcting these PSB predisposing/maintaining factors.

However, the most important question is consistently being ignored – can a person’s physical shape/posture/structure/biomechanics be the cause of their lower back pain?

Is development of LBP associated with PSB factors?

In the last two decades the PSB model has been eroded by clinical studies examining the relationship between PSB factors and lower back pain (Fig. 1).



1. Trunk asymmetry, thoracic kyphosis and lumbar lordosis in teenagers and developing LBP in adulthood.
2. Low muscle strength, low muscle endurance, or reduced spinal mobility and erector spinea pairs imbalances during extension.
3. Variations in lumbar lordosis and thoracic kyphosis. Increased lumbar lordosis and sagittal pelvic tilt on back pain during pregnancy. Differences in regional lumbar spine angles or range of motion.
4. Disc degeneration, spina bifida, transitional lumbar vertebra, spondylolysis and spondylolisthesis.
5. Pelvic obliquity and the lateral sacral base angle pelvic asymmetry.
6. Hamstrings and psoas tightness.
7. Inflexibility of the lower extremities or leg length discrepancy.
8. Correcting foot mechanics have no effect on preventing back pain.

Figure 1 – Many postural–structural–biomechanical (PSB) factors have failed to show an association or to be the cause of lower back pain.

Prospective studies are particularly useful to examine the causal relationship between PSB factors and LBP. In these studies groups of asymptomatic individuals are assessed for PSB factors initially and tracked over several years noting the episodes of LBP. Other less ideal studies compare subjects with LBP to an asymptomatic group. However these studies can only be used to inform us about the changes that are due to the condition but they cannot indicate its cause, that is, the consequence of LBP is not necessarily its cause. This distinction is important clinically. Often the PSB assessment is made when the patient is already in pain, once the individual/body has reorganised to cope with the condition.

Spinal curves, asymmetry and motion

There was the lack of association between postural spinal asymmetry, thoracic kyphosis and lumbar lordosis in teenagers and developing LBP in adulthood (Papaioannou et al., 1982; Dieck, 1985; Poussa, 2005). Even obvious increases in lordosis and sagittal pelvic tilt during pregnancy lack an association with back pain (Franklin & Conner-Kerr, 1998). Stronger predictors of the development of back pain during pregnancy were body mass index, history of hypermobility and amenorrhea, low socioeconomic class, previous LBP, posterior fundal location of placenta and fetal weight to LBP with radiation to leg (Orvieto et al., 1990; Mogren & Pohjanen, 2005).

In adults, the extent of lumbar lordosis as well as the presence of scoliosis failed to show an association with back pain (Dieck, 1985; Norton, 2004; Haefeli et al., 2006; Christensen & Hartvigsen, 2008, syst. rev.). Also differences in regional lumbar spine angles or range of motion between the segments failed to show an association with the future development of LBP (Hellsing, 1988b; Burton & Tillotson, 1989; Hamberg-van Reenen, 2007, syst. rev.; Mitchell et al., 2008).

Segmental pathomechanics

One important area to examine is whether the profound biomechanical changes brought about by segmental pathologies can give rise to lower back symptoms.

A systematic review from 1997 suggests an association between disc degeneration and nonspecific low back pain (van Tulder et al., 1997, syst. rev.). However, it might not be the cause of it—there is strong evidence that X-ray and MRI findings have no predictive value for future LBP or disability (Waddell & Burton, 2001, review). Several studies since have failed to show a clear relationship between spinal/

disc degeneration and LBP (Savage et al., 1997; Borenstein et al., 2001; Carragee et al., 2005; Jarvik et al., 2005; Kanayama et al., 2009; Kalichman et al., 2010). In a population-based study of 34,902 Danish twins 20-71 years of age, there were no meaningful differences in the frequency in LBP between younger and older individuals (Leboeuf-Yde et al., 2009), although greater degenerative changes are expected in older individuals.

In studies that show some relationship between disc degeneration and LBP it has been suggested that the genes that play a part in the heritability of back pain also play a part in disc degeneration, that is, pain may not be due to the mechanical changes in the spine but to shared biological factors (Battie et al., 2007). These hereditary factors are not associated with the shape of the back but linked to variations in the collagen and immune-repair system/processes between individuals (Paassilta et al., 2001; Valdes et al., 2005; Battie et al., 2009; Videman, 2009a). It was demonstrated in twins that as much as 47%–66% of spinal degeneration is due to hereditary and shared environmental factors, whereas only 2%–10% of the degeneration can be explained by physical stresses imposed by strenuous occupations or sporting activities (Battie, 1995; Battie et al., 2009; Videman et al., 2006, 2007).

