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**Is pain the cause of altered biomechanical functions
in back pain sufferers?**

Jonathan M. Williams, MManipTher, #

Inam Haq, MD, MBBS,

Raymond Y. Lee, PhD,

From the School of Human and Life Sciences, Roehampton University, Whitelands

College, Holybourne Avenue, London, SW16 4JD, UK (Williams, Lee).

From Mayfield House, Brighton and Sussex Medical School, University of Brighton,

Falmer, Brighton, BN1 9PX, UK (Haq).

corresponding author

E-mail: williamj25@roehampton.ac.uk

Tel: +44 (0)20 8392 3539

Fax: +44 (0)20 8392 3531

1 **Is pain the cause of altered biomechanical functions in back pain sufferers?**

2 **Abstract**

3 Alterations in movement patterns and muscle activities of the lumbar spine are
4 frequently observed in patients with back pain. However there is considerable
5 disagreement as to the underlying causative mechanisms. It is imperative to identify
6 these mechanisms so that clinical management can be rationalized. One popular
7 theory suggests these alterations are “pain-driven”. This review presents a systematic
8 appraisal, including effect size calculation of studies utilizing experimentally induced
9 pain and experimental pain relief models to explore this concept. Fifteen studies were
10 identified using MEDLINE and a review of reference lists. Experimentally induced
11 pain did produce changes in electromyography and gait-related kinematics. However
12 these effects were not universal. Experimental pain relief studies produced mixed
13 results for improving maximal muscle function. These methodologies shared similar
14 threats to validity and failed to fully answer the question due to methodological
15 limitations; however possible mechanisms as to the effects of pain are discussed.
16 Further research is required to resolve if pain is indeed the variable driving functional
17 changes in kinematics and muscle functions observed in back pain sufferers, and if it
18 is to what degree. Only then can management strategies begin to be rationalized.

19 **Keywords:** Back pain; Kinematics; Pain; Review; Spine

20 **PsycINFO classification:** 3380 Rehabilitation

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1 Is pain the cause of altered biomechanical functions in back pain sufferers?

2 1. Introduction

3 Low back pain (LBP) is a major health and socioeconomic burden, and a leading
4 cause of disability (Frymoyer, 1988). LBP sufferers often display changes in
5 biomechanical behavior of the trunk including, but not limited to, alterations in
6 movement patterns and muscle activity. The nature of these alterations remains poorly
7 understood. Lumbar kinematics and muscle functions have long been a key focus
8 within LBP and are vitally important as they are strongly associated with risk of low
9 back pain onset and LBP reporting (Norman et al., 1998; Stevenson, Weber, Smith,
10 Dumas, & Albert, 2001; van Nieuwenhuyse et al., 2004) and are included in
11 governmental guidelines on safe handling and impairment measurement (Cocchiarella
12 & Andersson, 2000; Waters, Putz-Anderson, Garg, & Fine, 1993).

13 Kinematic alterations have commonly been identified in sufferers of LBP (Marras,
14 Davis, Ferguson, Lucas, & Gupta, 2001; Marras et al., 1999; Marras & Wongsam,
15 1986; Shum, Crosbie, & Lee, 2005a, 2005a; Wong & Lee, 2004) and the higher order
16 kinematics, such as velocity and acceleration, strongly correlate with the loss of
17 functions and disability (Marras et al., 1995, 2001, 1999; Marras & Wongsam, 1986;
18 Novy et al., 1999; Shum et al., 2005a, 2005b; Shum, Crosbie, & Lee, 2007). There
19 have also been attempts to study changes in muscle behavior (Hodges & Moseley,
20 2003; van Dieën, Selen, & Cholewicki, 2003) however no universal consensus exists.

21 Attempts to explain changes in amplitude of muscle activation have been complicated
22 by two conflicting models, the pain-spasm-pain model (Travell, Rinzler, & Herman,
23 1942) and the pain-adaption model (Lund, Donga, Widmer, & Stohler, 1991). The
24 pain-spasm-pain model predicts that pain will induce muscular hyperactivity or spasm
25 which would in-turn cause pain. However, the pain-adaption model predicts that when

1 pain is present, muscle activation patterns are altered according to their particular
2 function.

3 Previous reviews (Marras et al., 1999; van Dieën et al., 2003) have focussed on
4 reporting the alterations or impairments observed in LBP sufferers and more recent
5 reviews have analysed effect sizes, beginning to outline which alterations are
6 associated with LBP (Geisser et al., 2005). Reviews however have not been able to
7 address the causative mechanisms, therefore a critical review of the current
8 understanding of the issue of pain and lumbar kinematics and muscle function is
9 required.

10 Management strategies often involve targeting pain relief to alter spinal biomechanics
11 and functions (Jette, Smith, Haley, & Davis, 1994). If the aim of treatment is to
12 restore the biomechanical behavior of the spine then it is imperative to identify the
13 underlying cause or mechanism responsible for the biomechanical changes so that
14 clinical management can be rationalized. The question remains as to whether pain
15 drives movement and muscle changes. Therefore, this review aims to explore the
16 concept of pain driven changes in lumbar kinematics and muscle functions examining
17 the experimental pain models employed in this area of research. It will discuss how
18 this information can be used in the development and justification of clinical
19 management models aimed at restoring the biomechanical behavior of the spine.
20 However, it should be acknowledged that many other variables have been suggested
21 to cause changes in biomechanical functions, including, but not limited to, spinal
22 stiffness (Lee et al., 2005), and fear of movement (Thomas & France, 2007; Thomas,
23 France, Sha, & Vander Wiele, 2008), however by far the most commonly cited is pain
24 (Hodges & Moseley, 2003). It is pain that will form the basis of this review,

1 concentrating on the immediate effects of pain induction and pain relief on
2 biomechanical functions.

