



**HAL**  
open science

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

Jonathan M. Williams, Inam Haq, Raymond Y. Lee

► **To cite this version:**

Jonathan M. Williams, Inam Haq, Raymond Y. Lee. Is pain the cause of altered biomechanical functions in back pain sufferers?. *Human Movement Science*, 2010, 29 (2), pp.311. 10.1016/j.humov.2009.12.001 . hal-00627138

**HAL Id: hal-00627138**

**<https://hal.science/hal-00627138>**

Submitted on 28 Sep 2011

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

## Accepted Manuscript

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

Jonathan M. Williams, Inam Haq, Raymond Y. Lee

PII: S0167-9457(09)00115-8

DOI: [10.1016/j.humov.2009.12.001](https://doi.org/10.1016/j.humov.2009.12.001)

Reference: HUMOV 1193

To appear in: *Human Movement Science*



Please cite this article as: Williams, J.M., Haq, I., Lee, R.Y., Is pain the cause of altered biomechanical functions in back pain sufferers?, *Human Movement Science* (2010), doi: [10.1016/j.humov.2009.12.001](https://doi.org/10.1016/j.humov.2009.12.001)

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

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

3

4 Jonathan M. Williams, MManipTher, #

5

6 Inam Haq, MD, MBBS,

7

8 Raymond Y. Lee, PhD,

9

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

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

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

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

14

15 # corresponding author

16 E-mail: [williamj25@roehampton.ac.uk](mailto:williamj25@roehampton.ac.uk)

17 Tel: +44 (0)20 8392 3539

18 Fax: +44 (0)20 8392 3531

19

20

21

22

23

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

21

22

23

24

# 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 Nieuwenhuysen 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

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

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

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

19 Three effect sizes were calculated for studies employing experimental pain relief, with  
20 small to moderate effect size for Sorensen test improvement displaying the largest  
21 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.

20

#### 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, Svarrer, & 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

1 that pain in some way effects the selection of movement velocity. It appears that  
2 induced pain sufferers are able to achieve equivocal speeds but “choose” not to. This  
3 suggests a resetting of the “velocity” control from the nervous system when the body  
4 is “in pain” as it is not task specific (Simmonds, 2006), a finding also evident in  
5 clinical LBP (Marras, Lewis, Ferguson, & Parnianpour, 2000; Marras & Wongsam,  
6 1986; Novy et al., 1999). It seems logical that this may be a strategy to reduce loads  
7 on sensitive tissues, as greater velocities are known to result in greater spinal loads  
8 (Callaghan et al., 1999; Cheng, Chen, Chen, & Lee, 1998). Interestingly however it  
9 has been noted that when asked to move as fast as possible, clinical LBP sufferers are  
10 often unable to achieve equivocal speeds to matched controls (Lee et al., 2000; Marras  
11 & Wongsam, 1986). The true reason for this remains unclear; however it may  
12 represent an unwillingness to evoke pain (if pain driven) or a loss of functional  
13 capacity of the lumbar spine suggesting a mechanism other than pain may be  
14 important.

15 In order to study the effects of experimentally induced pain on trunk muscle function  
16 electric shock and hypertonic saline have been utilized with a focus on paraspinal and  
17 abdominal muscles. Voluntary arm movements coupled with a painful stimulus  
18 (electric shock) show a gradual process of activation change in both the lower  
19 abdominals (transversus abdominus/internal oblique) and, although less dramatic, in  
20 external oblique (Moseley & Hodges, 2005). This gradual process of reduced  
21 activation of the lower abdominals has been argued to represent an adaptation towards  
22 an alternate trunk muscle strategy and appears to suggest that pain (experimentally  
23 induced) may have the capacity to drive change in trunk muscle activation strategies.  
24 Moreover, following the pain-movement coupling, a period of uncoupling was  
25 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

