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Review

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James Steele, Stewart Bruce-Low, Dave Smith

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Abstract

Objective: 'Disuse' and 'Deconditioning' in relation to low back pain (LBP) are terms often used interchangeably. Discussions of 'disuse' refer to general physical inactivity, which evidence suggests does not differ between symptomatic and asymptomatic persons. 'Deconditioning' refers to a decrease in function, commonly both cardiovascular/aerobic fitness and muscular strength/endurance again noting little difference. However, examination of decreased function relating specifically to lumbar extensor musculature deconditioning has yet to be examined corroborating all possible methods. Thus, this review attempts reappraise the deconditioning hypothesis in LBP specifically considering lumbar extensor deconditioning.

Methods: A literature review was conducted examining both cross sectional and prospective data on specific lumbar extensor deconditioning and LBP. A narrative approach and 'snowballing' style literature search was used involving initial use of PubMed and Google Scholar databases searching up to December 2012. Included where studies utilising the following three research methods allowing specific induction of the role of such deconditioning; 1) strength/endurance testing of the isolated lumbar extensor musculature, 2) imaging and histochemical examination of the lumbar extensor musculature, and 3) fatigue testing of the lumbar extensor musculature using electromyography.

Results/Findings: Despite issues interpreting individual studies due to methods, the majority of evidence suggests LBP is associated with decreased strength/endurance, atrophy, and excessive fatigability of the lumbar extensors. Prospective studies also suggest lumbar extensor deconditioning may be a common risk factor predicting acute low back injury and LBP.

Conclusions: The hypothesis of specific lumbar extensor deconditioning as being a causal factor in LBP is presently well supported. It is by no means the only causative factor and further research should more rigorously test this hypothesis addressing the methodological issues highlighted regarding previous studies. However, its role suggests specific exercise may be a worthwhile preventative and rehabilitative approach.

REVIEW

A Reappraisal of the Deconditioning Hypothesis in Low Back Pain: Review of Evidence from a Triumvirate of Research Methods on Specific Lumbar Extensor Deconditioning

James Steele[a], Stewart Bruce-Low[a], Dave Smith[b]

[a]Southampton Solent University, Southampton, Hampshire UK

[b]Manchester Metropolitan University, Crewe, Cheshire

Address for correspondence: Centre for Health, Exercise and Sport Science, Southampton Solent University, East Park Terrace, Southampton, Hampshire SO14 0YN UK. Tel: +442380 319606; Fax: +442380 332627;

Key words: strength; endurance; atrophy; CSA; muscle fibres; EMG; fatigue

[Short title: The deconditioning hypothesis in low back pain]

ABSTRACT

Objective: 'Disuse' and 'Deconditioning' in relation to low back pain (LBP) are terms often used interchangeably. Discussions of 'disuse' refer to general physical inactivity, which evidence suggests does not differ between symptomatic and asymptomatic persons. 'Deconditioning' refers to a decrease in function, commonly both cardiovascular/aerobic fitness and muscular strength/endurance again noting little difference. However, examination of decreased function relating specifically to lumbar extensor musculature deconditioning has yet to be examined corroborating all possible methods. Thus, this review attempts reappraise the deconditioning hypothesis in LBP specifically considering lumbar extensor deconditioning.

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Introduction

Defining the Disuse/Deconditioning Hypothesis - 'Disuse' OR 'Deconditioning'?

The 'Disuse Syndrome' was originally described by Bortz II[1] and more recently has been reviewed by Verbunt et al.[2,3]. The rationale behind Disuse Syndrome is that pain causes low levels of physical activity (i.e. avoidance behaviour or guarded movement[4]) which contribute to deconditioning and chronicity in low back pain (LBP), and cause the further interrelated physical and psychological changes shown in figure 1. In essence, it proposes that injury and pain precede deconditioning and potentially many of LBP's symptoms, leading to a 'vicious cycle.'

Verbunt et al.[2] however, have suggested that the hypothesis that 'disuse' (i.e. defined as a *decrease in physical activity levels*) is a cause of LBP may be incorrect. They highlight that activity levels are in fact similar between symptomatic and asymptomatic participants, suggesting that lack of physical activity due to the presence of pain or injury may not

contribute to the presence of deconditioning or LBP[5]. Indeed a more recent study has also highlighted that physical activity levels appear to not change as a result of LBP, even as it develops from acute into chronic LBP (CLBP)[6]. This suggests that symptomatic CLBP participants may not suffer from development of disuse after the initial incidence of LBP. It seems possible, therefore, that the direction of temporal relationships in the 'Disuse Syndrome' model may be unfitting in its usual presentation (figure 1). This is not to suggest that deconditioning as a result of existing pain and its related behaviours is not a possibility, indeed the presence of injury has been shown to affect muscular function and could therefore instigate deconditioning itself, or at the least further enhance its development[7,8].

Instead, 'deconditioning' (i.e. defined as a *decrease in function*) may be first implicated as a potential cause of low back injury and pain, as opposed to LBP leading to 'disuse' and then to 'deconditioning'. Indeed Verbunt et al.[2] attempt to distinguish between 'disuse' and 'deconditioning'; however in both their definitions they inevitably invoke general physical inactivity ('disuse') as being responsible for 'deconditioning' and that this inactivity is the result of pain. Here we instead differentiate between 'disuse' and 'deconditioning' and pose that the disuse syndrome model does not consider what first causes or increases the probability of injury or pain occurring (which *may* lead to further 'disuse') in the first place.

In addition, the disuse model appears to imply that freak injury may account for the majority of LBP[7], yet due to the widespread prevalence of LBP it seems unlikely that freak injury could account for the majority of cases. Bigos et al.[9] demonstrate exactly why this is a concern, reporting that accidents such as slips or falls, despite resulting in higher

cost injuries, are very uncommon with regard to cause of injury; lifting or materials handling, however, was most commonly considered a cause. Dysfunction due to deconditioning could potentially affect such actions leading to fatigue and altered joint biomechanics, subsequently causing injuries and instigating mechanisms by which pain results. Then, at this point, the cycle by which a reduction in activity levels further promote chronicity, and the changes associated with it, may begin to have an influence.

The model may therefore require an addition that considers the initial injury in the first place (figure 2). A high percentage of low back injury and acute LBP develops into CLBP[10,11] and so it seems logical that something must affect the risk of low back injury in the first instance. Indeed Adams and colleagues[12] have recently commented on the pertinent fact that all *'chronic back pain always starts as acute back pain.'* Thus, logically, something must first be responsible for the initiation of acute pain. The remainder of the existing model is likely correct in describing the process of developing chronicity after initial injury has occurred. What specific factor is most important in determining whether that initial injury and acute pain occurs in order for it to become chronic, however, is the more interesting question.

Many prior reviews on the topic of deconditioning in LBP have utilised a broad focus encompassing decrease in function of both cardiovascular/aerobic fitness as well as muscular strength/endurance[2,3,13,14]. These reviews suggest that deconditioning of these kinds is not apparent in those with CLBP. However, the studies considered have utilised varied methods of examining this association, many of which are not entirely specific and these are explained throughout this article. The lack of consideration of the specific study methodologies used by previous authors has perhaps contributed to the vague distinctions

between ‘disuse’ and ‘deconditioning’ as well as the shift in focus from physical risk factors towards a more cognitive based appraisal of LBP and the effects of rehabilitation[15]. A more specific analysis of the literature, focusing upon specifically located deconditioning and in consideration of the methodological limitations of prior techniques, might therefore be found to yield contrasting conclusions regarding the presence of deconditioning in LBP and the models relationships.

Specific Disuse/Deconditioning of the Lumbar Extensors

The important role of the lumbar extensor musculature, the Erector Spinae (ES; i.e. iliocostalis lumborum and longissimus thoracis) and both deep and superficial lumbar Multifidus (MF), in providing stability to the lumbar spine, has been alluded to in numerous studies[16-24]. Prior reviews of the literature have indicated that, although it is difficult to distinguish which muscles provide the greatest relative contribution to spinal stability, their importance in co-operatively contributing to lumbar spinal stability is clear[23,25]. The relative contribution of individual muscles will vary depending on the specific task being performed[26]; however, MacDonald et al.[23] explain that all lumbar extensors can contribute towards stability of the intervertebral segments through compression of the vertebral unit and increased joint stiffness. Clearly the lumbar extensor musculature playing such an important role in providing stability to the lumbar spine suggests that deconditioning and dysfunction in these muscles could lead to changes in stability and biomechanics. This change in biomechanics may result in increasing passive tissue stresses and potentially impart an injury or pain response in structures of the lumbar spine[27].

Aim and Approach of this Review

The focus upon 'disuse' and 'deconditioning' in a general sense has led to much incongruity in drawing specific conclusions regarding LBP. In light of the potential role of the lumbar extensors in controlling stability and, in dysfunction, altering biomechanics which might lead to injury and pain causing mechanisms/symptoms, there is certainly potential for specifically located deconditioning to relate to LBP. The aim of this review therefore is to test this hypothesis by examining the evidence reporting the nature of the relationship between specific deconditioning of the lumbar extensors and LBP from a triumvirate of research methods including:

- Strength/endurance testing of lumbar extensor musculature,
- Imaging and histochemical examination of the lumbar extensor musculature
- Fatigue testing of the lumbar extensor musculature using electromyography

Three sections will follow, each covering the three research methods highlighted as they have been used in cross sectional examination of symptomatic CLBP participants compared with asymptomatic healthy participants. A fourth section shall examine prospective studies that have sought to examine the effect of deconditioning using these methods upon development of LBP in asymptomatic participants. Throughout, any methodological concerns and considerations with studies shall be highlighted initially and noted whilst discussing such studies. In light of those methodological issues discussed in each section it will also be noted which types of studies were excluded from consideration (however, those excluded are still summarised within the full summary tables in the appendices). Given the broad scope of this review a narrative approach utilising a 'snowballing' style literature search[28] was used initially involving PubMed and Google

Scholar databases searching up to December 2012 utilising search terms including combinations and synonyms of 'low back pain' low back injury' 'lumbar' 'back' 'spine' 'extensors' 'lumbar extension' trunk extension' 'erector spinae' 'multifidus' 'iliocostalis lumborum' 'longissimus thoracis' 'strength' 'endurance' 'atrophy' 'cross sectional area' 'fat infiltration' 'muscle density' 'histochemistry' 'fibre type' 'electromyography' 'fatigability' etc. In addition previous reviews and any located articles reference lists were searched. This was selected as the best way to locate, examine and synthesise the maximum amount of information in the various sections covered, thus initial inclusion criteria were based upon applicability to this particular area of discussion, and whether studies had utilised the above noted research methods, before applying specific exclusion criteria (noted in each section of this review).