3 **2. Methods**

4 To be included in this review studies needed to meet the following criteria. Articles
5 needed to investigate either the effects of experimentally induced pain or that of
6 experimental pain relief related specifically to the low back region. The review was
7 limited to these methodologies as it is thought they studied the effects of pain as a
8 separate variable. All measurements had to be completed immediately and include
9 either lumbar kinematics or muscle function. Immediate measures only were selected
10 in an attempt to maximize the impact of altering just one variable; pain. Searches were
11 completed of Medline 1948-2009 (English language only) using a variety of terms
12 including LBP, experimentally induced pain, pain-relief, kinematics, and
13 biomechanics, along with reference lists of retrieved articles. Fifteen studies matching
14 the above criteria were retrieved and are presented in Tables 1 and 2. A systematic
15 review of the methodology of these studies was completed using a modified version
16 of the criteria list as suggested by Downs and Black (1998) (see Appendix 1) and the
17 results are presented in the appropriate column of Tables 1 and 2. Effect size
18 calculations were also carried out, the results of which are presented in Tables 1 and
19 2. It has been suggested that an effect size of 0.2 is small, 0.5 is moderate and > 0.8 is
20 large (Cohen, 1988, 1992).

21

22 **3. Results**

23 Studies utilizing the method of experimentally induced pain share common
24 methodologies and are at risk of common threats to validity. All studies failed to
25 report potential confounding issues (question 5) and adjust for any of these issues

(question 25). All studies failed to clearly report any adverse effects or an absence of adverse effects. Baseline characteristics are not often reported as initially the subjects are “normal”. These studies were all carried out in laboratory environment therefore all studies score poorly on question 13. Due to the nature of the experimental method blinding the subject and randomizing is not always possible, seen in the scoring of questions 14, 23, 24 and 25. One potential source of bias is the failure to blind data processing for trial type (question 15).

Table 2 outlines the results of the systematic methodological analysis for studies of experimental pain relief with most studies struggling to control confounding variables. Poor reporting of adverse effects is visible by low scores to question 8. This line of research enquiry often relies on a convenience or consecutive sample providing a threat to external validity.

Results of effect size calculations are presented in Tables 1 and 2. Thirteen effect size calculations were possible for experimentally induced pain studies, where the largest effect size was obtained for changes in onset time of deep lumbar multifidus during shoulder flexion. Large effect sizes were also evident for onset of transversus abdominus during shoulder flexion, thickness of transversus abdominus during abdominal hollowing, and mean amplitude of EMG for erector spinae during gait.

Three effect sizes were calculated for studies employing experimental pain relief, with small to moderate effect size for Sorensen test improvement displaying the largest effect size.

22

23 **4. Discussion**

24 **4.1 Methodological analysis**

1 The reporting of confounding issues is important to understanding the factors
 2 influencing the results, however despite the lack of reporting in experimentally
 3 induced pain models, it could be argued that the method of using “normals” is a good
 4 way of controlling or minimizing the impact of these confounding variables. It is
 5 however not clear whether pre-existing traits impact on the experience of induced
 6 pain and therefore affect the results. The importance of confounding variables in
 7 experimental pain relief studies should not be understated. It is well known that LBP
 8 populations are far from homogenous making it very difficult to control for these
 9 confounding variables, questioning the true meaning of the results. Due to the
 10 multifactorial nature of LBP, studies of this nature using a sample of convenience or
 11 consecutive samples are likely to contain mixed sub-groups. The reporting of adverse
 12 effects is important to determine the safety profile of specific interventions, which is
 13 imperative in pain relief trials if the interventions are to be advocated. Poor reporting
 14 of these factors in both groups means the safety of the experimental method of
 15 inducing pain is not clear and the clinical usefulness of pain relief strategies employed
 16 is not clear. The lack of blinding of investigators is commonly observed in these
 17 studies; however the importance of such is questionable as in this type of quantitative
 18 research processing methods are often automated by computer programs which
 19 remain consistent throughout the analysis.

21 **4.2 Experimental pain models**

22 As can be seen from Table 1, three studies utilizing induced pain to investigate the
 23 kinematics of gait. Clear attenuations in gait have been demonstrated in LBP sufferers
 24 (Keefe & Hill, 1985), however induced pain failed to alter relative phases of gait
 25 (Arendt-Nielsen, Graven-Nielsen, Sværre, & Svensson, 1995) or trunk coordination

1 in the transverse plane (Lamoth et al., 2004). It was deduced that the key determinant
2 of relative phase trunk coordination was in fact walking velocity not pain.