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

11

12 Unfortunately these studies only examined very simple activities such as arm  
13 movement and fail to provide answers as to the effect of experimental pain on muscle  
14 activation during more functional tasks. Studies on functional tasks have provided  
15 conflicting results due to methodological differences in analysis techniques and tasks  
16 completed (see Table 1). During lumbar flexion, following right sided erector spinae  
17 muscle injection, a loss of bilateral flexion relaxation response in the erector spinae  
18 was observed, something highly correlated to LBP sufferers (Geisser et al., 2005;  
19 Watson, Booker, Main, & Chen, 1997) along with reduced activation during the  
20 return from flexion, a time normally associated with high activation levels (Zedka et  
21 al., 1999). However, when the subjects were guided to complete the flexion motion  
22 identical to the painless trial (equivocal range and velocity), only the injected side  
23 displayed alteration (Zedka et al., 1999). Therefore it appears that the spine still has  
24 the functional reserve to achieve more selective muscle activation patterns in these  
25 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**



1 Obviously in order to overcome the limitations of the experimental pain model a  
2 painful clinical sample could be studied and the effects of pain relief investigated. The  
3 effect of pain relief on muscle testing has shown mixed results (see Table 2). Using  
4 the Biering-Sorensen test with a group of chronic LBP sufferers, Rashed, Koller,  
5 Haykowsky, and Jamieson (2003) showed that intravenous opioid increased  
6 performance some 28% compared with placebo. Furthermore Jarzem, Harvey, Arcaro,  
7 and Kaczorowski (2005) displayed 15% gains in maximal isometric lifting capacity  
8 following TENS induced pain relief, compared with sham TENS. Conversely, Holm,  
9 Friis, Brox, Gunderson, and Steen (2000) utilizing bilateral zygapophyseal joint  
10 injections to induce pain relief in chronic sufferers, failed to detect a significant  
11 change in muscle function as measured by an isokinetic through-range resistance task.  
12 It is important to note however that the researchers struggled to achieve a significant  
13 reduction in pain in all three studies with visual or verbal analogue scale (VAS)  
14 changes ranging from 0.9 to 21.2 mm. This may reflect the underlying pathological  
15 changes or subtle differences in baseline characteristics (see Table 2), or the inherent  
16 difficulty of reducing pain when using maximal muscle test outcomes. This notion is  
17 further complicated by psychometric testing, suggesting the Biering -Sorenson test  
18 examines pain tolerance and motivation, rather than muscular endurance (Novy et al.,  
19 2002) and that fluid injected into joints may have an inhibitory effect on muscle  
20 activation (Spencer, Hayes, & Alexander, 1984).

21 Using zygapophyseal joint injections in chronic LBP, Lilius, Laasonen, Myllynen,  
22 Harilainen, and Gronlund (1989) displayed immediate improvements in flexion and  
23 rotation range of motion following an 18.3mm reduction in VAS (see Table 2). This  
24 ROM change was not universal as no difference was seen in extension or lateral  
25 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**