No association has been found between congenital abnormalities in the lumbar spine and pain in that area (spina bifida, transitional lumbar vertebra, spondylolysis and spondylolisthesis; van Tulder et al., 1997, syst. rev.; Luoma et al., 2004; Brooks et al., 2009). Although spina bifida and transitional vertebra may not be the cause of LBP, they may determine the pain levels (Taskaynatan et al., 2005, weaker study).

Another popular and enduring biomechanical concept is the spinal “neutral zone”. It claims to be related to stability and LBP (Panjabi, 1992a,b, 2003; Suni et al., 2006). This mechanical concept is derived from mathematical models and cadaver experiments on which an extensive amount of spinal joint damage had to be inflicted before the findings could fit the model (Gracovetsky, 2005). Since its inception three decades ago, no study exists to show a correlation between mechanical changes in the neutral zone changes and LBP (Leone et al., 2007, review).

The disparity between pathomechanics and symptomatology can be observed in other segmental conditions. For example, in a MRI study of patients with nerve root pain it was found that the degree of disc displacement, nerve root enhancement or nerve compression did not correlate with the magnitude of the patients’ subjective pain or level of functional disabil-

ity (Karppinen et al., 2001; see also Beattie et al., 2000). However there is a strong association between severe nerve compression, disc extrusion and distal leg pain (Beattie et al., 2000).

Non-spinal structures

Studies have also failed to identify an association between other structures beyond the spine and back pain. For example, there is no correlation between pelvic obliquity/asymmetry and the lateral sacral base angle and lower back pain (Dieck, 1985; Levangie, 1999a,b; Fann, 2002; Knutson, 2002).

Leg length differences as a cause for back pain has been debated for the last three decades. It is estimated that about 90% of the population has a leg length inequality with a mean of 5.2 mm. The evidence suggests that for most people anatomic leg-length inequality is not clinically significant (Papaioannou et al., 1982; Grundy & Roberts, 1984; Dieck, 1985; Fann, 2002; Knutson, 2005, review), until the magnitude reaches approximately 20 mm (Gurney, 2002, review; Knutson, 2005, review). Although some earlier studies comparing people experiencing back pain with asymptomatic controls suggest a correlation (Giles & Taylor, 1981; Friberg, 1983, 1992), more relevant are prospective studies in which no correlation was found between leg length inequality and LBP (Hellsing, 1988a; Soukka et al., 1991; Nadler, 1998).

Patients who have acquired their leg length differences later in life as consequence of disease or surgery may also help to shed light on the relationship between pathomechanics and LBP. Individuals who developed a shorter leg due to Perthe's disease had a poor correlation between leg-length inequality, lumbar scoliosis and low-back disorders, assessed several decades after the onset of the condition (Yrjönen et al., 1992). In studies of patients who had marked changes in leg length due to hip fractures or replacement, such changes were not associated with back pain assessed several years after surgery (Gibson et al., 1983; Edeen et al., 1995; Parvizi et al., 2003).

One of the arguments in favour of an association between leg length differences and LBP is the supposed success of heel lifts in reducing back pain (Giles & Taylor, 1981; Gofton, 1985; Helliwell, 1985; Friberg, 1983, 1992; Brady et al., 2003, review). However, all these studies failed to include controls or sham heel lift (such as inefficient soft foam lift).

Prospective studies of inflexibility of the lower extremities and hamstrings and psoas tightness also fail to predict future episodes of LBP (Hellsing, 1988c; Nadler, 1998).

As for foot biomechanics there is strong evidence that orthotic corrections have no effect on preventing back pain (Sahar et al., 2007, syst. rev.).

Surprisingly even whole body changes such as overweight/obesity have a low association with LBP (Leboeuf-Yde, 2000, syst. rev.). Contrary to common beliefs, a recent study has shown that cumulative or repetitive loading due to higher body mass (nearly 30 pounds on average) was not harmful to the discs. The study found a slight delay in disc desiccation (L1-L4) in the heavier men when compared with their lighter twin brothers (Videman, 2009b).