3 In contrast, Moe-Nilssen, Ljunggren, and Torebjork (1999) were able to show that
4 experimentally induced pain did indeed attenuate walking kinematics, but these
5 attenuations were not in the relative phase couples or phases of gait but were actually
6 in walking velocity itself. As can be seen in Table 1 previous studies had controlled
7 for walking velocity therefore masking this temporal kinematic change. Moe-Nilssen
8 et al. (1999) asked the subject to walk at a velocity of their choosing which varied on
9 prompting. This protocol enabled clear reductions in overall velocity to become
10 visible, something strongly associated with LBP sufferers (Keefe & Hill, 1985;
11 Lamoth, Daffertshofer, Meijer, & Beek, 2006; Lamoth, Stins, Pont, Kerckhoff, &
12 Beek, 2008; Lee, Simmonds, Etnyre, & Morris, 2007). This change results in more in-
13 phase relative coupling of the trunk (Lamoth et al., 2004) less spinal motion, lower
14 joint forces, and a situation closer to static loading for the lumbar spine (Callaghan,
15 Patla, & McGill, 1999).

16 It is clear that if equivocal speeds are investigated, induced pain does not alter lumbar
17 kinematics during gait, however when verbal cueing for speed, i.e., preferred speed,
18 or the interpretation of “as fast as possible”, alterations are seen in that slower speeds
19 are adopted regardless of the speed requested. Interestingly this is not only seen
20 during gait, it is also evident during forward bending. Zedka, Prochazka, Knight,
21 Gillard, and Gauthier (1999) showed that induced pain altered lumbar kinematics
22 during forward bending by reducing the velocity of movement, along with 10-40%
23 reduction in range of motion. Unfortunately effect size calculations were not possible
24 due to poor reporting of actual numbers. However subjects managed to move at
25 equivocal velocity and range when prompted by an accelerometer further suggesting

that pain in some way effects the selection of movement velocity. It appears that induced pain sufferers are able to achieve equivocal speeds but “choose” not to. This suggests a resetting of the “velocity” control from the nervous system when the body is “in pain” as it is not task specific (Simmonds, 2006), a finding also evident in clinical LBP (Marras, Lewis, Ferguson, & Parnianpour, 2000; Marras & Wongsam, 1986; Novy et al., 1999). It seems logical that this may be a strategy to reduce loads on sensitive tissues, as greater velocities are known to result in greater spinal loads (Callaghan et al., 1999; Cheng, Chen, Chen, & Lee, 1998). Interestingly however it has been noted that when asked to move as fast as possible, clinical LBP sufferers are often unable to achieve equivocal speeds to matched controls (Lee et al., 2000; Marras & Wongsam, 1986). The true reason for this remains unclear; however it may represent an unwillingness to evoke pain (if pain driven) or a loss of functional capacity of the lumbar spine suggesting a mechanism other than pain may be important.

In order to study the effects of experimentally induced pain on trunk muscle function electric shock and hypertonic saline have been utilized with a focus on paraspinal and abdominal muscles. Voluntary arm movements coupled with a painful stimulus (electric shock) show a gradual process of activation change in both the lower abdominals (transversus abdominus/internal oblique) and, although less dramatic, in external oblique (Moseley & Hodges, 2005). This gradual process of reduced activation of the lower abdominals has been argued to represent an adaptation towards an alternate trunk muscle strategy and appears to suggest that pain (experimentally induced) may have the capacity to drive change in trunk muscle activation strategies. Moreover, following the pain-movement coupling, a period of uncoupling was completed where a return to the original activation patterns were observed, further

1 suggesting pain may be the key instigator for these changes. Unfortunately no real
2 numbers were reported making the interpretation of the magnitude of effect or an
3 effect size calculation not possible. Importantly this method of inducing pain enables
4 the observation of non-immediate changes whilst minimising the impact of other
5 potential variables. Saline injection is very short lasting and is unable to study the
6 subtle changes occurring over time, whereas electric shock can be delivered over a
7 longer time period allowing the lumbar system time to adapt to the noxious stimulus.
8 Adopting similar methodology, but using saline injections to induce pain, EMG
9 results have shown a consistent pattern of reduced or delayed activation during
10 voluntary arm movements (Hodges, Moseley, Gabrielsson, & Gandevia, 2003;
11 Moseley et al., 2004). These findings are believed to mirror that of small clinical LBP
12 trials (Hodges & Richardson, 1996, 1999). These studies follow a similar protocol
13 involving a static posture onto which the subject performs a rapid shoulder movement.
14 This relatively simple task relies on the adoption of identical postures throughout due
15 to the effect of small postural changes on trunk muscle EMG (Claus, Hides, Moseley,
16 & Hodges, 2009; O'Sullivan et al., 2002, 2006), however no postural measures were
17 conducted to ensure this criteria was controlled. It is important to note that the
18 impressive effect sizes reported for these studies represent a measure of statistical
19 assurance rather than magnitude of effect. The magnitude of difference for muscle
20 onset relative to deltoid, compared with controls was 28ms and 10.3ms for transversus
21 abdominus (Hodges et al., 2003; Moseley et al., 2004). Interestingly isotonic saline
22 (not painful) also had a significant impact on the latency of onset of transversus
23 abdominus compared with controls, with the magnitude of delay in onset being 5.2ms,
24 along with a delay of 25.1ms for superficial multifidus suggesting factors other than
25 pain may at least be of some significance (Hodges et al., 2003). Furthermore, when

1 studying truly comparable experimental conditions, namely isotonic saline with
2 hypertonic saline, the onset difference was 5.1 ms for transversus abdominus. These
3 results are further complicated by the use of visual inspection to detect EMG onset
4 rather than an automated computer algorithm resulting in a potential source of bias
5 (Allison, 2003; DiFabio, 1987; Hodges & Bui, 1996). The clinical significance of
6 such a small delay in muscle onset is not well understood.