Strength and Endurance of the Lumbar Extensor Musculature in LBP

Considerations for Studies of Strength and Endurance of the Lumbar Extensor Musculature

An initial consideration when looking at studies of muscular performance should be that of the false duality between definitions of muscular strength and endurance expressed by many authors especially within the field of exercise and LBP[29,30]. Muscular endurance can be defined as being *absolute* (i.e. the number of repetitions/time performed at a given resistance), or *relative* (i.e. the number of repetitions/time performed at a given percentage of a 1 repetition maximum [1RM] or other maximum strength measurement)[31-33]. For example, a pre training 1RM of 100kg might produce 10 repetitions at an absolute value of 70kg, which is also the relative value of 70%1RM. However, after a training intervention where the 1RM has improved to 120kg, a participant will almost certainly be capable of greater than 10 repetitions at the absolute value of 70kg, but likely still only produce a

maximum of 10 repetitions at the relative value of 70% 1RM (now 84kg). This example shows an increase in maximal strength (1RM) leading to an increase in absolute muscular endurance (i.e. an increase in number of repetitions at the fixed submaximal weight). Research supports this concept[34]. However, research does not support the idea that the same is true of relative loads, but rather that similar maximal repetitions are possible[34,35]. In practice, with relevance to the deconditioning hypothesis and for the LBP participant, low strength would translate to low absolute endurance, high strength to high absolute endurance and vice versa. Therefore, presuming average external loads typically experienced (i.e. in working conditions etc.) might remain constant, an increase in strength would theoretically mean an increase in endurance at those absolute loads experienced. Thus it would seem logically erroneous to attempt to draw a distinction between the two and to claim that one is more important than the other with regards to LBP[29]. Indeed Mannion[7] has commented that the hypothesis of fatigability as being associated with LBP is essentially analogous to the hypothesis of insufficient strength (both being manifestations of lumbar extensor deconditioning relevant to the deconditioning hypothesis).

Numerous studies have sought to identify the relationship between functional measures of strength and endurance of the lumbar extensor muscles. However, it should be highlighted that the validity of a number of methods of tests for strength and/or endurance of the lumbar spine are questionable due to methodological difficulties; a primary concern being whether sufficient pelvic restraints have been utilised. Essentially, tests have either been performed to examine *trunk* extension (TEX), OR, isolated *lumbar* extension (ILEX; testing utilising pelvic stabilisation through use of a semi seated position with rear pelvic restraint and a belt across the thighs). In considering lumbar extensor deconditioning TEX

studies require careful reflection along with corroboration of more valid test measures i.e. ILEX. If the pelvis is not stabilised during testing of extension then it is impossible to determine the actual source of measured extension torque during tests of strength and may involve the hip extensors[36-42] contributing to overstate torque measures[43-45], due to the longer moment arms over which the gluteus and hamstrings exert force, and their relatively larger cross-sectional areas[46]. At most only 3^{o} of pelvic rotation[47], likely a result of soft tissue compliance, occurs during ILEX testing of this kind. Lack of pelvic restraint perhaps partly explains the inconsistent reproducibility of TEX endurance tests[48-52] as compared with the consistency of ILEX strength and endurance testing[53-56]. Indeed, despite the aforementioned relationship between strength and absolute endurance there is poor relationship between tests of ILEX strength and TEX endurance[57]. This highlights that the tests may utilise different musculature.

Although tests of ILEX are more valid representations of lumbar extensor function due to TEX being a compound movement requiring additional rotation of the pelvis through the hip extensor musculature[36,37,41-47,58-60], a large number of studies have made use of tests measuring TEX. Smidt et al.[43] also explain that consideration of both tests of TEX and ILEX are indeed valuable when interpreted together as they allow both a deductive, and further an inductive, approach to identify the so called 'weak link' within the kinetic chain and thus we initially considered both studies in this review. Beimborn & Morrissey[61] reviewed early literature on trunk muscle performance in LBP suggesting a consistent association with reduced TEX strength in symptomatic participants, as well as further studies. These TEX studies have been summarised in the appendices provided and appear to show inconsistent associations; some results supporting a link between TEX strength/endurance and LBP[37,48,62-82], some which do not[66,69,71,72,81,83-90].

The inconsistency of both TEX tests of strength and endurance should not be surprising as hip extensor deconditioning appears to not be associated with CLBP[91] and as explained, without appropriate restraint of the pelvis the musculature of the hip extensors will serve to confound results. However, despite hip extensor deconditioning having apparently little association with LBP it seems some other aspect of TEX, perhaps ILEX, may be associated with it. As TEX is composed of both hip and lumbar extension it therefore seems logical that tests should attempt to remove the involvement of the hip extensors to examine ILEX. Thus, of key importance and inclusion to this review are studies that have used appropriate methods of testing ILEX. As shall also be noted, previous surgery may have implications for the results of studies examining the deconditioning hypothesis[92-95] and ideally participants with prior surgery should be excluded. However, only one study utilising ILEX has controlled for this factor yet may suffer from its own shortcoming of small sample size. Thus in the following section all ILEX studies have been examined with this limitation noted.

Isolated Lumbar Extension Studies

The validity of the extension test used is of great importance in examining the association between strength, endurance and LBP, therefore studies that have considered this are potentially more useful in answering the question of whether specific lumbar extension deconditioning is associated with LBP. Unfortunately in comparison with studies of TEX, studies of ILEX are relatively scarce. However, studies utilising testing that appropriately restrains the pelvis consistently report significantly reduced ILEX strength in symptomatic CLBP participants compared with asymptomatic controls[96-98]. Other studies[99-102] have

further reported reduced strength results from symptomatic participants compared to normal values obtained from healthy asymptomatic controls in other research[53].

There is, however, only one study by Lariviere et al.[40] of ILEX using valid restraints that does not support the link between specific deconditioning and CLBP. Lariviere et al.[40] reported no difference between asymptomatic and symptomatic CLBP participants in strength reported as maximum torque, or endurance reported as repetitions performed at a load equal to 60% maximum voluntary contraction (MVC). They commented however in discussion that the small sample size (n=18) used may have meant a lack of the typical multifactorial heterogeneity in their non-specific CLBP group, potentially impacting the generalisation to CLBP of this aspect of their research. It may also have resulted in a type II statistical error (i.e. failure to reject the null hypothesis). Other larger studies that have supported the link between ILEX weakness and CLBP have used in some instances upwards of 100 symptomatic participants and demonstrate reduced strength compared to healthy norms [99,100]. In addition and in particular, the study by Nelson et al.[99] of 895 CLBP participants suggested that a range of diagnoses existed in their sample (Patients' diagnoses included non-specific CLBP, degenerative disc/arthritic disease, lumbar disc syndrome or spondylolisthesis/spondylolysis) and thus was likely quite representative of the typical heterogeneity of CLBP populations. Age, stature and body mass were also similar between groups in the study by Lariviere et al.[40], however this was also reportedly the case for a number of other studies supporting the association[96-98] and so is unlikely to explain the difference in results.

One limitation of studies supporting the link between reduced ILEX strength and LBP is that these studies either did not report whether they excluded[97,99-102], or chose not to

exclude[96,98], participants who had undergone previous surgery. Lariviere et al.[40] did exclude those having undergone previous lumbar surgery and thus this may explain the different results found by these investigators. As has been noted, previous surgery can have potentially deleterious consequences to the lumbar extensor musculature anatomy[92-95] and so might be thought to interfere with ILEX strength in symptomatic participants. Although a number of TEX studies have excluded those with previous surgery, with some supporting[37,74,77-82] and some refuting[88,89] an association between deconditioning and LBP, we must consider the inherent limitations of this approach already highlighted when specifically concerned with the lumbar extensors. There is certainly potential for further research to clarify whether differences in ILEX strength do indeed exist independent of previous lumbar surgery.

A final concern is the lack of statistical comparison with healthy controls groups in some studies[99-102]. Though these results are consistent with those that have conducted statistical comparisons[96-98] this is a weakness and again something to be ensured in future research.

It is noted that only one study reported upon tests of isolated lumbar extension endurance[40]. However due to the inherent relationship between strength and endurance it seems logical that the reported reduced lumbar extension strength in CLBP would be indicative of a reduced endurance also. The limitations discussed above also apply to this aspect of the study however, and there is further scope for research specifically examining this.

Summary of Strength and Endurance Studies of the Lumbar Extensor Musculature

Of the studies examined, those employing sufficient pelvic restraints as their means of assessing lumbar extension have consistently reported results that lend support to the association of specific lumbar extensor deconditioning with CLBP[96-102] with only one exception[40]. It seems clear that when valid testing of ILEX is used, most evidence suggests a link between specific lumbar extension deconditioning and CLBP. However, it is unclear from purely this area of research whether this may in fact be due to the presence of previous surgery. Studies controlling for this factor utilising a larger sample size should be conducted to further test this. Table 1 summarises the findings of these studies.