Neuromuscular factors

Although not fully within the scope of this article, motor control of the trunk is relevant in relation to muscle function and posture. Certain neuromuscular components also failed to show a clear association with LBP.

Although earlier studies demonstrated an association between muscle endurance and LBP (Biering-Sørensen, 1984; Alaranta et al., 1995), a recent systematic review found strong evidence that low trunk muscle endurance is not associated with LBP (Hamberg-van Reenen, 2007, syst. rev.). This review found inconclusive evidence for an association between low trunk muscle strength and LBP. Also there is no association between erector spinae pairs imbalances during extension and LBP (Reeves et al., 2006; Hamberg-van Reenen, 2007, syst. rev.; Van Nieuwenhuyse et al., 2009). Furthermore, no study to date has shown that back pain is due to timing differences in specific muscle such as transversus abdominis (Lederman, 2010b, see Discussion). These control changes have been observed only in individuals who already have back pain. They probably represent the outcome rather than the cause of back pain (Lederman, 2010a, see Discussion).

Two studies using the same methodology appear to demonstrate that in athletes a delayed reflex muscle response at the trunk could increase the risk of lower back as well as knee injury (Cholewicki et al., 2005; Zazulak et al., 2007). Unfortunately, the obvious was not examined in these studies—the reflex response to a sudden perturbation of the trunk should have been examined in other body areas (e.g., a control recording from the leg). This would have helped establish whether the injuries are due to delayed muscle onset-timing, specific to the trunk, or the alternative more plausible explanation that athletes with sluggish muscle reaction times/reflexes may be more susceptible to injury.

Postural behaviour factors

An area that is often assessed in manual and physical therapy is how “correctly” a person is using their body—their “postural behaviour”. It is believed that prolonged postural stresses at work or sporting activities could be the cause of LBP. The results of recent systematic reviews challenge these widely held beliefs. These studies demonstrate lack of association between work-related posture and LBP. They include postures such as prolonged standing, bending, twisting, awkward postures (kneeling or squatting) sitting posture at work and prolonged sitting at work and leisure time (Hartvigsen et al., 2000, syst. rev.; Bakker et al., 2009, syst. rev.; Chen et al., 2009, syst. rev.; Roffey et al., 2010, syst. rev.; Wai et al., 2010, syst. rev.). Also physical leisure time activities such as sport or exercises, sitting and prolonged standing/walking were found not to be associated with LBP (Bakker et al., 2009, syst. rev.). Heavy manual lifting is strongly associated with LBP, however the effect size is considered to be modest (Waddell & Burton, 2001, review).

Prediction of back pain by PSB assessment

In a recent prospective study on young workers ($n = 692$) examined by physical therapists, PSB factors failed to show a correlation with future development of LBP (Van Nieuwenhuysse et al., 2009). A number of factors were evaluated including iliac crest height inequality, scoliosis, lumbar flexion, extension and lateral flexion, length of hamstring muscles and strength testing in the motor distribution of L4/L5/S1.

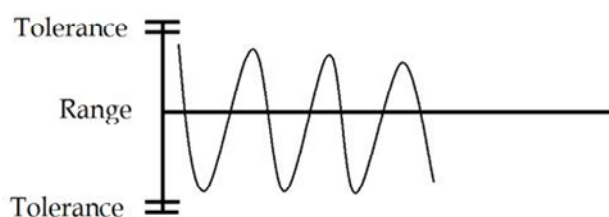
Summary points:

- Postural and structural asymmetries cannot predict back pain and are unlikely to be its cause.
- Local and global changes in spinal biomechanics are not demonstrably the cause of back pain.
- A PSB model is not suitable for understanding the causes of back pain.

Biological not mechanical dimension

There seems to be a disparity between pathomechanics of the body and the experience of a low back condition. Why does the body, which in essence appears so mechanical (joints, levers), behave symptomatically in such a non-mechanical and perhaps unexpected way?