7
8 It has been previously reported that trunk kinematics are affected by the induction of
9 pain and this often takes the form of reduced velocity of motion. In the analysis of the
10 experimental method used by the above studies, reported kinematic data regarding the
11 moving arm is often insufficient. Moseley and Hodges (2005) report only deltoid
12 EMG parameters, whereas Hodges et al. (2003) and Moseley et al. (2004) only report
13 *p*-values for peak acceleration. The reporting of shoulder movement velocity is
14 critical as this has a large effect on trunk muscle onsets during this experimental
15 protocol (Hodges & Richardson, 1997). As the magnitude of such an effect has been
16 reported as a 294-ms delay in transversus abdominus onset for slow limb movement
17 compared with 19 ms for preferred speed, it is unclear if the delays outlined by these
18 studies are the result of pain or are the manifestation of minor alterations in shoulder
19 movement velocity.

20
21 Similar findings have been observed using ultrasound imaging where changes
22 suggestive of reduced activation were observed for transversus abdominus, during
23 abdominal hollowing and lumbar multifidus during prone limb raising (Kiesel, Uhl,
24 Underwood, & Nitz, 2008). The findings were further replicated using a novel

functional magnetic resonance imaging method, displaying changes suggestive of reduced activation of both lumbar multifidus and erector spinae during a Sorensen manoeuvre (Dickx et al., 2008). These studies tested activity in a static condition removing any velocity deviations which may confuse interpretation, suggesting that pain may indeed attenuate changes in muscle activation. However, it should be remembered that thickness is a morphological parameter which may not directly reflect muscle function and that there are inherent difficulties with accurate re-positioning of the ultrasound probe leading to significant errors in thickness measurement, around 6-10% for multifidus cross-sectional area (Stokes, Rankin, & Newham, 2005).

Unfortunately these studies only examined very simple activities such as arm movement and fail to provide answers as to the effect of experimental pain on muscle activation during more functional tasks. Studies on functional tasks have provided conflicting results due to methodological differences in analysis techniques and tasks completed (see Table 1). During lumbar flexion, following right sided erector spinae muscle injection, a loss of bilateral flexion relaxation response in the erector spinae was observed, something highly correlated to LBP sufferers (Geisser et al., 2005; Watson, Booker, Main, & Chen, 1997) along with reduced activation during the return from flexion, a time normally associated with high activation levels (Zedka et al., 1999). However, when the subjects were guided to complete the flexion motion identical to the painless trial (equivocal range and velocity), only the injected side displayed alteration (Zedka et al., 1999). Therefore it appears that the spine still has the functional reserve to achieve more selective muscle activation patterns in these temporary pain states, but an alternative strategy is adopted. This could reflect an

1 attempt to avoid asymmetrical loading associated with unilateral muscle activity or a
 2 more gross reaction where the nervous system switches to function in an altered 'pain
 3 mode' regardless of the location of pain. This finding has also been seen during gait
 4 (Arendt-Nielsen et al., 1995; Lamothe et al., 2004). This suggests increased activation
 5 at a time normally associated with little or no activity, along with decreased activity
 6 during a time normally associated with large activation. These superficial muscle
 7 activation changes appear to mirror that of the pain-adaptation model (Lund et al.,
 8 1991); however these biomechanical changes are only evident during self-selection of
 9 velocity during functional tasks. The reason for these changes is unclear. The
 10 choosing of slower functional movements may be the cause or effect of muscle
 11 activity changes. These changes may result in a reduction of movement velocity or a
 12 reduction in movement velocity may cause an increase in superficial muscle activity.
 13 Furthermore these changes may be the cause or result of changes in the deep trunk
 14 musculature. Reductions in movement velocity and alterations in superficial trunk
 15 muscle activity may result in an alteration in the deep muscle activation requirements
 16 for highly specific and coordinated activities.

17

18 Caution should be exercised when extrapolating these results to clinical pain as
 19 experimentally induced pain fails to closely mimic clinical pain. The resultant pain is
 20 always constant in nature with very little deviation except a gradual reduction over
 21 time. The pain source in these subjects is likely to be the nociceptors within the
 22 muscle, irritated chemically and locally, the presence of which in true clinical LBP is
 23 not known. It is also noteworthy that these experiments often involve injections at the
 24 level L3 (see Table 1) whereas clinically the highest incidence in LBP is known to be
 25 the two lower levels. Furthermore, due to the transient nature any alteration in central

1 pain processing will be minimal, as will the levels of concern regarding uncertainty
2 about the personal meaning of their LBP.