If it is the case that deconditioning exists independent of previous surgery, a number of possible explanations may exist for the apparent association between ILEX deconditioning and CLBP: pain, anticipation of pain or pain avoidance behaviours, interfering with trunk muscle function; lack of motivation in asymptomatic participants; even deliberate malingering in some cases. Studies as early as those of McNeill et al.[62] suggested that the reduced TEX strengths seen in symptomatic compared with asymptomatic participants are most likely explained by the participants' avoidance of either large tensions in the posterior soft tissue or large compressive force on the lumbar motion segments. This conclusion would seem reasonable being that there was an absence of studies of the lumbar extensor musculature in CLBP showing *in vivo* the condition of the lumbar musculature at the time of the study by McNeill et al.[62], to corroborate with the empirical findings on function. Indeed, strength is a product of both muscular force and the moment arm about which it acts, but the measurement of strength is significantly affected by volitional exertion. The concerns of McNeill et al.[62] were well justified in the absence of evidence specifically implicating muscular deconditioning *in vivo*. Evidence that has subsequently examined this, however, provides important information regarding the

presence of specific lumbar deconditioning of the lumbar extensors in LBP. As such the next section shall detail and discuss this evidence.

Imaging and Histochemical Studies of the Lumbar Extensor Musculature in LBP

Considerations for Imaging and Histochemical Studies of the Lumbar Extensor Musculature

As suggested, the data on reduced ILEX function should be further corroborated with studies specifically examining the lumbar extensor musculatures condition *in vivo*. Documentation of their roles in support and stability of the lumbar spine has motivated a large body of research examining their anatomical and histochemical condition in relation to LBP. Broadly, these studies can be divided into those that have examined the gross anatomy of the lumbar musculature (using imaging study i.e. magnetic resonance imaging [MRI] or computed tomography [CT]) and those that have examined the histochemical nature (through use of muscle biopsy), or 'microanatomy' of the lumbar musculature.

Here we will review both, yet whilst doing so consider the many factors that may affect and limit the conclusions that can be drawn from these studies. Surgery via posterior approach can result in alteration of the lumbar musculature[92] which can be lasting[93,94]. However there is evidence that only gross surgery, such as that for disc herniation, has this effect and that laminectomy and nucleotomy does not impart this damage to the musculature[95]. This is an important factor when considering the population examined. In some cases participants undergoing acute surgery have been examined and this presents an issue with determining whether deconditioning was present before surgery or is merely a result of surgery; indeed both may be the case[92].

Another issue that is involved in studies that have drawn bilateral (i.e. left and right) comparisons for evidence of asymmetry, or multiple vertebral level comparisons, is lack of asymptomatic controls. If deconditioning is present more on one side than the other, or more confined to a particular vertebral level, it is often considered that atrophy is local to symptoms[103]. However without an asymptomatic group to compare it is impossible to say whether the asymptomatic side of symptomatic participants is normal or indeed atrophied itself, though to a lesser degree than the symptomatic side.

Additionally age significantly impacts upon muscle degeneration[104-106]. Research that has compared symptomatic participants to age-matched asymptomatic controls is more valuable in determining the association of deconditioning with LBP. Other considerations include the validity of semi-quantitative analysis of images[107] and the value of measuring cross sectional area (CSA) as compared to muscle density or fatty infiltration in imaging studies[74].

An issue with many of the studies examining so called 'normal' or 'asymptomatic' muscle histochemistry is that they have utilised biopsy samples from autopsy[108] or from acute disc herniation patients undergoing surgery[109,110]. This is justified by the assumption that short duration of spinal dysfunction would have little impact upon muscle condition[110], and early suggestions are that surgical procedure has little impact upon muscular condition due to asymmetric differences being unrelated to the side of herniation[109]. Due to the possible association between deconditioning and the initiation of LBP, these disc herniation surgery studies may perhaps be more indicative of the typical muscle condition that predisposes LBP development if it is known that the biopsies were taken before surgery began; however this is often not reported. It is important to remember that gross surgical

procedures such as those for disc herniation have themselves been shown to have an impact upon the musculature[95].

As a result of these concerns our discussion focuses upon studies that have appropriately controlled for these factors (i.e. exclusion of previous surgery, control of age between groups). In addition, consideration of the potential for the presence of either deconditioning confined to a particular side or vertebral level will be compared to the potential for a general deconditioning. As with the TEX studies, those studies that have not considered such methodological factors as highlighted in this section have been summarised in the appendices provided and appear to show inconsistent associations both for imaging[75,76,94,101,103,106,111-119] and histochemical studies[92-94,106,109,120-126].

Imaging Studies of the Lumbar Musculature

Firstly, the imaging studies that have examined the gross anatomy of the lumbar musculature will be reviewed. As noted there are numerous studies on this topic that have not controlled for the potentially confounding factors of age and previous lumbar surgery[75,76,94,101,103,106,111-119]. Of those examined for this review a handful of studies[74,91,107,127,128] did control for these factors and the results of them are summarised here (note that though some studies have examined the psoas also, this review focuses upon the lumbar extensors).

Several studies have examined the CSA of either the paraspinal muscles as a whole group[91,127], the ES muscle group[74,127], and in one the quadratus lumborum (QL; which can initiate lumbar extension when bilaterally contracted)[91]. Kamaz et al.[91] examined absolute total paraspinal muscle CSA and found significant reduction in CLBP participants at the

lower level of L4 but not at the upper. There was however a significant reduction in CSA of the QL at the upper level. Danneels et al.[127] found no difference in normalised ES CSA between symptomatic or asymptomatic CLBP participants. In addition, however, they also examined total paraspinal muscle CSA and did report significantly reduced CSA in CLBP participants. Comparing these results is problematic as Kamaz et al.[91] did not normalise their values. Though Danneels et al.[127] did not find a reduction in CSA of the ES, they did in the MF and attributed the reduction in total paraspinal CSA to the reduction in the CSA of MF.

Another study by Hultman et al.[74] comparing participants with intermittent LBP, CLBP and healthy age matched controls found no difference between groups for ES CSA. They did however find a significant reduction in ES density in the CLBP group. This study potentially brings the value of CSA as a sole measure of deconditioning or atrophy into question and may explain some of the disparity in results of other studies. Indeed as in other physiological measures the absolute measurements of a particular variable (i.e. CSA, in a similar vein to mitochondrial volume[129]; or capillary density[130,131] that have been discussed elsewhere[132]) are often less valid than relative measures of density and the same might apply to muscle CSA and muscle density. Muscle density may therefore be more representative of muscle atrophy as changes such as fatty infiltration may serve to maintain absolute CSA but would indicate that muscle density had indeed reduced. Yet in light of this Mengiardi et al.[107] when comparing age matched controls found no difference in ES fat percentage. Studies examining muscle density/fat content are expanded upon below.

CSA of the MF has also been examined by two studies, both of which support a link between reduced MF CSA and CLBP[91,127]. Both Kamaz et al.[91], and Danneels et al.[127] demonstrated that MF CSA was significantly reduced compared with healthy age matched controls. Kamaz et al.[91] found these results consistent at both the upper and lower level of L4 and Danneels et al.[127] at just the lower L4 level.

When the ES has been examined for differences in muscle density or fat content between asymptomatic and symptomatic participants there have been contrasting findings. As noted, although they did not find any evidence of reduced CSA, Hultman et al.[74] noted significant reduction in muscle density of the ES in CLBP participants. Danneels et al.[127] however also found no difference in ES muscle CSA without fat between their age matched groups and so they suggested that fatty infiltration may be more closely associated with age than indicative of atrophy[104-106]. Mengiardi et al.[107] when comparing age matched controls, also found no difference in longissimus fat percentage using both a quantitative and semi-quantitative analysis.

In considering the MF Danneels et al.[127] also reported no difference in muscle CSA without fat for the MF, despite an overall reduction in MF CSA. In contrast, however, Mengiardi et al.[107] did find a significant difference in fat percentage of the MF when considering the results of their quantitative analysis. The results of their semi-quantitative analysis however found no difference and it seems reasonable to suggest that this lack of difference may stem from observer error.

Moderate differences between studies may perhaps be explained by the level of measurement. It has certainly been suggested and evidenced by some that atrophy of the

ES or MF may be dependent upon level and side of symptoms due to denervation atrophy[103,115]. However other evidence has suggested that a general atrophy at all levels and both sides may exist in CLBP participants compared with controls and that asymmetry is merely more pronounced in those with specific symptoms of radiculopathy[117]. Although age was controlled in one study between groups[103] and participants with previous surgery were excluded from the other two[115,117] the lack of control of one or the other between these studies renders difficulty in concluding whether deconditioning and atrophy is level or side specific or whether it is indeed more general. This is certainly an area requiring further research, the results of which may have important implications for prevention and treatment through use of exercise particularly considering the different approaches used to address these i.e. resistance exercise or motor control training.

Although not comparing asymptomatic and symptomatic participants, one other study is worthy of mentioning which did control for the influence of age and previous surgery. Kang et al.[128] examined CSA, both absolute and normalised to disc CSA, and used semi-quantitative analysis of fat content of the ES and MF in a group of CLBP participants as controls and a group of CLBP participants with degenerative kyphosis preparing to undergo corrective surgery. Although they were not able to compare their groups to healthy asymptomatic controls they did note that reduced CSA, both absolute and normalised, and more severe fat content, were found in the kyphosis group compared with the controls, and regression analysis showed MF atrophy to be most strongly associated. These results are interesting considering the influence of the musculature upon spinal stability and certainly present an area of future research to examine whether the general atrophy often seen in CLBP is more severe in light of structural dysfunctions, and whether this is causative or instead a result of these more severe conditions.