This paradox arises from the way in which people are educated to perceive the musculoskeletal system—primarily as a mechanical entity and only minimally as a biological entity. In this biomechanical model the musculoskeletal system is seen as a precision engine where every system, organ and cell works in perfect harmony within itself and other body systems. All joints and body masses are in some anatomically ideal relationship to each other. Muscles are in anatomical–physiological–functional balance with motoneurons firing synchronously in perfect harmony. Injury, damage, “disease” or the experience of a back “condition” are seen as the consequence of some disturbance in this harmonious relationship. However, this sequence of events is not evident in the body/spine. Unlike mechanical systems the causation and experience of a spinal condition seem to be unrelated to PSB factors. They seem to largely reside within the biological dimension and



A. Mechanical systems.

Figure 2—Differences between mechanical and biological systems in regards to biological reserve and tolerance.

A. Mechanical system often has a set range with little tolerance before failure. **B.** Biological systems and processes often have a wide physiological range, less defined end range and a large potential adaptive range. **C.** In mechanical systems events that exceed the system’s range and tolerance often result in damage and progressive failure. In biological systems overloading can result in two possibilities: **D.** Acute overloading will result in damage and eventual repair, or permanent damage but without loss of function or symptomatology. **E.** Chronic overloading often results in adaptation and expansion of the physiological range.