3

4 In summary it seems from the results of these studies that experimentally induced pain
5 results in an automatic attenuation of range of motion and reduction in movement
6 velocity. Induced pain results in an elevation of superficial muscle activity which is
7 often bilateral, if autonomous selection of velocity and range are permitted. This may
8 represent a protective response and act as a method of reducing range and velocity of
9 motion. Functional tasks appear to display EMG changes which correspond to an
10 increase in activation at a time normally associated with little or no activity in the
11 ipsilateral erector spinae. This is consistent with aspects of the pain-adaptation model,
12 which unfortunately is not seen consistently in LBP sufferers (van Dieën et al., 2003).
13 However not consistent with this model is the small delay in onset activation seen in
14 the deep muscles during shoulder movements. It is true that some of these changes
15 appear to mirror those present in LBP sufferers clinically and may suggest therefore
16 that they represent pain induced changes. It is clear that the musculoskeletal system is
17 capable of achieving equivocal ranges and velocities if guided, along with more
18 selective EMG patterns, suggesting an alteration in the nervous system control of
19 movement parameters when in an experimentally induced pain state. Future studies
20 should take care to identify and control for confounding variables and adjust the
21 method or analysis accordingly.

22

23 **4.3 Pain relief models**

Obviously in order to overcome the limitations of the experimental pain model a painful clinical sample could be studied and the effects of pain relief investigated. The effect of pain relief on muscle testing has shown mixed results (see Table 2). Using the Biering-Sorensen test with a group of chronic LBP sufferers, Rashid, Koller, Haykowsky, and Jamieson (2003) showed that intravenous opioid increased performance some 28% compared with placebo. Furthermore Jarzem, Harvey, Arcaro, and Kaczorowski (2005) displayed 15% gains in maximal isometric lifting capacity following TENS induced pain relief, compared with sham TENS. Conversely, Holm, Friis, Brox, Gunderson, and Steen (2000) utilizing bilateral zygapophyseal joint injections to induce pain relief in chronic sufferers, failed to detect a significant change in muscle function as measured by an isokinetic through-range resistance task. It is important to note however that the researchers struggled to achieve a significant reduction in pain in all three studies with visual or verbal analogue scale (VAS) changes ranging from 0.9 to 21.2 mm. This may reflect the underlying pathological changes or subtle differences in baseline characteristics (see Table 2), or the inherent difficulty of reducing pain when using maximal muscle test outcomes. This notion is further complicated by psychometric testing, suggesting the Biering -Sorenson test examines pain tolerance and motivation, rather than muscular endurance (Novy et al., 2002) and that fluid injected into joints may have an inhibitory effect on muscle activation (Spencer, Hayes, & Alexander, 1984).

Using zygapophyseal joint injections in chronic LBP, Lilius, Laasonen, Myllynen, Harilainen, and Gronlund (1989) displayed immediate improvements in flexion and rotation range of motion following an 18.3mm reduction in VAS (see Table 2). This ROM change was not universal as no difference was seen in extension or lateral flexion. Similar results were obtained when using TENS for pain relief which resulted

1 in small gains in flexion and extension ROM. However despite significant results,
2 actual change in ROM were either not reported (Jarzem et al., 2005) or very small, 1.4
3 cm and 1.5 degrees for flexion and rotation respectively (Lilius et al., 1989). Despite
4 the fact that no attempts were made to correlate pain reduction and functional change,
5 the findings do suggest that minor pain relief may be capable of changing lumbar
6 kinematics; however the clinical significance of the magnitude of change could be
7 questioned.

8 It seems evident that the voluntary selection of speed is not only affected by
9 experimental and clinical pain (Simmonds & Rebelo, 2003; Zedka et al., 1999) it may
10 be affected by pain-relief. In studies investigating velocity it is evident that pain relief
11 is capable of increasing movement velocity (Davis, & Kotowski, 2005; Simmonds, &
12 Rebelo, 2003). Unfortunately, Davis and Kotowski (2005) failed to present actual
13 pain data or describe the interventions (massage, chiropractic, physical therapy or
14 acupuncture) making the correlation between pain relief and kinematic change
15 difficult. In their study investigating time taken to complete a repeated sit-to-stand
16 task at three different self-selected speeds, Simmonds and Rebelo (2003) were able to
17 show that that the fastest speed achieved by the chronic LBP group was equivocal to
18 the preferred speed of the control group. These results suggest that chronic LBP
19 subjects may actually be unable to achieve the same movement speeds, something
20 observed previously in a clinical population (Marras et al., 2000; Marras & Wongsam,
21 1986; Novy et al., 1999; Simmonds & Rebelo, 2003). Unfortunately it is not clear
22 whether the task evoked pain at the time of testing, resulting in an unwillingness to
23 move faster due to pain provocation or whether the LBP sufferer just doesn't have the
24 functional capacity to produce the same speed, due to some unknown mechanism.
25 However, following pain relief induced by a superficial heat wrap, the LBP group