Though disparate perhaps due to methodological issues, those studies reviewed (having excluded previous surgery and controlling for age) all suggest some form of deconditioning and atrophy, either reduced CSA, reduced density or fatty infiltrations, being present in both the ES, and the MF in of CLBP participants compared with asymptomatic controls[74,91,107,127]. Considering that both play important roles in lumbar spine stability[23] this is potentially evidence for a plausible role of deconditioning in LBP.

Histochemical Studies of the Lumbar Musculature

Imaging studies offer valuable insight into the gross anatomical condition of the musculature. Histochemical studies on the other hand are able to provide further detail considering individual fibre size, density and differentiation between differing fibre types, as well as identifying specific pathological changes such as presence of small angulated fibres, target/core targetoid fibres, and fibre type grouping or group atrophy[7]. Mannion[7] has reviewed and highlighted a number of important findings from this area. She concludes by highlighting the difficulty of distinguishing cause and effect of fibre type characteristics (i.e. whether the observed characteristics existed prior to onset of LBP, or were a consequence of the presence of LBP). Her review also discussed fibre characteristics in relation to electromyographic (EMG) analysis of the lumbar spine musculature and this will be discussed further in the following section. Here we will further consider the findings reported by Mannion[7] along with more recent findings. Again, studies have considered both the ES[77,94,106,109,120,121,133] and MF (both deep and superficial)[92,93,109,122-126].

When it has been made clear in studies that biopsies were taken before surgery then the direction of the association between deconditioning and LBP might be better identified. However, biopsies are frequently taken during surgery or this is often not clarified[93,94,109,120-123,125,126]. Where it is not specified it is instead prudent to assume that biopsy was taken during the operation meaning we need to treat the results from these studies with caution. One study has shown that pathological changes are present before surgery although further denervation is apparently caused by surgery as shown in biopsies taken afterward[92] which certainly suggests that deconditioning may be present before surgery is initiated and thus associated with conditions for which surgery is recommended.

Studies of the histochemical condition of the paraspinal muscles in symptomatic CLBP participants that have not undergone surgery suggest the presence of fibre atrophy, pathological changes, and fibre type ratio alteration [106,124]. Neither of these cited studies, however, included asymptomatic controls. Zhao et al.[124] conducted bilateral comparisons and suggested that different findings between sides were affected by location of herniation, and that differences existed among those with central, bilateral and unilateral pain. Prospective evidence has suggested that herniation can cause change in muscle activity, which might cause denervation atrophy[134]. Again however the absence of an asymptomatic control group renders the same difficulty as in other studies[103,115] when drawing conclusions (i.e. it is not known if the side without herniation was atrophied also).

Only one study has been conducted in the absence of the potential confounding influence of surgery, has controlled for the confounding effects of age, and also included a matched asymptomatic control group[77]. Crossman et al.[77] reported no difference in fibre size or fibre ratios between participant groups and that both had a predominance of type I fibres.

This is in contrast to Mannion's[7] earlier conclusions that symptomatic participants have a higher proportion of type IIX fibres. However Crossman et al.[77] did not note the specific location of their biopsy sample and thus it is not clear whether these results refer to the ES, MF or the paraspinal musculature as a whole.

Summary of Imaging and Histochemical Studies of the Lumbar Extensor Musculature

Although evidence suggests that deconditioning is indeed present in some form in symptomatic participants there is considerable disparity in methodologies in both imaging and histochemical studies. Data from imaging studies appear more consistent in their findings of some form of atrophy[74,91,107,127] as opposed to those from histochemical studies; however only one histochemical study has controlled for previous surgery and age[77]. Indeed although general deconditioning may be present in LBP, the findings of Crossman et al.[77] suggest that dominance of an adverse fibre type is perhaps not. Table 2 summarises these studies.

Crossman et al.[77] also suggest that the differences in functional tests between asymptomatic and symptomatic participants' strength/endurance discussed in the previous section may therefore be due to the influences of psychological disturbance or motivation. However, we should consider that it is not only fibre type distribution that influences fatigue resistance but also capillary density, enzymatic activities and associated metabolic processes[7,132]. So it is unsurprising that there is not a distinct relationship between this single variable and its associated end effect. Mannion[7] highlights that, because functional tests (i.e. strength/endurance) can be confounded by psychological disturbance, EMG should be employed to circumvent this and record more objective indices of muscle activation and fatigue. Indeed this measure might be considered to account for the many

factors influencing fatigue due to its ability to accurately predict it[135] and that it also has a close association with physiological indicators of fatigue[136-138]. Cooper et al.[139] have shown that greater EMG amplitude increases occur during a test to fatigue in symptomatic participants (both surgical and non-surgical, suggesting a similarity underlying the two groups) compared with asymptomatic participants and suggested that it indicated an increased central drive secondary to muscle wasting or denervation. Thus EMG and other activation studies therefore may provide further insight into the deconditioning hypothesis and LBP.

Evidence suggests reduced strength/endurance in symptomatic participants which is further corroborated with *in vivo* evidence of muscular deconditioning being present. Further, and in consideration of the aforementioned concerns with participant effort in functional tests, the following section will complete the triumvirate of areas covered in examining deconditioning of the lumbar extensor musculature in LBP by reviewing studies that have employed EMG to assess fatigability.

Electromyography Studies of Fatigue in the Lumbar Extensor Musculature in LBP

Considerations for Electromyographic Fatigue Analysis of the Lumbar Extensor Musculature

In consideration of the effect that deconditioning, and thus fatigability, may have on LBP, EMG has been used to attempt to control for influence of psychological disturbance or participant motivation on functional measures of endurance[7]. Thus the information presented by these studies is also useful in examining the deconditioning hypothesis by corroborating evidence from the prior two sections which may support a link between lumbar extensor deconditioning and LBP. EMG can be used to interpret muscle activation

and muscle force[140] but can also be used to more objectively demonstrate fatigability[7,135]. EMG is limited in many regards by such confounding factors as crosstalk (readings from synergist muscles), depth of active motor units from surface electrode, amplitude related to motor units and muscle fibre-types, variable firing rates, muscle-fibre length, velocity and contraction type[141-146]. Cross talk is of particular issue when differentiating specific lumbar extensor musculature[147]. However, in considering power spectrum analysis of rate of change in EMG spectral variables[148] (i.e. root mean square amplitude, mean, median or mode frequency slopes, initial frequencies etc.) for determining fatigability these might perhaps not be so confounding an issue as they would presumably remain constant systematic errors while such EMG parameters would change with fatigue.

When looking at LBP populations we should consider that the MVC-normalised EMG signal amplitude measured may perhaps be influenced by insincere effort[149,150]. Roy et al.[151] however, have shown that EMG measures of fatigability provide accurate classification independently of MVC, suggesting their greater objective power in discriminating between symptomatic and asymptomatic groups compared to simply measuring relative activation levels.

Although it seems EMG measures of fatigability are more valid, a point must be considered when interpreting their results; that is, whether participants performed exercise to momentary muscular failure (MMF; i.e. maximal intensity of effort)[32,33,152]. These studies should not surprisingly show a difference in fatigue indices from start to end of exercise performance within all groups participating in testing, but presumably would show no difference in between-group comparisons as both will be maximally fatigued. Change in fatigue indices over a fixed number of repetitions or time (i.e. as an *absolute*

measure) would instead be the most appropriate means of detecting fatigue-related differences between symptomatic and asymptomatic groups, and, considering the issue with normalising to MVC in LBP participants, should preferably utilise an absolute load. In some studies the absolute load utilised has been the participant's torso mass during TEX. Thus an important consideration for between-group comparisons of fatigue during TEX is whether body or torso mass was similar.

Geisser et al.[153] have conducted a meta-analysis of the use of trunk surface EMG comparing asymptomatic and symptomatic participants and comment that EMG recordings from non-maximal tasks are likely to be more reliable than those involving maximal exertions. However, we should remember that both absolute and relative amplitude levels will be subject to the aforementioned limitations of EMG including an insincere effort, whereas fatigue may not be. EMG measures of fatigability should objectively quantify fatigue independently of MVC[151] where a significant change in fatigue is unlikely to be seen if insincere effort is put forth. Geisser et al.[153] reported an effect size of zero for EMG measures of fatigability during isometric trunk exertions, suggesting no difference between symptomatic and asymptomatic participants. However, a difficulty lies in interpreting these results partly due to the methodological differences of studies included but also because Geisser et al.[153] do not clarify EMG locations and whether extensor or flexor musculature, or a combination of the both, was being examined. Nor do they comment in more detail on the intensity of effort of the activity (i.e. whether it was performed to MMF or to an absolute time/number of repetitions). Their meta-analysis included 7 studies[37,78,87,98,135,154,155] examining EMG measures of fatigability in LBP; however, a number of studies also examining fatigue indices that were present at the time of its publication were not included[151,156-159]. Being that EMG measures

of fatigability are more valid and applicable to our present discussion of the deconditioning hypothesis we will further examine the studies analysed by Geisser et al.[153] along with those not included in their analysis, as well as further studies that have been conducted more recently[40,79-81,88].

For sake of clarity in this review, although numerous methods of analysing the EMG signal for determination of fatigability exist between studies, here these methods are collectively referred to as EMG 'fatigue indices', as a critical comparison of the specific methods of analysis is beyond the scope of this review[160]. Due to the difficulty of cross talk between the paraspinal musculature when using surface EMG[147], we do not attempt to differentiate between, for example, the ES or MF, and instead consider the studies reviewed to offer information regarding the lumbar extensor musculature as a whole. Being that, as previously noted, surgery can have considerable confounding effects upon the lumbar extensor musculature[92-95], we have focused in this section upon those studies which have controlled for this[77,88,135,154]. Again as with previous sections those studies excluded from discussion (in this case those not controlling for surgery or those which have had participants perform exercise to MMF[37,40,78-81,87,98,151,155-158]) have been summarised within the appendices.