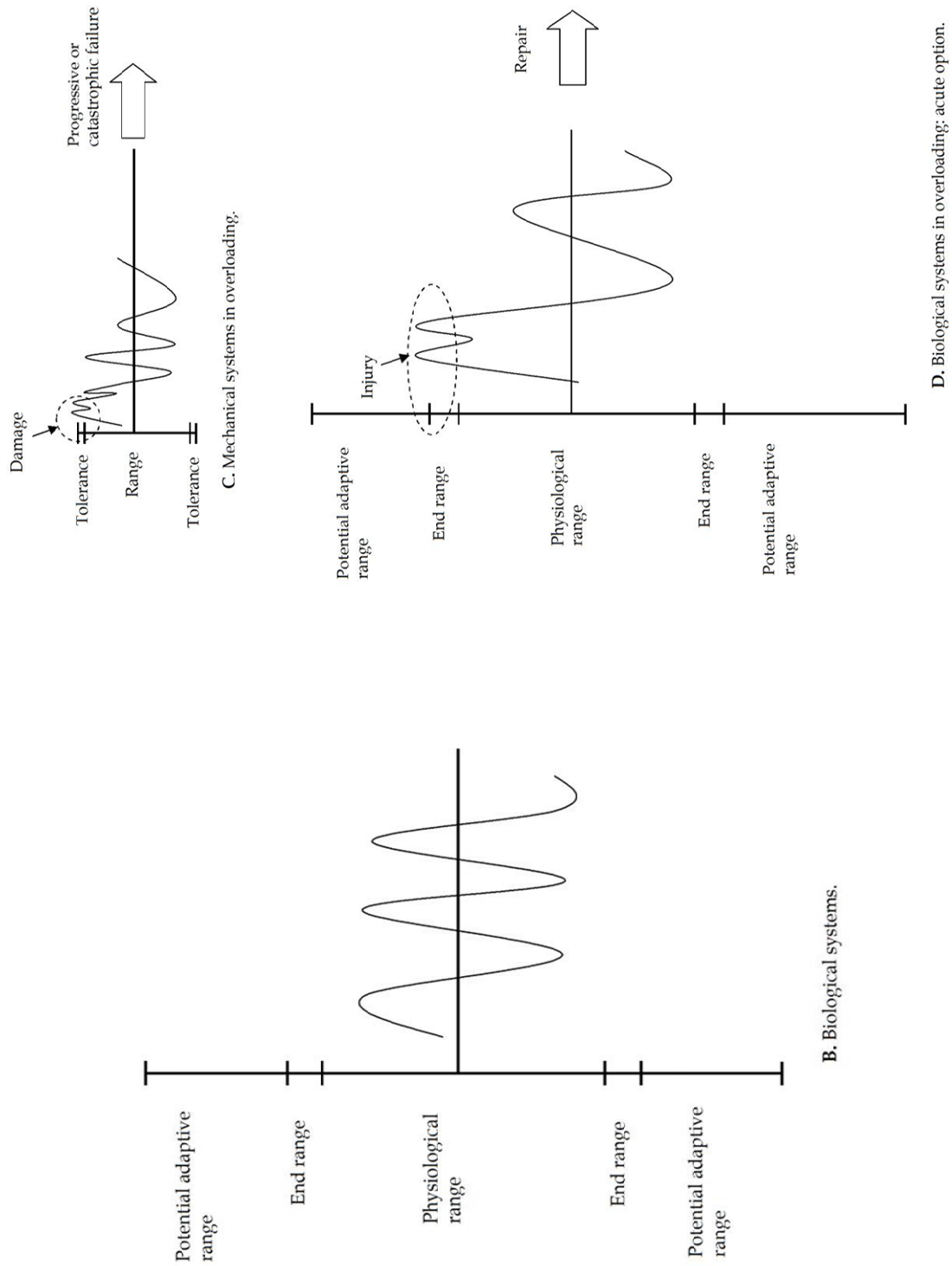


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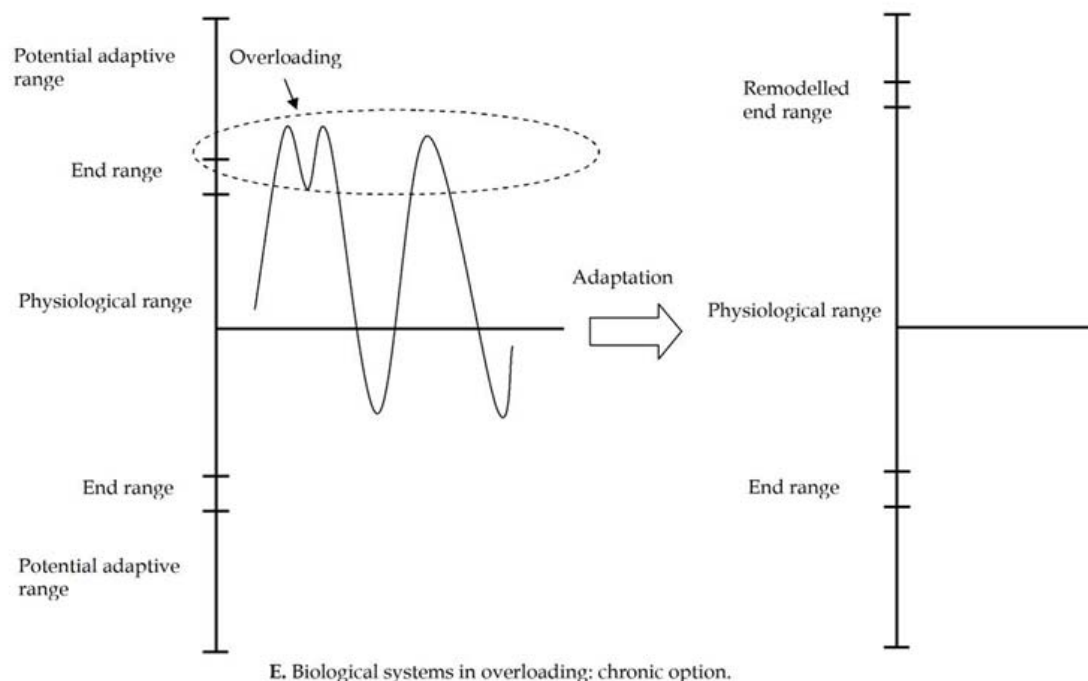


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hence, the disparity between PSB factors such as disc degeneration and LBP.

Within a biological dimension the structure (spine) is capable of self-repair and is able to adapt and change according to needs and demands (Fig. 2). But crucially, being a human with a highly evolved nervous system means that the structure is within the awareness. It is also under the influence of our emotions as well as the will and the actions taken. Therefore a person's cognitions and behaviour will have important implications to their recovery from LBP (Lederman, 2010a, see Discussion). Humans are also capable of experiencing pain and suffering—something a washing machine cannot do (yet). This has led to the emergence of a biopsychosocial model for LBP replacing the traditional PSB model.

Biological reserves and tolerance

The mechanical view of the body contains also anatomical and functional ideals—a form of utopian view of the body. The utopian view gives rise to the expectation that, like machines or computers, the body has to work in perfect precision/synchrony. The question that arises is does it really matter if these PSB factors or minor control changes exist and would they cause some catastrophic failure in the musculoskeletal system? It was apparent from the

studies discussed above that the spine can undergo profound physical changes that are well tolerated without the development of a symptomatic condition. What is observed here is that biological systems contain reserve capacity to accommodate for loss without failure/symptoms.