1 significantly increased their sit-to-stand speed, interestingly only at the preferred
 2 speed. Therefore pain relief may have resulted in an adaptation of the lumbar spine
 3 system through its neural control causing a shift away from its “pain” setting
 4 (Simmonds, 2006) at the preferred speed. This may to some extent explain the
 5 positive clinical effects associated with the application of topical heat (Nadler,
 6 Steiner, Erasala, et al., 2003; Nadler et al., 2002; Nadler, Steiner, Petty, et al., 2003).
 7 Moreover, due to the specific nature of the changes it could be argued that the effects
 8 were unlikely due to changes in the deeper tissues known to be influenced by heat
 9 (Bass et al., 2007), but rather due to the simple relief of pain.
 10 In summary, there is some evidence to suggest that pain relief results in an automatic
 11 increase in movement velocity when the self selection of speed is permitted during
 12 functional tasks. It appears feasible that pain relief may be able to alter the ROM but
 13 the true magnitude of effect suggests questionable clinical significance. It remains
 14 unclear as to how pain relief affects muscle function as the results are variable.
 15 Isometrically, performance is improved; however through range strength testing
 16 shows no effect. It is questionable how an improvement in maximal muscle testing
 17 relates to functional daily tasks, which seldom require the full capacity of the lumbar
 18 muscles and no EMG studies have been conducted. Future research should
 19 concentrate on careful selection of inclusion criteria in an attempt to create a relatively
 20 homogenous sample and minimize confounding variables, along with careful
 21 reporting of adverse effects to determine the clinical application of the specific pain
 22 relief strategies investigated.

23

24 **5. Conclusion**

This paper has provided a contemporary review of the most current understanding of the relationship between pain and biomechanical functions of the trunk. It has identified several important biomechanical features associated with LBP. These relate to the altered kinematic patterns and associated changes in muscle functions during self-selected tasks. The reason for these changes remains unclear. There is clearly a relationship between movement velocity and activity changes in the superficial or deep trunk muscles and we do not know their cause and effect or how the deep and superficial muscles interact. It is suggested that the CNS may require more “time” to control and coordinate movements, modify muscle activities and thus minimize pain provocation. On the other hand, it is entirely possible that these adaptations could be detrimental to spinal health and function. Pain may induce the cascade of movement and muscle changes which represent sub-optimal function, providing an ongoing mechanism for symptom provocation.

Understanding the mechanisms behind these alterations would significantly enhance the understanding of their functions. Clinical management may involve interventions which optimize pain relief or target some other mechanisms causing biomechanical change. Therefore further studies are required to isolate and identify individual mechanisms and to test their influence on trunk muscle functions and kinematics.

Only once the key mechanisms underpinning the alteration in function in clinical LBP populations are identified can clinicians and researchers alike begin to employ rational and specific interventions resulting in the restoration of normal biomechanical behavior of the trunk.

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16

Appendix

Checklist for measuring study quality (Downs & Black, 1998)

1. Is the hypothesis/aim/objective of the study clearly described?
2. Are the main outcomes to be measured clearly described in the Introduction or Methods section?
3. Are the characteristics of the patients included in the study clearly described ?
4. Are the interventions of interest clearly described?
5. Are the distributions of principal confounders in each group of subjects to be compared clearly described?
6. Are the main findings of the study clearly described?
7. Does the study provide estimates of the random variability in the data for the main outcomes?
8. Have all important adverse events that may be a consequence of the intervention been reported?
9. Have the characteristics of patients lost to follow-up been described?
10. Have actual probability values been reported (e.g. 0.035 rather than <0.05) for the main outcomes except where the probability value is less than 0.001?
11. Were the subjects asked to participate in the study representative of the entire population from which they were recruited?
12. Were those subjects who were prepared to participate representative of the entire population from which they were recruited?
13. Were the staff, places, and facilities where the patients were treated, representative of the treatment the majority of patients receive?
14. Was an attempt made to blind study subjects to the intervention they have received ?
15. Was an attempt made to blind those measuring the main outcomes of the intervention?
16. If any of the results of the study were based on "data dredging", was this made clear?
17. In trials and cohort studies, do the analyses adjust for different lengths of follow-up of patients, or in case-control studies, is the time period between the intervention and outcome the same for cases and controls ?
18. Were the statistical tests used to assess the main outcomes appropriate?
19. Was compliance with the intervention/s reliable?
20. Were the main outcome measures used accurate (valid and reliable)?
21. Were the patients in different intervention groups (trials and cohort studies) or were the cases and controls (case-control studies) recruited from the same population?
22. Were study subjects in different intervention groups (trials and cohort studies) or were the cases and controls (case-control studies) recruited over the same period of time?
23. Were study subjects randomised to intervention groups?
24. Was the randomised intervention assignment concealed from both patients and health care staff until recruitment was complete and irrevocable?
All non-randomised studies should be
25. Was there adequate adjustment for confounding in the analyses from which the main findings were drawn?
26. Were losses of patients to follow-up taken into account?