Fatigability Studies of the Lumbar Musculature

The studies reviewed utilising measurements of EMG fatigue indices have examined differences between asymptomatic and symptomatic participants using different methods. Some have used both discriminant analysis and regression to identify whether such measures can successfully classify participants, and others have drawn simpler between group comparisons.

Roy and colleagues have performed several studies examining fatigue indices in LBP, one of which controlled for both factors noted[135]. They examined fatigue indices during 60 second standing isometric TEX contractions at 40%, 60% and 80% MVC. Discriminant analysis correctly classified between asymptomatic controls and symptomatic CLBP participants at 40% MVC (92% controls, 82% CLBP) and 80% MVC (84% controls, 91% CLBP), however results were less favourable at 60% MVC (67% controls, 75% CLBP; a later study by Peach & McGill[155] clarifies this anomaly though it should be noted they do not note whether those with previous surgery were excluded). This study also looked at two level analysis (Lumbar level and %MVC level) finding that fatigue indices at L5 for 80% MVC showed the most favourable classification (75% controls, 75% CLBP).

In an early study by Mayer et al.[154] participants performed an isometric TEX hold using a roman chair in the same manner as the Biering-Sorensen test. Participants performed a series of 10 holds for 15 seconds each with a rest period of 10 seconds between holds. Between group comparisons of fatigue indices for both the first 5 holds, as well as the full 10, demonstrated significantly greater fatigue in the symptomatic CLBP group than in the asymptomatic controls before completion of an intensive rehabilitation program. After the program the difference between groups was reduced and still significant for the 10 holds, yet there was no significant difference when data for 5 trials were compared.

Humphrey et al.[79] considered a range of fatigue indices calculated from power spectrum analyses during a back lift test. They reported significant differences in fatigue indices between CLBP participants and controls. In addition they reported that logistic regression showed high sensitivity and specificity in classifying CLBP participants. However,

although the variables considered could discriminate between symptomatic CLBP participants and asymptomatic controls there were varying degrees of accuracy. They noted that those variables that could potentially be affected by load (peak amplitude, median frequency) were less accurate as predictors; however, load independent variables (such as initial median frequency and half width) offered a higher degree of accuracy. Humphrey et al.[79] also included a group of participants with a past history of LBP. No variables were able to discriminate these from either the CLBP participants or the controls though this was suggested to be due to the comparatively small sample size for this group (healthy controls n = 175; CLBP participants n = 145; past history participants n = 30).

A later study by Da Silva et al.[88] however, offers contrasting results. They found no significant difference between asymptomatic controls or symptomatic CLBP participants in fatigue indices between groups for 60 second contractions at 50% MVC for the Biering-Sorenson test, a standing extension test, and also a semi-crouching back lift test. It is unclear as to the reason for this contrasting finding; however, Da Silva et al.[88] suggest that the CLBP group studied may not have been sufficiently impaired to demonstrate a difference based upon the low results from the Oswestry disability questionnaire (~12%).

Another study by Crossman et al.[77] has also reported no difference in fatigue indices between healthy asymptomatic and symptomatic CLBP participants during standing TEX using 60%MVC for 60 seconds and also during the Biering-Sorenson test. Crossman et al.[77] comment that this may perhaps not be surprising due to the lack of differences in histochemical analysis of fibre typing in their participants. However they also note concerns regarding the loads used by CLBP during the 60%MVC TEX test in particular, speculating that these participants may not have given a sincere MVC and thus they may

have been using <60%MVC during this test. As noted earlier MVCs have been evidenced to be affected by this[149,150] and as a result we noted that the use of an absolute load may be of greater validity in determining differences in fatigability. Crossman et al.[77] did also have participants perform the Biering-Sorenson TEX test, also reporting similar body mass between groups, which suggest that the absolute loading between groups for this test was similar. However this test was performed to MMF and so it is again unsurprising that no differences in fatigue were found. Yet, CLBP participants did demonstrate significantly lower endurance times and so assuming they did perform the test to MMF (and that also healthy controls did) the lower endurance time might indicate greater fatigability. However it again must be noted that this is a test of TEX and so the endurance time is not specifically indicative of the lumbar extensors.

Unfortunately, considering the potentially confounding effect of relative load being influenced by insincere MVCs in CLBP participants, we are left with only the results of Mayer et al.[154] which do suggest greater fatigability in CLBP participants. It is difficult to discern whether any other factors may have affected the results between studies apparently supporting the presence of deconditioning through EMG fatigue indices[79,135,154] and those suggesting it is not present[77,88]. All have used a range of electrode placement sites (T10/L1/L2/L3/L4/L5), many in different combinations, a range of tests (standing TEX, prone TEX, back lift test), both relative and absolute loads as noted, and also a range of test timings (30 seconds, 60 seconds and 10 repetitions of 15 seconds); as such it is unclear as to what effect these variables may have upon the study's findings.

Summary of Electromyography Studies of Fatigue of the Lumbar Extensor Musculature

In summary, of the studies reviewed, it appears that objective measures of fatigability show contrasting results. Those controlling for previous surgery and using standardised timed protocols show some evidence in support[79,135,154] and some against[77,88] the presence of deconditioning. One study that has also controlled for the potentially influencing factor of sincere effort by CLBP participants does, however, suggest the presence of some degree of deconditioning[154]. Table 3 summarises these studies.

Thus far it has been evidenced that there may be an association between measures of, and variables associated with, lumbar extensor deconditioning (i.e. reduced strength/endurance testing of lumbar musculature, deconditioning shown by imaging and histochemical examination of the lumbar musculature, and increased fatigability of the lumbar musculature shown by EMG fatigue indices) and LBP, hence providing support towards the deconditioning hypothesis. Theoretically, muscular deconditioning could lead to instability and altered joint biomechanics and thus result in injuries (either single macro-trauma or cumulative micro-trauma) which may instigate pain causing mechanisms. Therefore we might expect that the presence of such deconditioning, whilst being a consistent association with CLBP, might also predict the development of LBP or incidence of low back injury in initially asymptomatic individuals also. Prospective studies have examined whether this is in fact the case and the following section will discuss the evidence implicating deconditioning's effect upon injury and development of LBP.

Prospective Studies of Lumbar Extensor Deconditioning in LBP

A concern with cross-sectional studies is that causation cannot be logically determined from association (it should also be noted that a lack of association does not necessarily

imply a cause and effect relationship does not exist). Despite a consistent association being one of the criteria for causation as determined by Austin Bradford Hill[161], and the consistency of some degree of deconditioning with CLBP, in addition to biological plausibility of which there is evidence implicating deconditioning, it still cannot solely be taken as evidence for a causative relationship, nor the direction of that causation. Prospective studies provide clearer indication for a temporal relationship between variables and allow us to consider whether the potential plausibility for deconditioning to actually lead to LBP can be evidenced.

Although in previous sections we have been selective over those studies discussed based upon methodological considerations highlighted, this section considers a more liberal range of literature. The reason for this is due to the relative paucity of prospective studies that have appropriately controlled for the factors previously highlighted in this review. Thus the studies reviewed in this section should be considered tentatively and it is noted that further research is required to definitively test the deconditioning hypothesis and the presence of a prospective relationship between deconditioning and LBP.

Prospective Evidence from Strength & Endurance in LBP

Biering-Sorensen et al.[68] found that weak TEX was a predictive residual sign of recurrent LBP or CLBP over a 1 year follow up, although did not significantly predict first time occurrence**.** A study by Leino et al.[69] indicated that there was little prognostic value of tests of dynamic TEX in predicting LBP over a 10 year period but suggested instead an effect of the latter upon the former (i.e. symptoms, or degree of symptoms at baseline, had prognostic value in predicting reduced trunk muscle function at follow up). Disappointingly, however, Leino et al.[69] omitted dynamic TEX tests from their follow up

testing. Initial testing consisted of prone dynamic TEX efforts while follow-up data are reported for standing isometric TEX efforts. This presents difficulty in interpreting the effect of LBP presence at base-line affecting TEX muscle function at follow up as it is a case of comparing different tests to identify change[162]. This makes the conclusions questionable. The dynamic tests consisted of the number of repetitions performed over 30 seconds which might be considered more specifically a test of the ability to complete TEX movement quickly, not TEX strength/endurance *per se*. The isometric test on the other hand was indeed a TEX MVC and thus a measure of TEX strength. The data from Leino et al.[69] compare two entirely different tests with no clear conclusions being evident. It would seem that there was little difference in relative risk of LBP development when low and high performers in dynamic extension were followed up. Contrastingly Rissanen et al.[163], utilising the same dynamic test reported significant prediction of back disorder disability over an average 12 year follow-up. However, such dynamic TEX testing does not really offer an appropriate presentation of muscle function[164-166] and so interpreting the predictive results from this in light of the deconditioning hypothesis is questionable. It should also be pointed out that again that tests of TEX are not specifically indicative of lumbar extensor muscle function.

A study by Newton et al.[167] using a more consistent study design of dynamic isokinetic TEX testing (i.e. utilising the same test both at baseline and follow up) in prospective evaluation of LBP development, however, also suggested that it held no predictive value. Despite this, prospective implementation of the same battery of isokinetic tests as a pre-employment fitness evaluation in order to place workers in appropriate job areas has been demonstrated that it can significantly reduce injury rates[168]. This suggests a potential

connection between physical function and task demands in predicting injury and perhaps explains the lack of predictive value in these tests when this is not considered[167].