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

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

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

23

24

1 **6. References**

2

- 3 Allison, G. T. (2003). Trunk muscle onset detection technique for EMG signals with  
4 ECG artefact. *Journal of Electromyography and Kinesiology*, *13*, 209-216.
- 5 Arendt-Nielsen, L., Graven-Nielsen, T., Sværre, H., & Svensson, P. (1995). The  
6 influence of low back pain on muscle activity and coordination during gait: A  
7 clinical and experimental study. *Pain*, *64*, 231-240.
- 8 Bass, C., Planchak, C., Salzar, R., Lucas, S., Rafaels, K., Shender, B., & Paskoff, G.  
9 (2007). The temperature-dependent viscoelasticity of porcine lumbar spine  
10 ligaments. *Spine*, *32*, E436-442.
- 11 Callaghan, J. P., Patla, A. E., & McGill, S. M. (1999). Low back three-dimensional  
12 joint forces, kinematics, and kinetics during walking. *Clinical Biomechanics*,  
13 *14*, 203-216.
- 14 Cheng, C. K., Chen, H. H., Chen, C. S., & Lee, S. J. (1998). Influences of walking  
15 speed change on the lumbosacral joint force distribution. *Bio-medical*  
16 *Materials and Engineering*, *8*, 155-165.
- 17 Claus, A. P., Hides, J. A., Moseley, G. L., & Hodges, P. W. (2009). Different ways to  
18 balance the spine. Subtle changes in sagittal spinal curves affect regional  
19 muscle activity. *Spine*, *34*, E208-E214.
- 20 Cocchiarella, L., & Andersson, G. B. J. (2000). *Guides to the evaluation of permanent*  
21 *impairment*. AMA Bookstore.
- 22 Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*, Hillsdale, NJ:  
23 Lawrence Erlbaum Associates.
- 24 Cohen, J. (1992). A power primer. *Psychological Bulletin*, *112*, 155-159.
- 25 Davis, K. G., & Kotowski, S. E. (2005). Preliminary evidence of the short-term  
26 effectiveness of alternative treatments of low back pain. *Technology and*  
27 *Health Care*, *13*, 453-462.
- 28 Dickx, N., Cagnie, B., Achten, E., Vandemaele, P., Parleviet, T., & Danneels, L.  
29 (2008). Changes in lumbar muscle activity because of induced muscle pain  
30 evaluated by muscle functional magnetic resonance imaging. *Spine*, *33*, E983-  
31 E989.
- 32 DiFabio, R. P. (1987). Reliability of computerized surface electromyography for  
33 determining the onset of muscle activity. *Physical Therapy*, *67*, 43-48.
- 34 Downs, S. H., & Black, N. (1998). The feasibility of creating a checklist for the  
35 assessment of the methodological quality both of randomised and non-  
36 randomised studies of health care interventions. *Journal of Epidemiology and*  
37 *Community Health*, *52*, 377-384.
- 38 Frymoyer, J. W. (1988). Back pain and sciatica. *The New England Journal of*  
39 *Medicine*, *318*, 291-300.
- 40 Geisser, M. E., Ranavaya, M., Haig, A. J., Roth, R. S., Zucker, R., Ambroz, C., &  
41 Caruso, M. (2005). A meta-analytic review of surface electromyography  
42 among persons with low back pain and normal, healthy controls. *The Journal*  
43 *of Pain*, *6*, 711-726.
- 44 Hodges, P. W., & Bui, B. H. (1996). A comparison of computer-based methods for  
45 the determination of onset of muscle contraction using electromyography.  
46 *Electroencephalography and clinical Neurophysiology*, *101*, 511-519.

- 1 Hodges, P. W., Moseley, G. L., Gabrielsson, A., & Gandevia, S. C. (2003).  
2 Experimental muscle pain changes feedforward postural responses of the trunk  
3 muscles. *Experimental Brain Research*, *151*, 262-271.