Table 1. Experimentally induced pain studies

Authors	Subjects and Task	Induced pain	Outcome measures	Results	Comments	Effect Size	Downs and Black Missing criteria
Arendt-Nielsen et al., (1995)	10 male Age: 23-30 No LBP Treadmill walk 4.0km/h for 1 minute.	0.5ml 5% saline. Depth: 15mm. Location: 40-45mm lateral to L3 (right).	Gait phases. EMG Amplitude. Right/Left Ratio. Peak activity at double stance phase (peak ₅₀). Mean EMG in contra-ipsilateral swing phases. Surface EMG placement: Lateral to Th 12, L2, L2 lat, L4 bilaterally.	Gait phases not affect by pain. EMG amplitude increased 8.5%. No change in ratio. Peak ₅₀ decreased 7.3%. Mean EMG during contra- and ipsilateral swing increased 15.1% and 19.2% respectively. No correlation between EMG and VAS (R=0.53).	Mean VAS 5.4±2.3. EMG ipsilateral to pain at L2 showed most significant changes.	1.2* (Mean EMG) 0.81* (Peak50) 1.04* (Contralateral swing) 1.03* (Ipsilateral swing)	3, 5, 8, 11, 12, 14, 15, 21-25
Zedka et al., (1999)	4 males and 1 female. Age: 20-55 Flexion (constrained lower limbs).	5% Saline. Depth: 40mm. Location: 30mm lateral to L3 (right).	Trunk displacement. Velocity of motion. EMG amplitude. Surface EMG placement: Lateral to L3	Reduced trunk displacement velocity. EMG amplitude during flexion unchanged. Loss of FRR bilaterally. EMG amplitude decreased during	Mean VAS 5.3±0.8. During guided painful movement EMG on contralateral side identical to painless trial.	NA	3, 5, 8, 10-12, 14, 15, 21-25

			bilaterally	return from flexion bilaterally.			
Lamoth et al., (2004)	8 male and 4 female. Age: 18-25 No LBP. Varied speed walking.	0.5ml 5% saline. Depth: 30mm. Location: 65mm lateral to L3 (right).	Relative phase coupling. EMG amplitude and patterns. Surface EMG placement: 30mm lateral to Th12, L2, L4 bilaterally	No effect on phase coupling. Elevated right EMG during ipsilateral swing for all locations and for L2 during contralateral swing phase. Elevated left EMG amplitude for ipsilateral swing phase.	Mean VAS 6.1±1.9. No correlations between pain intensity and EMG findings.	NA	5, 8, 12, 15, 22, 24, 25
Moe- Nilssen et al., (1999)	3 male and 19 female. Age: 20-49 Walking at five self adjusted speeds.	1ml 6% saline Depth: 15.2mm Location: 34mm lateral to Th12 or L1 on the left.	Trunk acceleration during walking: AP axes, ML axes, Vertical axes	Significant attenuation of acceleration in AP and ML axes when pain evident. Not for vertical axes.	Mean VAS 6.1±1.49. 15 of 20 showed correlation R ² 0.36-0.89 between pain and gait changes.	0.75* (AP accel) 0.27* (Vertical accel) 0.75* (Mediolateral accel)	5, 8, 9, 12, 14, 15, 22- 25
Moseley et al., (2004)	5 male and 3 female. Age: 32±7 No LBP. Shoulder flexion (standing)	1-1.5ml 5% saline. Depth: 30mm. Location: 50mm lateral to L4.	Temporal and Spatial parameters of EMG (related to reaction times). Intramuscular EMG: deep LM, TrAb,	Delay in onset of TrAb in painful condition. Control reaction time = 154±15ms. Pain = 189±19ms.	Mean VAS ?6.2 (taken from graph). Superficial injection of lidocaine included. Variable superficial muscle	2.19 (emg onset TrAb)	5, 8, 10- 12, 14, 22-25

			sup LM, OE, OI.		alteration in response to pain.		
Hodges et al., (2003)	5 male and 2 female. Age: 28.6±3.6 No LBP. Shoulder flexion (standing)	1.5ml 5% saline. Depth: 35mm. Location: 60mm lateral to L4.	Temporal and Spatial parameters of EMG (related to reaction times). Intramuscular EMG: TrAb, OE, OI, deep LM, sup LM.	Onset of TrAb-actual difference: Control= -18.9±2.4 (ms). Isotonic Saline = -13.7±4.0 (ms). Hypertonic Saline = -8.6±4.1 (ms). Onset of deepLM-actual difference: Control=47.3±5.5 (ms). Hypertonic Saline = 24.4±5.5 (ms).	Mean VAS 6.2±1.0. Superficial injection of lidocaine included.	3.31 (emg onset TrAb). 4.49 (emg onset deep LM).	5, 8, 10-12, 14, 22-25
Moseley and Hodges, (2005)	7 male and 9 female. Age: 24±5 No LBP. Shoulder flexion (sitting)	Noxious cutaneous electric stimulation bilaterally over the PSIS.	Temporal and Spatial parameters of EMG (related to reaction times). Surface EMG placement: TrAb/OI, EO and deltoid.	Significant and progressive delay in TrAb/OI and significant and progressively earlier onset of OE during pain trials.	No actual figures presented. Resolution of pre-pain on removal of noxious stimulus.	NA	5, 7, 8, 10-12, 14, 22-25
Kiesel et al., (2008)	6 male. Age: 26±7.3 No LBP. Abdominal 'drawing-in' (crook lying);	1.5ml 5% saline. Depth: 35mm. Location: 60mm lateral to	Ultrasound measured thickness of TrAb and LM at rest and during contraction.	Significant difference in thickness of TrAb and LM during contraction.	Optional 0.5ml 1% lidocaine subcutaneously.	1.31 (us thickness TrAb) 0.53 – 0.63 (us thickness LM)	5, 8-12, 14, 22-26

	prone limb raise.	L4.					
Dickx et al., (2008)	15 male Age: 23.33±0.8 No LBP. Sorenson test.	1.5ml 5% saline. Depth: 25mm. Location: 40mm lateral to L4.	Muscle functional MRI.	Decreased muscle activity in LM and erector spinae during painful exercise. Results show difficulty activating in the presence of pain.	5.3-5.9 VAS during exercise. Exercised at 40% of 1RM	0.000032 – 0.58 (shifts in T2 value in ms)	5, 8, 11, 12, 14, 15, 22-25