Batti'e et al.[169] reported that greater strength was actually a risk factor for report of back problems over a 4 year period. However, closer inspection of their results shows that this was only significant for arm and leg lift strength and that torso lift (TEX) was not significant. When their data were adjusted for age there were no significant correlations. Another prospective study has reported that a reduced trunk extension/flexion strength ratio is a significant risk factor for development of LBP over a 5 year period[170]. That extensor deconditioning may be more significant than flexor deconditioning in those with LBP has been highlighted in previous research[50,62,63,75,91,101,112,127] and it thus is interesting that a greater relative deconditioning of the extensors is shown to be predictive of future LBP. Kujala et al.[171] on the other hand suggested that neither isometric nor dynamic TEX performance were predictive of first time LBP in addition to strength ratio being unrelated in their sample group. However, their results did indicate a significant effect of musculoskeletal loading as well as reporting that taller participants (who may experience greater loading due to a greater external TEX moment) were more likely to develop LBP. Thus their results are somewhat supportive of the concepts conveyed by Reimer et al.[168], and also Chaffin[172] and Keyserling et al.[173] using the same test battery as Batti'e et al.[169], in that strength relative to physical demands is important. So although the population studied did not differ in their initial strength, those who engaged in heavier loading were weaker relative to their loading demands[171].

Associations between weak TEX and LBP have also been reported in younger populations[73,86,174] The studies of Salminen et al.[73,174] involved a 3 year follow up and

showed weak TEX associated with LBP at baseline and follow up. Despite this there was no predictive validity of TEX in development of LBP at follow-up. Studies have, however, also examined adolescents, showing prospective associations between TEX weakness and development of LBP[170,175].

TEX endurance has also been used in prospective studies. Poor TEX endurance has been identified as a risk factor for LBP incidence in some studies[68,83,175]. However, one study's findings indicate that it has no use in predicting future LBP[84]. Gibbons et al.[84] note however that the difference in results between theirs and previous studies may be due to type II error. Their sample size (n = 43) for follow up in incidence of LBP after initial testing was much lower than the sample used by Biering-Sorensen[68] (n = 982), and also the samples used by Luoto et al.[83] (n = 126) and Sjolie & Ljunggren[175] (n = 86) which might suggest that their data would be more likely to present a type II error (i.e. a failure to reject the null hypothesis) from a lack of statistical power through low sample size. Thus, the larger and more numerous studies do indicate the predictive potential of low TEX endurance in development of LBP.

Adams et al.[176] conducted a large prospective study examining physical factors; including TEX endurance and back lift test MVC and examined EMG fatigue indices over 20 seconds. Their results suggest back lift strength was not predictive of LBP, but TEX endurance time was. An earlier study by Mostardi et al.[177] that demonstrated no predictive value of strength also performed a back lift test. In spite of this, another larger study (n = 1652) has also employed the same back lift method amongst other fitness measures and found that there was significant predictive value between those with the lowest, middle

and best strength and fitness; the least fit sustaining the greatest proportion of low back injuries and the most fit sustaining the least[178].

Where many previous studies have used less valid measurements of lumbar function (i.e. TEX testing), another prospective study by Mooney et al.[179] examined low back injury rates and their relationship to ILEX strength. One hundred and fifty two shipyard workers were tested for ILEX strength and followed up for 2 years. In this period 9% (n = 14) reported low back injuries; however only 2 of these had below normal ILEX strength. These injury rates (9%), however, are considerably less than those reported for many other US industries[180]. The majority of the workers tested in the study by Mooney et al.[179] had normal ILEX strength. Thus the relatively low rates of injury for the participant sample as a whole actually suggest that normal strength may be protective and that the injuries that were sustained were potentially outliers. Indeed, of the injuries reported the highest incidence was within the heavy work categories and thus these injuries may have represented accidents during heavy work[9] whereby task demands exceeded physical function[168,171-173]; however, no further detail was reported on the nature of the sustained injuries.

Prospective Evidence from MRI & EMG in LBP

Although most prospective studies have examined the role of deconditioning from a perspective of functional tests of strength and endurance, there have been others that have examined imaging tests of the lumbar extensor musculature as well as EMG fatigue indices of the lumbar extensors. Gibbons et al.[84], using MRI, examined CSAs, proton density weighted signals and T2-weighted signal intensities of the ES, QL and psoas major prospectively yet found no significant predictive value from any of these variables.

Participants who suffered from LBP during the follow up period did show slightly higher signal intensities which might be indicative of greater fatty infiltration and thus muscular deconditioning. The fact that these were not found to be significant may be a result of a type II error again due to the sample size used (n=128). Although similar to the higher sample sizes in studies of TEX endurance, the authors are not aware of any other prospective imaging studies and so, unlike the endurance tests, this cannot be compared and confirmed.

Adams et al.[176] also utilised EMG fatigue indices in their study yet found that there was no predictive value over 3 years follow-up. Another study by Mannion et al.[159], however, reported prospective data for 200 young nurses who had never before suffered from serious LBP. EMG fatigue indices were recorded at baseline and followed up for 12 months. The result showed that greater fatigability significantly predicted development of first time LBP. Stevenson et al.[181] reported on a variety of variables included in a predictive model of LBP over a 2 year period, including EMG fatigue indices in the final model which were significantly predictive of LBP occurrence in the previous 6 months. Finally a study by Heydari et al.[182] has also examined EMG fatigue indices prospectively in 105 participants with no previous history of LBP. They also reported that greater fatigability was predictive of subjects self-rating of LBP at 2 year follow-up.

Summary of Prospective Studies

It seems that a number of prospective studies are suggestive of deconditioning as potentially etiological within development of LBP[68,84,170,174,175]. These studies have predominantly employed methods examining TEX strength, endurance and trunk extension/flexion ratios and so, as highlighted in the discussion of studies examining

strength and endurance, it must be considered that there are limitations to these methods. However, one study has prospectively examined ILEX, yet, due to the limitations of this study, and depending on perspective, its data could be interpreted either in support of or against lumbar extensor deconditioning as being causative in LBP development[179]. Evidence from other methods of examining deconditioning is contrasting. MRI shows no predictive value[84]; however, as discussed there is a lack of other imaging studies to compare this result to. In contrast, it appears that EMG fatigue indices may be predictive of LBP development[159,181,182]. Thus, though disparate there is certainly some prospective evidence to support the deconditioning hypothesis. Table 4 summarises these studies.

Discussion

It would appear that there certainly exists evidence indicative of some role specifically of lumbar extensor deconditioning in LBP, which may be causative, yet there is certainly scope for improving earlier studies with more appropriate examination of this relationship. The association of deconditioning specifically of the lumbar extensors in those with CLBP, and as a prospective risk factor for development of LBP, has been demonstrated in numerous studies and with various different methods. Studies conducting specific testing of ILEX evidence that weakness appears localised to the lumbar extensor musculature[96-102] as compared with the quite contrasting evidence utilising TEX. Imaging studies also demonstrate that deconditioning is consistently found in the ES, MF and QL of those with CLBP[74,91,107,127]. However whether this is level or side specific is unclear[103,115,117] and it appears that adverse muscle fibre composition is perhaps not present[77]. Finally, excessive fatigability of the lumbar extensors in symptomatic participants has been evidenced more objectively through use of EMG fatigue indices analysis[135,154,79]. Thus it seems that specific deconditioning of the lumbar extensor musculature may be a common factor in

LBP lending evidence towards the deconditioning hypothesis and to the speculation of other authors regarding its important role[60,183-186]. Further support is shown through prospective studies highlighting that deconditioning may be a risk factor for initial development of LBP[68,83,159,170,174-176,179,181,182].

However, it should be noted that although a body of research exists to support this hypothesis, there also exists some contrasting evidence to refute it which has been conducted with similarly rigorous methodology[40,77,84,88,176,179]. Indeed we have noted throughout this review the concerns with many of the methodologies employed in much previous research, even amongst the more carefully controlled studies. As such, though the hypothesis is by no means refuted, it still requires further rigorous testing that may be found to either further support or more definitely refute it.

For now however, we contend that the hypothesis presents a convincing explanation of LBP. The evidence reviewed herein is also supported by other areas of research considered as important to determining causality by the criteria put forth by Austin Bradford Hill[161]; criteria such as biological plausibility and experimental reversibility[187,188]. Evidence shows that specifically addressing lumbar extensor deconditioning through ILEX resistance exercise programs in CLBP provides significant reductions in pain and disability[60,97,99,189-200]. There is also evidence suggesting that improvements in ILEX strength correlate with reductions in pain and disability[99,200]. In addition there is evidence that prospectively addressing lumbar extensor deconditioning through ILEX resistance training reduces risk of further low back injury occurring[201-203]. Thus there is evidence for a relatively consistent prospective and cross sectional association, biological plausibility through biomechanical modelling studies of lumbar

spine stability, experimental reversibility, and also evidence for prospective strengthening to reduce injury risk. These factors combine to offer why deconditioning is perhaps a quite robust account of why LBP is such a wide ranging condition.

One issue that many authors have with this explanation of LBP however is very clearly summarised by Crossman et al.[77]. They note that, of the studies suggesting the presence of lumbar extensor deconditioning in LBP, *"in none of these studies were any mechanisms offered up to explain how "normal" paraspinal muscle could "dysfunction" to predispose to LBP."* Yet we suggest that the lumbar extensors as an isolated muscle group may exist in a potential state of specific chronic 'disuse,' and thus become 'deconditioned' in the first instance independent of physical activity levels due to their anatomy[60]. Indeed this specific state of disuse may stem from the lumbo-pelvic anatomy that is a consequence of our species' evolutionary history; in essence relatively weak lumbar extensors comparable to strong hip extensors[204]. This seems further apparent as most forms of activity and exercise appear to provide little to no conditioning effect[183,186,205-210]. Although, as noted in the introduction, 'disuse' is often considered as a general reduction in physical activity, it seems here that 'disuse' could instead be specifically considered as applicable to the lumbar extensors due to the difficulty in conditioning them, thus leading to their 'deconditioning'. In a sense, specific 'disuse' may lead to specific 'deconditioning' of the lumbar extensors, which may further lead to injury and LBP. But this is not simply a reduction in general activity levels; it is due to the inability to effectively maintain their condition as a consequence of their anatomy as the hip extensors appear to 'take-over' much of the load bearing[36,41,42] .