- 4 Hodges, P. W., & Moseley, L. G. (2003). Pain and motor control of the lumbopelvic  
5 region: Effect and possible mechanisms. *Journal of Electromyography and*  
6 *Kinesiology*, *13*, 361-370.
- 7 Hodges, P. W., & Richardson, C. A. (1996). Inefficient muscular stabilization of the  
8 lumbar spine associated with low back pain: A motor control evaluation of  
9 transversus abdominis. *Spine*, *21*, 2640-2650.
- 10 Hodges, P. W., & Richardson, C. A. (1997). Relationship between limb movement  
11 speed and associated contraction of the trunk muscles. *Ergonomics*, *40*, 1220-  
12 1230.
- 13 Hodges, P. W., & Richardson, C. A. (1999). Altered trunk muscle recruitment in  
14 people with low back pain with upper limb movement at different speeds.  
15 *Archives of Physical Medicine and Rehabilitation*, *80*, 1005-1012.
- 16 Holm, I., Friis, A., Brox, J. I., Gunderson, R., & Steen, H. (2000). Minimal influence  
17 of facet joint anesthesia on isokinetic muscle performance in patients with  
18 chronic degenerative low back disorders. *Spine*, *25*, 2091-2094.
- 19 Jarzem, P. F., Harvey, E. J., Arcaro, N., & Kaczorowski, J. (2005). Transcutaneous  
20 electrical nerve stimulation [TENS] for short-term treatment of low back pain-  
21 Randomised double blind crossover study of sham versus conventional TENS.  
22 *Journal of Musculoskeletal Pain*, *13*, 11-17.
- 23 Jette, A. M., Smith, K., Haley, S. M., & Davis, K. D. (1994). Physical therapy  
24 episodes of care for patients with low back pain. *Physical Therapy*, *74*, 101-  
25 115.
- 26 Keefe, F. J., & Hill, R. W. (1985). An objective approach to quantifying pain behavior  
27 and gait patterns in low back pain patients. *Pain*, *21*, 153-161.
- 28 Kiesel, K. B., Uhl, T., Underwood, F. B., & Nitz, A. J. (2008). Rehabilitative  
29 ultrasound measurement of select trunk muscle activation during induced pain.  
30 *Manual Therapy*, *13*, 132-138.
- 31 Lamoth, C. J. C., Stins, J., Pont, M., Kerckhoff, F., & Beek, P. J. (2008). Effects of  
32 attention on the control of locomotion in individuals with chronic low back  
33 pain. *Journal of NeuroEngineering and Rehabilitation*, *5*, 13.
- 34 Lamoth, C. J. C., Daffertshofer, A., Meijer, O. G., & Beek, P. J. (2006). How do  
35 persons with chronic low back pain speed up and slow down? Trunk and  
36 pelvis coordination and lumbar erector spinae activity during gait. *Gait and*  
37 *Posture*, *23*, 230-239.
- 38 Lamoth, C. J. C., Daffertshofer, A., Meijer, O. G., Moseley, G. L., Wuisman, P. I. J.  
39 M., & Beek, P. J. (2004). Effects of experimentally induced pain and fear of  
40 pain on trunk coordination and back muscle activity during walking. *Clinical*  
41 *Biomechanics*, *19*, 551-563.
- 42 Lee, C., Simmonds, M., Novy, D., & Jones, S. (2000). A comparison of self-report  
43 and clinician measured physical function among patients with low back pain.  
44 *Archives of Physical Medicine and Rehabilitation*, *82*, 227-231.
- 45 Lee, C., Simmonds, M. J., Etnyre, B. R., & Morris, G. S. (2007). Influence of pain  
46 distribution on gait characteristics in patients with low back pain. Part 1:  
47 Vertical ground reaction force. *Spine*, *22*, 1329-1336.
- 48 Lee, R. Y. W., Tsung, B., Tong, P., & Evans, J. (2005). Posteroanterior mobilisation  
49 reduces the bending stiffness and the pain response of the lumbar spine. In  
50 *International Conference on Movement Dysfunction* (pp. 39). Edinburgh, UK.