So does it matter, for example, that patients with CLBP may have localised wasting of the multifidus at L4–L5 (Hides et al., 2008)? Probably not, during standing and walking the trunk muscles are minimally activated (Andersson, 1996). In standing the deep spinal erectors, psoas and quadratus lumborum are virtually silent. In some subjects there is no detectable EMG activity in these muscles. During walking rectus abdominis has an average activity of 2% maximal voluntary contraction (MVC) and external oblique 5% MVC (White & McNair, 2002). During standing “active” stabilisation is achieved by very low levels of co-contraction of trunk flexors and extensors, estimated at < 1% MVC rising up to 3% MVC when a 32-kg weight is added to the torso. A back injury is estimated to raise these values by only 2.5% MVC for the unloaded and loaded models (Cholewicki et al., 1997). During bending and lifting a weight of about 15 kg co-contraction increases by only 1.5% MVC (van Dieen et al., 2003). This means that an individual will have to lose substantial mus-

cle mass and force production ability before such daily activities will be adversely affected. However, the biological reserve allows for such losses without a negative effect on spinal function or the development of a condition. Indeed, men tend to naturally lose 25% of their muscle mass between the ages 50 and 75 years without any detrimental effect. It is estimated that when the loss of this muscle reserve reaches 30% it will limit normal function in an older individual (Marcell, 2003).

Biological reserve is likely to explain why foot mechanics, leg length differences, pelvic torsion timing delays or any other PSB factors do not result in a symptomatic spinal condition. The system is capable of tolerating and compensating for these factors within the available surplus.

This reserve capacity can be seen elsewhere in the body. For example, partial or full thickness rotator cuff tears are found in a third of asymptomatic individuals over the ages of forty (Sher et al., 1995). These structural losses are not associated with pain or loss of shoulder function. In an evaluation of 100 asymptomatic volunteers (19–88 years) acromioclavicular joint osteoarthritis were present in three-fourths of the shoulders, one-third had subacromial spurs. Also found were changes in the peribursal fat plane and the presence of fluid in the subacromial–subdeltoid bursa. Joint fluid was observed in nearly all subjects (Needell et al., 1996). Furthermore, pathomechanics of the gleno-humeral joint was found to be similar in both symptomatic and asymptomatic individuals (Yamaguchi et al., 2000). We can assume that these pathomechanical changes would also be associated with profound neuromuscular changes, yet without giving rise to symptoms or functional loss. It is intriguing why some individuals develop a symptomatic condition while others remain asymptomatic.

Conflict within the PSB model

There is also a logical conflict within a PSB model. If persistent PSB factors lead to injury/damage/pain no one would ever recover from a simple back pain condition, whether acute, recurrent or chronic. Under a PSB model they would be expected to progressively get worse to the point of total disability; in the same way that damaged/unsynchronized machines gradually fail. Within a mechanical model, for example, the back condition of a person with leg length inequality would be expected to deteriorate over time. However, this does not seem to be a common occurrence. The symptomatology of LBP is variable and individuals may experience extended pain-free periods

without progressive worsening or increase in the frequency of their condition (Streiner, 2001; Carragee et al., 2006; Hartman, 2009). This logical conflict applies to all PSB factors described so far including motor control, proprioceptive and muscular changes (See Discussion in Lederman, 2010a).

The utopian view of the body raises several more questions. Is there ever a perfect PSB balance and does it matter? Do individuals develop/experience a condition when this balance is affected? Should we try to fix everybody even if they are not symptomatic? Where do we start, how do we decide which imbalance/asymmetry is more important?

Concession to the PSB model

Perhaps there is a critical level where PSB factors will exceed the reserve of the system. This can be either from gross PSB asymmetry/imbalance or in extreme physical demands. For example, there may be an association between severe scoliosis and back pain or substantial nerve root compression and leg pain (Beattie et al., 2000; Haefeli et al., 2006). In sports, extreme physical demands combined with underlying PSB factors may increase the likelihood for LBP (Ogon et al., 2001; Iwamoto et al., 2004). However this leaves us with the question of what to do with these findings in sports: correct the PSB factors or introduce better management of training and game schedule?

There is a catch with this concession in regards to clinical management. If the asymmetry/imbalance is severe it is unlikely that manual therapy or even exercise can substantially modify it (see below). On the other hand if the asymmetry/imbalance is minor or moderate it is unlikely to contribute to the patient's LB condition.