It should be made clear that it is not the intention of this review to argue for a singular cause of LBP. Although prospective evidence is suggestive of initial deconditioning being a risk factor for development of acute low back injury, LBP and various pain causing mechanisms, and that the majority of acute cases develop into CLBP, this is unlikely to be the only potential causative factor. Many other risk factors have indeed been reported. It is even possible that deconditioning is in fact a result of the impact of pain and other symptoms in some instances[7] and it is likely that both directions of causality could manifest. That being said, however, a body of evidence would appear to implicate specific lumbar extensor deconditioning in LBP, potentially as a primary factor predisposing injury (figure 2), and thus warrants an addition to the general conceptualisation of the 'Disuse/Deconditioning Syndrome.' This also strongly justifies an exercise based approach designed to effectively recondition the lumbar extensor musculature, regardless of the direction of causality.

That the deconditioning associated with LBP appears for the most part to be mainly localised to the lumbar extensors specifically also warrants that preferably a specific approach towards reconditioning be utilised. Both Helmhout et al.[211] and Mayer et al.[212] emphasise the issue with many reviews that consider 'exercise' as a single class of treatment without consideration to the variation in exercise approaches that have been applied. Many studies of exercise have also been criticised as lacking an adequate description of the precise exercises used[211,212]. Previous Cochrane reviews have not adequately described, defined and categorised the 'exercise' studies they have examined, potentially explaining the generally unfavourable conclusions drawn[213,214]. The Cochrane reviews have been specifically criticised for this flaw and their wide-sweeping conclusions[213-216]. Indeed we have also raised this issue of specificity[60,200,217,218]. As

noted, research has shown that the lumbar extensors are notoriously difficult to train unless the pelvis is appropriately restrained in order to provide ILEX[183,186,205-210,219]. That we have presented here that deconditioning may be specifically located in the lumbar extensors supports the contention that exercise approaches should specifically address this.

Conclusion

This review has provided a reconsideration of the importance of the deconditioning hypothesis as it relates to the development of, and association with, LBP. Deconditioning of specifically the lumbar extensors appears to be a consistent factor in LBP. However many of the studies reviewed herein have contained various methodological flaws and so such a conclusion should remain tentative. Future work should seek to further clarify this relationship by acknowledging these and aim to improve upon the previous research. In addition, the results of this review should perhaps be considered in the design of exercise-based rehabilitation approaches to LBP and also as preventative approaches.

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References

1. Bortz II WM. The disuse syndrome. West J Med 1984; 141(5): 691–694
2. Verbunt JA, Seelen HA, Vlaeyen JW, et al. Disuse and deconditioning in chronic low back pain: concepts and hypotheses on contributing mechanisms. Eur J Pain 2003; 7(1): 9–21
3. Verbunt JA, Smeets RJ, Wittink HM. Cause or effect? Deconditioning and chronic low back pain. Pain 2010; 149: 428-430
4. Main CJ, Watson PJ. Guarded movements: development of chronicity. J Musculoskelet Pain 1996; 4: 163–170
5. Verbunt JA, Westerterp KR, Van Der Heijden GJ, et al. Physical activity in daily life in patients with chronic low back pain. Arch Phys Med Rehabil 2001; 82: 726–730
6. Bousema EJ, Verbunt JA, Seelen HA, et al. Disuse and physical deconditioning in the first year after the onset of back pain. Pain 2007; 130(3): 279–286
7. Mannion AF. Fibre type characteristics and function of the human paraspinal muscles: normal values and changes in association with low back pain. J Electromyogr Kinesiol 1999; 9: 363–377
8. Panjabi MM. A hypothesis of chronic back pain: ligament subfailure injuries lead to muscle control dysfunction. Eur Spine J 2006; 15: 668–676
9. Bigos SJ, Spengler DM, Martin NA, et al. Back injuries in industry: A retrospective study II. Injury factors. Spine 1986; 11(3): 246–251

10. Papageorgiou AC, Croft PR, Thomas E, et al. Influence of previous pain experience on the episode incidence of low back pain: results from the South Manchester Back Pain study. Pain 1996; 66(2-3): 181–185
11. Croft PR, Macfarlane GJ, Papageorgiou AC, et al. Outcome of low back pain in general practice: a prospective study. BMJ 1998; 316(7141): 1356–1359
12. Adams MA, Stefanakis M, Dolan P. Healing of a painful intervertebral disc should not be confused with reversing disc degeneration: implications for physical therapies for discogenic back pain. Clin Biomech 2010; 25: 961–971
13. Wittink H, Michel TH, Wagner A, et al. Deconditioning in patients with chronic low back pain. Fact or fiction? Spine 2000; 25(17): 2221–2228
14. Smeets RJ, Wade D, Hidding A, et al. The association of physical deconditioning and chronic low back pain: A hypothesis oriented systematic review. Disabil Rehabil 2006; 28(11): 673–693
15. Steiger F, Wirth B, de Bruin ED, et al. Is a positive clinical outcome after exercise therapy for chronic non-specific low back pain contingent upon a corresponding improvement in targeted aspects(s) of performance? A systematic review. Eur Spine J 2011; 21(4): 575-598
16. Donisch EW, Basmajian JV. Electromyography of deep back muscles in man. Am J Anat 1972. 133(1), 25 – 36
17. Bogduk N, Twomey LT. Clinical Anatomy of the Lumbar Spine. Edinburgh: Churchill Livingstone, 1987
18. Panjabi M, Abumi K, Duranceau J, et al. Spinal stability and intersegmental muscle forces. A biomechanical model. Spine 1989; 14(2): 194–200

19. Crisco JJ, Panjabi MM. The intersegmental and multisegmental muscles of the lumbar spine. A biomechanical model comparing lateral stabilizing potential. Spine 1991; 16(7): 793–799
20. Cholewicki J, Panjabi MM, Khachtrayan A. Stabilizing function of trunk flexor-extensor muscles around a neutral spine posture. Spine 1997; 22(19): 2207– 2212
21. Solomonow M, Zhou BH, Harriz M, et al. The ligamento-muscular stabilizing system of the spine. Spine 1998; 23(23): 2552–2562
22. Moseley GL, Hodges PW, Gandevia SC. Deep and superficial fibers of the multifidus are differentially active during voluntary arm movements. Spine 2002; 27(2): E29–36
23. MacDonald DA, Moseley GL, Hodges PW. The lumbar multifidus: Does the evidence support clinical beliefs? Man Ther 2006; 11: 254–263
24. Rosatelli AL, Ravichandrian K, Agur AM. Three-dimensional study of the musculotendinous architecture of the lumbar multifidus and its functional implications. Clin Anat 2008; 21: 539–546
25. Cholewicki J, Van Vliet JJ. Relative contribution of trunk muscles to the stability of the lumbar spine during isometric exertions. Clin Biomech 2002; 17(2): 99–105
26. Ladin Z, Murphy KR, DeLuca CJ. Mechanical recruitment of low back muscles. Theoretical predictions and experimental validation. Spine 1989; 14(9): 927–938
27. Farfan HF, Gracovetsky S. The nature of instability. Spine 1984; 9(7): 714–719
28. Greenhalgh T, Peacock R. Effectiveness and efficiency of search methods in systematic reviews of complex evidence: audit of primary sources. BMJ 2005; 331: 1064–1065
29. McGill SM. Low back disorders: evidence-based rehabilitation and prevention. 2nd ed. Human Kinetics: Champaign, 2007
30. Norris CM. Back Stability: Integrating science and therapy. Champaign: Human Kinetics, 2008

31. Stone WJ, Coulter SP. Strength/endurance effects from three resistance training protocols with women. J Strength Cond Res 1994; 8: 231–4

32. Carpinelli R, Otto RM, Winett RA. A critical analysis of the ACSM position stand on resistance training: insufficient evidence to suort recommended training protocols. J Exer Physiol 2004; 7: 1–60

33. Fisher J, Steele J, Bruce-Low S, et al. Evidence based resistance training recommendations. Med Sport 2011; 15(3): 147–162

34. Hickson RC, Hidaka K, Foster C. Skeletal muscle fibre-type, resistance training, and strength-related performance. Med Sci Sport Exer 1994; 26: 593-598.

35. Mazzetti SA, Kraemer WJ, Volek JS, et al. The Influence of direct supervision of resistance training on strength performance. Med Sci Sports Exerc 2000; 32: 1175-84.