EMG, electromyography; VAS, visual analogue scale; L1, 2, 3, 4, 5, respective lumbar vertebrae; Th12, 12th thoracic vertebrae;

FRR, flexion relaxation response; AP, anterior-posterior; ML, medio-lateral; LM, lumbar multifidus; sup LM, superficial lumbar multifidus; OE, obliquus externus; OI, obliquus internus; TrAb, transversus abdominus; US, ultrasound scan; MRI, magnetic resonance imaging; RM, repetition maximum; NA, numbers not available.

Table 2. Experimental pain-relief studies.

Authors	Subjects and Tasks	Pain relief model	Outcome measures	Results	Comments	Effect Size	Down and Blacks Missing Criteria
Rashiq et al., (2003)	17 male and 11 female. Age: 54, range 23-78. CLBP duration: 10-420 months. Mixed diagnosis. Sorenson test.	IV fentanyl 1µg/kg. Significant pain relief reported (0.9 on VRS)	Sorenson test time	Saline injection Sorenson score = 60±42s Fentanyl injection Sorenson score = 77±49s	Minimal pain relief. Large variation in performance.	0.38	5, 8, 12, 25

Holm et al., (2000)	38 male and 49 female. Age: 48, range 22-79. CLBP duration: 12.3 yrs. Mixed diagnosis. Isokinetic Dynamometer for trunk flexion/extension at 60°/s and 120°/s.	Bilateral ZA joint injection L5/S1 ± L4/5. Significant pain relief (21.2mm).	Total work (joule) at 60°/s and 120°/s.	No significant difference in performance following injection.	Weak correlation between decrease in pain and increase in muscle performance.	0.0003-0.18	3, 5, 8-12, 14, 15, 21, 23-25
Lilius et al., (1989)	48 male and 61 female. Age: 44, range 19-64. CLBP >3/12. ROM and observed scored disability battery (blinded scorer).	Intra-articular steroid injection to ZA joints. Pericapsular steroid injection. Intra-articular saline injection. Significant pain relief across groups (mean: 18.3mm).	VAS (pain) Disability score. ROM.	Significant improvements in likert scale for disability. Improvements in flexion and rotation ROM.	ROM measured with tape measure. Rotation measured with a compass. Questionable clinically significant result. Significant placebo effect.	0.08	5, 11, 12, 21, 22, 24, 25
Jarzem et al., (2005)	29 male and 21 female.	TENS (50%	VAS (pain). ROM	Statistically significant	Real numbers not reported.	NA	5, 7, 8, 11, 12, 21, 22,

	Age: 38.9±14.5. CLBP >3/12 (no leg pain). Physical battery.	reduction in pain).	(gravity goniometer). Isometric lifting capacity. Maximum repetitions of physical test battery.	gain in ROM. 15% gain in isometric lift capacity post 'real' TENS. Statistically significant increase in maximum repetitions for all tasks			24, 25
Davis and Kotowski, (2005)	3 male and 3 female. Age: 42.7±13.8. 4 male and 2 female. Age: 37.0±4.7. 6 female. Age: 44.3±8.5. 2 male and 4 female. 44.0±21.1.	Acupuncture Chiropractic Massage Physical Therapy	Adapted RMDQ and NASS LSOAI. Functional capacity evaluation (LMM).	Acupuncture ↑ lat vel; lat acc; Tw vel. ↓ Tw acc. Chiropractic ↑ lat vel; lat acc; Tw vel; Tw acc Massage ↑ lat vel; lat acc; Tw vel; Tw acc. PT ↑ lat vel; lat acc; Tw vel ↓ Tw acc.	No direct measurement or reporting on pain scores.	NA	3-8, 11, 12, 14, 15, 21-25
Simmonds and Rebelo, (2003)	5 male and 10 female. Age: 44.4±6.4. LBP (undefined acute or chronic). Matched controls. Repeated sit to stand (5) at 3 self varied speeds.	Superficial heat wrap to both groups – 40mins. Significant pain relief (t=3.2, p=0.006, actual	VAS (pain) Total time to complete sit to stand task.	Pain relief resulted in significant increase in preferred speed only.	Actual difference in region of 5s (calculated from (Simmonds, 2006)).	NA	Conference proceeding only

		numbers not reported).					
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IV, Intravenous; VRS, verbal rating scale; CLBP, chronic low back pain; ZA, Zygopophyseal; ROM, range of motion; VAS, visual analogue scale; Flex, Flexion; TENS, transcutaneous electrical nerve stimulation; RMDQ, Roland Morris Disability Questionnaire; NASS LSOAI, North American Spine Society Lumbar Spine Outcome Assessment Instrument; LMM, Lumbar Motion Monitor; *, data retrieved from graph; lat vel, lateral velocity; lat acc, lateral acceleration; Tw vel, twisting velocity; Tw acc, twisting acceleration; ↓↑, decreased, increased.

ACCEPTED MANUSCRIPT