Summary points:

- The experience of a condition or disease is organised within the biological–psychological dimensions of the individual. The contribution of biomechanical factors is not clear.
- Body systems seem to have reserve capacity to allow for asymmetry and imperfections to exist without failure or symptoms.

The three clinical hurdles

The PSB model introduces unnecessary complexity and hurdles to practice. The first hurdle to overcome in the PSB model is the inability to identify/define the critical level where PSB factors contribute to the individual's back pain. This critical level is impossible to predict on an individual basis.

If we were to overlook this obstacle, the next hurdle to overcome is the reliability of assessing PSB factors. It is now well established that many of the examinations that assess PSB factors are either low on validity or reliability, in particular, the more precise/minute examinations such as leg length differences, tissue textures, pelvic angles and individual vertebral positions (McCaw & Bates, 1991; Mannello, 1992; Panzer, 1992; Levangie, 1999a,b; Hestbaek & Leboeuf-Yde, 2000, syst. rev.; Dunk et al., 2004; Seffinger et al., 2004; van Trijffel et al., 2005; Hollerwöger, 2006; May et al., 2006; Paulet & Fryer 2009).

Even if we were to overlook the two former hurdles, there is yet a third one to overcome—are manual techniques or specific exercise effective in modifying inherent PSB factors? Can foot mechanics, leg length differences, pelvic tilts, vertebral positions and spinal curves be permanently changed, solely, by these clinical tools?

Permanent adaptive musculoskeletal changes require physical overloading well above the person's default daily use (See Discussion in Lederman, 2005). Such an adaptation depends on the length and frequency of exposure to overloading. For example, strength training requires overloading by progressive increase in resistance and duration/frequency; an improvement in running endurance is achieved by running further and often, and so on (Henriksson & Hickner, 1996). Conversely, a cessation of exercise will result in rapid reversal of these training gains. In the context of PSB factors, it is expected that tremendous forces, well above the daily physical stresses, would be required to reposition/adjust/correct any structural misalignments. These would have to be applied on a daily basis over several months or even years. A termination of treatment is likely to result in rapid reversal of PSB gains, unless the individual is able to self-maintain them by specific exercise. The winner in this competition-in-adaptation is ultimately the one most practised, that is, the default PSB state/behaviour of the individual (See Discussion in Lederman, 2005, 2010a).

There are no known studies that examine the influence of manual techniques on PSB factors in the medium- or long-term, in particular at the cessation of the treatment. In essence, tensional forces (e.g., stretching) are required in order to induce adaptive connective tissue or muscle length changes. These can be applied within different time scales, as a sudden tensional force such as in spinal manipulation or forces applied from several seconds to minutes, such as in manual stretching or exercise. Sudden application pulse of tension as in manipulation is only likely

to produce transient tissue lengthening (creep deformation), lasting no more than a few minutes (Light et al., 1984; Roberts & Wilson, 1999). Manual stretching of muscles or exercise for several minutes will have a transient lengthening effect lasting up to an hour (Magnusson, 1998; Magnusson et al., 1995). Longer-term stretching over several weeks will activate and maintain specialized cellular processes in muscle and connective tissues that account for permanent tissue elongation (Williams et al., 1986; Goldspink et al., 1992; Arnoczky et al., 2002; Bosch et al., 2002). However, these tissue lengthening processes tend to revert to the default-use at the cessation of treatments (Harvey et al., 2002). For example, a break of four weeks completely abolishes the gains of six weeks of stretching (Willy et al., 2001).

Orthodontic braces to correct the bite are an example of the enormity of the task required to produce permanent PSB changes. A teenager is expected to wear the fixed braces for several years. It is followed by wearing a night brace for several more years to prevent the adaptation to revert back to the default. Similarly, spinal curves are determined by the shape of the vertebra and discs as well as every other tissue connected to them (Lonstein, 1999; Marks & Qaimkhani, 2009). Therefore, a spinal brace worn daily for many years slightly straightens scoliosis, but the curves tend to gradually regress when the brace is removed (Maruyama, 2008, syst. rev.; Maruyama et al., 2008).

It would require a herculean effort to modify many of the inherent PSB factors discussed so far. As such, the therapeutic investment in correcting PSB factors is irrational, in particular, as it is unlikely to influence the course of the patient's condition.