- 1 Lilius, G., Laasonen, E., Myllynen, P., Harilainen, A., & Gronlund, G. (1989).  
2 Lumbar facet joint syndrome. *Journal of Bone and Joint Surgery-British*, 71-  
3 B, 681-684.
- 4 Lund, J. P., Donga, R., Widmer, C. G., & Stohler, C. S. (1991). The pain-adaptation  
5 model: A discussion of the relationship between chronic musculoskeletal pain  
6 and motor activity. *Canadian Journal of Physiology and Pharmacology*, 69,  
7 683-694.
- 8 Marras, W. S., Davis, K. G., Ferguson, S. A., Lucas, B. R., & Gupta, P. (2001). Spine  
9 loading characteristics of patients with low back pain compared with  
10 asymptomatic individuals. *Spine*, 26, 2566-2574.
- 11 Marras, W. S., Ferguson, S. A., Gupta, P., Bose, S., Parnianpour, M., Kim, J.-Y., &  
12 Crowell, R. R. (1999). The quantification of low back disorder using motion  
13 measures. Methodology and validation. *Spine*, 24, 2091-2100.
- 14 Marras, W. S., Lewis, K. E. K., Ferguson, S. A., & Parnianpour, M. (2000).  
15 Impairment magnification during dynamic trunk motion. *Spine*, 25, 587-595.
- 16 Marras, W. S., Parnianpour, M., Ferguson, S. A., Kim, J.-Y., Crowell, R. R., Bose, S.,  
17 & Simon, S. R. (1995). The classification of anatomic- and symptom- based  
18 low back disorders using motion measure models. *Spine*, 20, 2531-2546.
- 19 Marras, W. S., & Wongsam, P. E. (1986). Flexibility and velocity of the normal and  
20 impaired lumbar spine. *Archives of Physical Medicine and Rehabilitation*, 67,  
21 213-217.
- 22 Moe-Nilssen, R., Ljunggren, A. E., & Torebjork, E. (1999). Dynamic adjustments of  
23 walking behaviour dependent on noxious input in experimental low back  
24 pain. *Pain*, 83, 477-485.
- 25 Moseley, G. L., & Hodges, P. W. (2005). Are the changes in postural control  
26 associated with low back pain caused by pain interference? *Clinical Journal of*  
27 *Pain*, 21, 323-329.
- 28 Moseley, G. L., Nicholas, M., & Hodges, P. W. (2004). Pain differs from non-painful  
29 attention-demanding or stressful tasks in its effect on postural control patterns  
30 of trunk muscles. *Experimental Brain Research*, 156, 64-71.
- 31 Nadler, S. F., Steiner, D. J., Erasala, G. N., Hengehold, D. A., Abeln, S. B., &  
32 Weingand, K. W. (2003a). Continuous low-level heatwrap therapy for treating  
33 acute nonspecific low back pain. *Archives of Physical Medicine and*  
34 *Rehabilitation*, 84, 329-334.
- 35 Nadler, S. F., Steiner, D. J., Erasala, G. N., Hengehold, D. A., Hinkle, R. T., Goodale,  
36 M. B., Abeln, S. B., & Weingand, K. W. (2002). Continuous low-level heat  
37 wrap therapy provides more efficacy than ibuprofen and acetaminophen for  
38 acute low back pain. *Spine*, 27, 1012-1017.
- 39 Nadler, S. F., Steiner, D. J., Petty, S. R., Erasala, G. N., Hengehold, D. A., &  
40 Weingand, K. W. (2003b). Overnight use of continuous low-level heatwrap  
41 therapy for relief of low back pain. *Archives of Physical Medicine and*  
42 *Rehabilitation*, 84, 335-342.
- 43 Norman, R., Wells, R., Neumann, P., Frank, J., Shannon, H., Kerr, M., & Group, O.  
44 U. B. P. S. (1998). A comparison of peak vs cumulative physical work  
45 exposure risk factors for the reporting of low back pain in the automotive  
46 industry. *Clinical Biomechanics*, 13, 561-573.
- 47 Novy, D. M., Simmonds, M. J., & Lee, C. E. (2002). Physical performance tasks:  
48 What are the underlying constructs? *Archives of Physical Medicine and*  
49 *Rehabilitation*, 83, 44-47.

- 1 Novy, D. M., Simmonds, M. J., Olson, S. L., Lee, C. E., & Jones, S. C. (1999).  
2 Physical performance: Differences in men and women with and without low  
3 back pain. *Archives of Physical Medicine and Rehabilitation*, *80*, 195-198.
- 4 O'Sullivan, P. B., Dankaerts, W., Burnett, A. F., Farrell, G. T., Jefford, E., Naylor, C.  
5 S., & O'Sullivan, K. J. (2006). Effect of different upright sitting postures on  
6 spinal curvature and trunk muscle activation in a pain-free population. *Spine*,  
7 *31*, E707-E712.
- 8 O'Sullivan, P. B., Grahamslaw, K. M., Kendell, M., Lapenskie, S. C., Moller, N. E., &  
9 Richards, K. V. (2002). The effect of different standing and sitting postures on  
10 trunk muscle activity in a pain-free population. *Spine*, *27*, 1238-1244.
- 11 Rashiq, S., Koller, M., Haykowsky, M., & Jamieson, K. (2003). The effect of opioid  
12 analgesia on exercise test performance in chronic low back pain. *Pain*, *106*,  
13 119-125.
- 14 Shum, G. L. K., Crosbie, J., & Lee, R. Y. W. (2005a). Effect of low back pain on the  
15 kinematics and joint coordination of the lumbar spine and hip during sit-to-  
16 stand and stand-to-sit. *Spine*, *30*, 1998-2004.
- 17 Shum, G. L. K., Crosbie, J., & Lee, R. Y. W. (2005b). Symptomatic and  
18 asymptomatic movement coordination of the lumbar spine and hip during an  
19 everyday activity. *Spine*, *30*, E697-E702.
- 20 Shum, G. L. K., Crosbie, J., & Lee, R. Y. W. (2007). Movement coordination of the  
21 lumbar spine and hip during a picking up activity in low back pain subjects.  
22 *European Spine Journal*, *16*, 749-758.
- 23 Simmonds, M., & Rebelo, V. (2003). Self-selected speed of movement during a  
24 repeated sit-to-stand task in individuals with and without LBP. *Fourth*  
25 *congress of european federation of the international association for the study*  
26 *of pain chapters, Prague, Czech Republic, September 2-6*.
- 27 Simmonds, M. J. (2006). Measuring and managing pain and performance. *Manual*  
28 *Therapy*, *11*, 175-179.
- 29 Spencer, J. D., Hayes, K. C., & Alexander, I. J. (1984). Knee joint effusion and  
30 quadriceps reflex inhibition in man. *Archives of Physical Medicine and*  
31 *Rehabilitation*, *65*, 171-177.
- 32 Stevenson, J. M., Weber, C. L., Smith, J. T., Dumas, G. A., & Albert, W. J. (2001). A  
33 longitudinal study of the development of low back pain in an industrial  
34 population. *Spine*, *26*, 1370-1377.
- 35 Stokes, M., Rankin, G., & Newham, D. J. (2005). Ultrasound imaging of lumbar  
36 multifidus muscle: Normal reference ranges for measurements and practical  
37 guidance on the technique. *Manual Therapy*, *10*, 116-126.
- 38 Thomas, J. S., & France, C. R. (2007). Pain-related fear is associated with avoidance  
39 of spinal motion during recovery from low back pain. *Spine*, *32*, E460-E466.
- 40 Thomas, J. S., France, C. R., Sha, D., & Vander Wiele, N. (2008). The influence of  
41 pain-related fear on peak muscle activity and force generation during maximal  
42 isometric trunk exertions. *Spine*, *33*, E342-E348.
- 43 Travell, J., Rinzler, S., & Herman, M. (1942). Pain and disability of the shoulder and  
44 arm. *Journal of the American Medical Association*, *120*, 417-422.
- 45 van Dieën, J., Selen, L. P. J., & Cholewicki, J. (2003). Trunk muscle activation in  
46 low-back pain patients, an analysis of the literature. *Journal of*  
47 *Electromyography and Kinesiology*, *13*, 333-351.
- 48 van Nieuwenhuysse, A., Fatkhutdinova, L., Verbeke, G., Pirenne, D., Johannik, K.,  
49 Somville, P., Mairiaux, P., Moens, G., & Masschelein, R. (2004). Risk factors