Summary points:

- A PSB model introduces unnecessary complexity at a conceptual level and in clinical assessment.
- Observational or physical assessments of PSB factors have no value in elucidating the causes for back pain.
- Clinical assessment of PSB factors assessed by manual and visual means may be unreliable.
- Such assessments are likely to be redundant and can be safely removed from clinical practice. This excludes assessment that aim to identify serious pathologies.
- PSB factors are unlikely to change in the long-term by manual techniques or even exercise, unless rigorously maintained (exercise).
- A PSB model may introduce an element of therapeutic failure as the aims and goals of this approach may not be attainable by manual therapy or even exercise.

Implications for practice

The lack of association between PSB factors and back pain has far-reaching implications for the way we conceptualize musculoskeletal conditions, the clinical examination and the goals/objectives of the techniques and the exercise prescribed.

From the evidence so far many of the clinical examinations assessing PSB factors have no obvious values in explaining why the patient has developed their back condition. It implies that the PSB model and the related clinical examinations are mostly redundant. Furthermore, there is compelling evidence that the PSB model may take us further away from understanding back pain. It has been consistently demonstrated that lower back pain recurrences, chronicity or disability can be better predicted from assessing biological, psychological and social factors (Carragee et al., 2006). For example, about 45%–55% of LBP conditions are attributed to hereditary factors (Battié, 1995; Paassilta et al., 2001; MacGregor et al., 2004; Valdes et al., 2005; Videman et al., 2006, 2009a,b; Battié et al., 2007, 2009). Several studies have shown that as much as 80% of serious LBP events and 93% of LBP disability events can be better predicted by biopsychosocial factors such as gender, abnormal psychometric testing, smoking and compensation issues (Carragee et al., 2006). In contrast, it is difficult to find any studies that identify predisposing structural factors for LBP, despite several decades of research into this condition (Bakker et al., 2009, syst. rev.).

The lack of association between PSB factors and LBP has also important implications for what we aim to achieve and for our choice of techniques and exercise used to manage the condition. We can no longer justify the use of manual techniques to readjust, correct or balance-out the misaligned structure. There is an urgent need to redefine what the therapeutic goals are, beyond relieving the patient's symptoms, for example, is there any value in providing long-term maintenance/preventative treatments for asymptomatic individuals?

In a prospective study using MRI scans it was demonstrated that recurrences of back pain over a period of five years were not associated with any progressive spinal damage (Carragee et al., 2006). Individuals were experiencing periods of pain and symptomatic recovery although their spinal condition remained unchanged. This is a regular phenomenon where a condition will exhibit natural variation around a certain symptomatic mean. At certain times the individual will experience periods of symptomatic quiescence (Streiner, 2001; Hartman, 2009; see

also Kongsted & Leboeuf-Yde, 2010). This implies that the therapeutic ideal of a “cure” may not be possible, as the underlying condition could still be present but is asymptomatic. Perhaps research and treatment should be directed towards finding better approaches to provide symptomatic relief during periods of pain as well as increasing the patient's participation in social, occupational and recreational activities (Waddell et al., 2008; Kendall et al., 2009). This attitude may be more realistic than the idealized clinical aspiration to provide a permanent cure by correcting PSB factors.

Finally and more complex is the therapists' education in the various manual and physical therapies where the PSB model is dominant. If this model is flawed what is the alternative clinical model and who is capable of teaching it?

The alternative: a Process Approach

A clinical alternative to the PSB model is a Process Approach model. The aim in this approach is to identify the processes underlying the patient's condition and provide the stimulation/signals/management/care that will support/assist/facilitate change. This approach has been extensively discussed in Lederman (2005) and will be discussed in a future article.

Summary and conclusion points

- PSB asymmetries and imperfections are normal variations – not a pathology.
- Neuromuscular and motor control variations are also normal.
- The body has surplus capacity to tolerate such variation without loss to normal function or development of symptomatic conditions.
- Pathomechanics do not determine symptomatology.
- There is no relationship between the pre-existing PSB factors and back pain.
- Correcting all PSB factors is not clinically attainable and is unlikely to change the future course of a lower back condition.
- This conclusion may well apply to many common musculoskeletal conditions elsewhere in the body (e.g., neck pain).

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