- 1 for first-ever low back pain among workers in their first employment.  
2 *Occupational Medicine*, 54, 513-519.
- 3 Waters, T. R., Putz-Anderson, V., Garg, A., & Fine, L. J. (1993). Revised NIOSH  
4 equation for the design and evaluation of manual lifting tasks. *Ergonomics*, 36,  
5 749-776.
- 6 Watson, P. J., Booker, C. K., Main, C. J., & Chen, A. C. (1997). Surface  
7 electromyography in the identification of chronic low back pain patients: The  
8 development of the flexion relaxation ratio. *Clinical Biomechanics*, 12, 165-  
9 171.
- 10 Wong, T. K. T., & Lee, R. Y. W. (2004). Effects of low back pain on the relationship  
11 between the movements of the lumbar spine and hip. *Human Movement*  
12 *Science*, 23, 21-34.
- 13 Zedka, M., Prochazka, A., Knight, B., Gillard, D., & Gauthier, M. (1999). Voluntary  
14 and reflex control of human back muscles during induced pain. *Journal of*  
15 *Physiology*, 520, 591-604.  
16

ACCEPTED MANUSCRIPT

**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<br>Age: 23-30<br><br>No LBP<br><br>Treadmill walk<br>4.0km/h for<br>1 minute. | 0.5ml 5% saline.<br><br>Depth: 15mm.<br><br>Location: 40-45mm lateral to L3 (right). | Gait phases.<br>EMG<br>Amplitude.<br>Right/Left Ratio.<br>Peak activity<br>at double stance phase (peak <sub>50</sub> ).<br>Mean EMG in contra-ipsilateral swing phases.<br>Surface EMG placement:<br>Lateral to Th 12, L2, L2 lat, L4 bilaterally. | Gait phases not affect by pain.<br>EMG amplitude increased 8.5%.<br>No change in ratio.<br>Peak <sub>50</sub> decreased 7.3%.<br>Mean EMG during contra- and ipsilateral swing increased 15.1% and 19.2% respectively.<br>No correlation between EMG and VAS (R=0.53). | Mean VAS 5.4±2.3.<br>EMG ipsilateral to pain at L2 showed most significant changes.                           | 1.2* (Mean EMG)<br>0.81* (Peak50)<br>1.04* (Contralateral swing)<br>1.03* (Ipsilateral swing) | 3, 5, 8, 11, 12, 14, 15, 21-25   |
| Zedka et al., (1999)          | 4 males and 1 female.<br>Age: 20-55<br><br>Flexion (constrained lower limbs).         | 5% Saline.<br><br>Depth: 40mm.<br><br>Location: 30mm lateral to L3 (right).          | Trunk displacement.<br>Velocity of motion.<br>EMG amplitude.<br>Surface EMG placement:<br>Lateral to L3   | Reduced trunk displacement<br>velocity.<br>EMG amplitude during flexion unchanged.<br>Loss of FRR bilaterally.<br>EMG amplitude decreased during   | Mean VAS 5.3±0.8.<br>During guided painful movement<br>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.<br>Age: 18-25<br><br>No LBP.<br><br>Varied speed walking.      | 0.5ml 5% saline.<br><br>Depth: 30mm.<br><br>Location: 65mm lateral to L3 (right).           | Relative phase coupling.<br><br>EMG amplitude and patterns.<br><br>Surface EMG placement: 30mm lateral to Th12, L2, L4 bilaterally | No effect on phase coupling.<br><br>Elevated right EMG during ipsilateral swing for all locations and for L2 during contralateral swing phase.<br><br>Elevated left EMG amplitude for ipsilateral swing phase. | Mean VAS 6.1±1.9.<br><br>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.<br>Age: 20-49<br><br>Walking at five self adjusted speeds.    | 1ml 6% saline<br><br>Depth: 15.2mm<br><br>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.<br><br>Not for vertical axes.   | Mean VAS 6.1±1.49.<br><br>15 of 20 showed correlation R <sup>2</sup> 0.36-0.89 between pain and gait changes.            | 0.75* (AP accel)<br><br>0.27* (Vertical accel)<br><br>0.75* (Mediolateral accel) | 5, 8, 9, 12, 14, 15, 22-25 |
| Moseley et al., (2004)     | 5 male and 3 female.<br>Age: 32±7<br><br>No LBP.<br><br>Shoulder flexion (standing) | 1-1.5ml 5% saline.<br><br>Depth: 30mm.<br><br>Location: 50mm lateral to L4.                 | Temporal and Spatial parameters of EMG (related to reaction times).<br><br>Intramuscular EMG: deep LM, TrAb,                       | Delay in onset of TrAb in painful condition.<br><br>Control reaction time = 154±15ms.<br><br>Pain = 189±19ms.  | Mean VAS ?6.2 (taken from graph).<br><br>Superficial injection of lidocaine included.<br><br>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.<br>Age: 28.6±3.6<br>No LBP.<br>Shoulder flexion (standing) | 1.5ml 5% saline.<br>Depth: 35mm.<br>Location: 60mm lateral to L4. | Temporal and Spatial parameters of EMG (related to reaction times).<br>Intramuscular EMG: TrAb, OE, OI, deep LM, sup LM. | Onset of TrAb- actual difference: Control= -18.9±2.4 (ms).<br>Isotonic Saline = -13.7±4.0 (ms).<br>Hypertonic Saline = -8.6±4.1 (ms).<br>Onset of deepLM- actual difference: Control=47.3±5.5 (ms).<br>Hypertonic Saline = 24.4±5.5 (ms). | Mean VAS 6.2±1.0.<br>Superficial injection of lidocaine included.                      | 3.31 (emg onset TrAb).<br>4.49 (emg onset deep LM).       | 5, 8, 10-12, 14, 22-25    |
| Moseley and Hodges, (2005) | 7 male and 9 female.<br>Age: 24±5<br>No LBP.<br>Shoulder flexion (sitting)      | Noxious cutaneous electric stimulation bilaterally over the PSIS. | Temporal and Spatial parameters of EMG (related to reaction times).<br>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.<br>Resolution of pre-pain on removal of noxious stimulus. | NA  | 5, 7, 8, 10-12, 14, 22-25 |
| Kiesel et al., (2008)      | 6 male.<br>Age: 26±7.3<br>No LBP.<br>Abdominal 'drawing-in' (crook lying);      | 1.5ml 5% saline.<br>Depth: 35mm.<br>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)<br>0.53 – 0.63 (us thickness LM) | 5, 8-12, 14, 22-26        |

|                      |  |   |                        |  |   |  |                             |
|----------------------|--|---|------------------------|--|---|--|-----------------------------|
|                      | prone limb raise.                                      | L4.   |                        |  |   |  |                             |
| Dickx et al., (2008) | 15 male<br>Age: 23.33±0.8<br>No LBP.<br>Sorenson test. | 1.5ml 5% saline.<br>Depth: 25mm.<br>Location: 40mm lateral to L4. | Muscle functional MRI. | Decreased muscle activity in LM and erector spinae during painful exercise.<br>Results show difficulty activating in the presence of pain. | 5.3-5.9 VAS during exercise.<br>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, 12<sup>th</sup> 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.<br>Age: 54, range 23-78.<br>CLBP duration: 10-420 months.<br>Mixed diagnosis.<br>Sorenson test. | IV fentanyl 1µg/kg.<br>Significant pain relief reported (0.9 on VRS) | Sorenson test time | Saline injection<br>Sorenson score = 60±42s<br>Fentanyl injection<br>Sorenson score = 77±49s | Minimal pain relief.<br>Large variation in performance. | 0.38        | 5, 8, 12, 25                     |

|                       |   |  |   |   |  |             |                               |
|-----------------------|---|--|---|---|--|-------------|-------------------------------|
| Holm et al., (2000)   | 38 male and 49 female.<br>Age: 48, range 22-79.<br>CLBP duration: 12.3 yrs.<br>Mixed diagnosis.<br>Isokinetic Dynamometer for trunk flexion/extension at 60°/s and 120° /s. | Bilateral ZA joint injection L5/S1 ± L4/5.<br>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.<br>Age: 44, range 19-64.<br>CLBP >3/12.<br>ROM and observed scored disability battery (blinded scorer).  | Intra-articular steroid injection to ZA joints.<br>Pericapsular steroid injection.<br>Intra-articular saline injection.<br>Significant pain relief across groups (mean: 18.3mm). | VAS (pain) Disability score.<br>ROM.    | Significant improvements in likert scale for disability.<br>Improvements in flexion and rotation ROM. | ROM measured with tape measure.<br>Rotation measured with a compass.<br>Questionable clinically significant result.<br>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).<br>ROM                      | Statistically significant   | Real numbers not reported.   | NA          | 5, 7, 8, 11, 12, 21, 22,      |

|                             |   |   |   |   |   |    |                            |
|-----------------------------|---|---|---|---|---|----|----------------------------|
|                             | Age: 38.9±14.5.<br>CLBP >3/12 (no leg pain).<br><br>Physical battery.   | reduction in pain).   | (gravity goniometer).<br>Isometric lifting capacity.<br>Maximum repetitions of physical test battery. | gain in ROM.<br>15% gain in isometric lift capacity post 'real' TENS.<br>Statistically significant increase in maximum repetitions for all tasks  |   |    | 24, 25                     |
| Davis and Kotowski, (2005)  | 3 male and 3 female.<br>Age: 42.7±13.8.<br>4 male and 2 female.<br>Age: 37.0±4.7.<br>6 female.<br>Age: 44.3±8.5.<br>2 male and 4 female.<br>44.0±21.1.  | Acupuncture<br><br>Chiropractic<br><br>Massage<br><br>Physical Therapy                                | Adapted RMDQ and NASS<br>LSOAI.<br>Functional capacity evaluation (LMM).                              | <b>Acupuncture</b><br>↑ lat vel; lat acc; Tw vel.<br>↓ Tw acc.<br><b>Chiropractic</b><br>↑ lat vel; lat acc; Tw vel; Tw acc<br><b>Massage</b><br>↑ lat vel; lat acc; Tw vel; Tw acc.<br><b>PT</b><br>↑ 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.<br>Age: 44.4±6.4.<br>LBP (undefined acute or chronic).<br>Matched controls.<br>Repeated sit to stand (5) at 3 self varied speeds. | Superficial heat wrap to both groups – 40mins.<br><br>Significant pain relief (t=3.2, p=0.006, actual | VAS (pain)<br>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). |  |  |  |  |  |
|--|--|------------------------|--|--|--|--|--|

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