36. Kankaanpaa M, Laaksonen D, Taimela S, et al. Age, sex, and body mass index as determinants of back and hip extensor fatigue in the isometric Sorenson back endurance test. Arch Phys Med Rehabil 1998; 79(9): 1069–1075

37. Kankaanpaa M, Taimela S, Laaksonen D, et al. Back and hip extensor fatigability in chronic low back pain patients and controls. Arch Phys Med Rehabil 1998; 79(4): 412–417

38. San Juan JG, Yaggie JA, Levy S, et al. Effects of pelvic stabilisation on lumbar muscle activity during dynamic exercise. J Strength Cond Res 2005; 19(4): 77–81

39. Da Silva RA, Lariviere C, Arsenault AB, et al. Pelvic Stabilization and Semisitting Position Increase the Specificity of Back Exercises. Med Sci Sports Exer 2009; 41(2): 435-443

40. Lariviere C, da Silva RA, Arsenault AB, et al. Specificity of a back muscle exercise machine in healthy and low back pain subjects. Med Sci Sports Exer 2010; 42(3): 592–599

41. Clark BC, Manini TM, Mayer JM, et al. Electromyographic activity of the lumbar and hip extensors during dynamic trunk extension exercise. Arch Phys Med Rehabil 2002; 83: 1547–1552
42. Clark BC, Manini TM, Ploutz-Snyder LL. Derecruitment of the lumbar musculature with fatiguing trunk extension exercise. Spine 2003; 28(3): 282–287
43. Smidt G, Herring T, Amundsen L, et al. Assessment of abdominal and back extensor function. Spine 1983; 8(2): 211–219
44. Petersen CM, Amundsen LR, Schendel ML. Comparison of the effectiveness of two pelvic stabilization systems on pelvic movement during maximal isometric trunk extension and flexion muscle contractions. Phys Ther 1987; 67(4): 534–539
45. Graves JE, Fix CK, Pollock ML, et al. Comparison of two restraint systems for pelvic stabilisation during isometric lumbar extension strength testing. J Orthop Sports Phys Ther 1992; 15(1): 37–42
46. Farfan HF. Muscular mechanism of the lumbar spine and the position of power and efficiency. Orthop Clin North Am 1975; 6: 135–144
47. Inanami H. Iwai Orthopedic Hospital rehabilitation program. Paper presented at International Spinal Rehabilitation Update Symposium, Daytona, 1991
48. Alaranta H, Hurri H, Heliovaara M, et al. Non-dynamometric trunk performance tests: reliability and normative data. Scand J Rehabil Med 1994; 26(4): 211-215
49. Moffroid M, Reid S, Henry SM, et al. Some endurance measures in persons with chronic low back pain. J Orthop Sports Physl Ther 1994; 20(2): 81–87
50. Mayer T, Gatchel R, Betancur J, et al. Trunk muscle endurance measurement: isometric contrasted to isokinetic testing in normal subjects. Spine 1995; 20: 920–927

51. McGill SM, Childs A, Liebenson C. Endurance times for low back stabilization exercise: clinical targets for testing and training from a normal database. Arch Phys Med Rehabil 1999; 80: 941–944

52. Latimer J, Maher CG, Refshauge K, et al. The reliability and validity of the Biering-Sorenson test in asymptomatic subjects and subjects reporting current or previous nonspecific low back pain. Spine 1999; 24: 2085-2090

53. Graves JE, Pollock ML, Carpenter DM, et al. Quantitative assessment of full range of motion isometric lumbar extension strength. Spine 1990; 15(4): 289–294

54. Robinson ME, Greene AF, O'Connor P, et al. Reliability of lumbar isometric torque in patients with chronic low back pain. Phys Ther 1992; 72(3): 186–190

55. Udermann BE, Mayer JM, Graves JE, et al. Quantitative assessment of lumbar paraspinal muscle endurance. J Athl Train 2003; 38: 259–262

56. Hager SM, Udermann BE, Reineke DM, et al. Quantification of lumbar endurance on a backup lumbar extension dynamometer. J Sports Sci Med 2006; 5: 656–661

57. Perez LT, Peiro OB, Dies TP, et al. Fuerza lumbar en jugadores de hockey hierba. Apunts. Medicina De L'Esport 2007; 155: 138-144

58. Fulton MN. Spinal Rehabilitation (part 1) Measuring true functional ability in clinical practice. Ocala, Florida: MedX Corporation, 1993

59. Pollock ML, Graves JE, Carpenter DM, et al. Muscle. In: Hochschuler SH, Cotler HB, Guyer RD. Rehabilitation of the Spine: Science and Practice. St. Louis: Mosby, 263 – 284, 1993

60. Smith D, Bruce-Low S, Bissell G. Twenty years of specific, isolated lumbar extension research: A review. J Orthop 2008; 5(1): e14

61. Beimborn DS, Morrissey MC. A review of the literature related to trunk muscle performance. Spine 1988; 13(6): 655–660

62. McNeill T, Warwick D, Andersson G, et al. Trunk strengths in attempted flexion, extension, and lateral bending in healthy subjects and patients with low back disorders. Spine 1980; 5(6): 529–538
63. Addison R, Shultz A. Trunk strength in patients seeking hospitalization for chronic low back disorders. Spine 1980; 5(6): 539-544
64. Takemasa R, Yamamoto H, Tani T. Trunk muscle strength in and effect of trunk muscle exercises for patients with chronic low back pain. The differences in patients with and without organic lumbar lesions. Spine 1995; 20(23): 2522–2530
65. Handa N, Yamamoto H, Tani T, et al. The effect of trunk muscule exercises in patients over 40 years of age with chronic low back pain. J Orthop Sci 2000; 5: 210–216
66. Thorstensson A, Arvidson A. Trunk muscle strength and low back pain. Scand J Rehabil Med 1982. 14(2), 69 – 75
67. Suzuki N, Endo S. A quantitative study of trunk muscle strength and fatigability in the low back pain syndrome. Spine 1983; 8(1): 69–74
68. Biering-Sorensen F. Physical measurements as risk indicators for low-back trouble over a one-year period. Spine 1984; 9(2): 106–119
69. Leino P, Aro S, Hasan J. Trunk muscle function and low back disorders: a ten year follow up study. J Chronic Dis 1987. 40(4), 289 – 96
70. Bayramoglu M, Akman MN, Kilinc S, et al. Isokinetic measurement of trunk muscle strength in women with chronic low back pain. Am J Phys Med Rehabil 2001; 80(9): 650–655
71. Nicholaisen T, Jorgensen K. Trunk muscle strength, back muscle endurance and low back trouble. Scand J Rehabil Med 1985. 17(3), 121 – 127

72. Holmstrom E, Moritz U, Andersson M. Trunk muscle strength and back muscle endurance in construction workers with and without low back disorders. Scand J Rehabil Med 1992; 24(1): 3–10

73. Salminen JJ, Maki P, Okansen A, et al. Spinal mobility and trunk muscle strength in 15 year old school children with and without low back pain. Spine 1992; 17(4): 405–411

74. Hultman G, Nordin M, Saraste H, et al. Body composition, endurance, strength, cross-sectional area, and density of MM erector spinae in men and women with and without low back pain. J Spinal Dis 1993; 6(2): 114–123

75. Parkkola R, Rytokoski U, Kormano M. Magnetic resonance imaging of the discs and trunk muscles in patients with chronic low back pain and healthy control subjects. Spine 1993; 18(7): 830–836

76. Mayer TG, Vanharanta H, Gatchel RJ, et al. Comparison of CT scan muscle measurements and isokinetic trunk strength in postoperative patients. Spine 1989; 14(1): 33–36

77. Crossman K, Mahon M, Watson PJ, et al. Chronic low back pain-associated paraspinal muscle dysfunction is not the result of a constitutionally "adverse" fiber-type composition. Spine 2004; 29(6): 628–634

78. Paasuke M, Johanson E, Proosa M, et al. Back extensor fatigability in chronic low back pain patients and controls: Relationship between electromyogram power spectrum changes and body mass index. J Back Musculoskelet Rehabil 2002; 16(1): 17–24

79. Humphrey AR, Nargol AVF, Jones APC, et al. The value of electromyography of the lumbar paraspinal muscles in discriminating between chronic-low-back-pain sufferers and normal subjects. Eur Spine J 2005; 14: 175-184

80. Suuden E, Ereline J, Gapeyeva H, et al. Low back muscle fatigue during Sorenson endurance test in patients with chronic low back pain: relationship between

electromyographic spectral compression and anthropometric characteristics. Electromyogr Clin Neurophysiol 2008; 48(3-4): 185–192

81. Lariviere C, Da Silva RA, Arsenault AB, et al. Specificity of a back muscle roman chair exercise in healthy and back pain subjects. Med Sci Sports Exerc 2011; 43(1): 157–164
82. Demoulin C, Grosdent S, Debois I, et al. Inter-session, inter-tester and inter-site reproducibility of isometric muscle strength measurements. Isokinet Exerc Sci 2006. 14, pp 317-325
83. Luoto S, Heliovaara M, Hurri H, et al. Static back endurance and the risk of low back pain. Clin Biomech 1995; 10(6): 323–324
84. Gibbons LE, Videman T, Battie MC. Isokinetic and psychosocial lifting strength, static back muscle endurance, and magnetic resonance imaging of the paraspinal muscles as predictors of low back pain in men. Scand J Rehabil Med 1997; 29(3): 187–191
85. Battie M, Bigos SJ, Fisher LD, et al. Isometric lifting strength as a predictor of industrial back pain reports. Spine 1989; 14(8): 851–856
86. Balague F, Damidot P, Nordin M, et al. Cross-sectional study of the isokinetic muscle strength among school children. Spine 1993; 18(9): 1199–1205
87. Suter E, Lindsay D. Back muscle fatigability is associated with knee extensor inhibition in subjects with low back pain. Spine 2001; 26(16): E361–366
88. Da Silva RA, Arsenault AB, Gravel D, et al. Back muscle strength and fatigue in healthy and chronic low back pain subjects: A comparative study of 3 assessment protocols. Arch Phys Med Rehabil 2005; 86: 722–729
89. Lariviere C, Bilodeau M, Forget R, et al. Poor back muscle endurance is related to pain catastrophizing in patients with chronic low back pain. Spine 2010; 35(22): E1178-E1186
90. Renkawitz T, Boluki D, Grifka J. The association of low back pain, neuromuscular imbalance and trunk extension strength in athletes. Spine J 2006; 6: 673-683

91. Kamaz M, Kiresi D, Oguz H, et al. CT measurement of trunk muscle areas in patients with chronic low back pain. Diagn Interv Radiol 2007; 13: 144–148

92. Weber BR, Grob D, Dvorak J, et al. Posterior surgical approach to the lumbar spine and its effect on the multifidus muscle. Spine 1997; 22(15): 1765–1772

93. Rantanen J, Hurme M, Falck B, et al. The lumbar multifidus muscle five years after surgery for lumbar intervertebral disc herniation. Spine 1993; 18(5): 568–574

94. Sihvonen T, Herno A, Paljarvi L, et al. Local denervation atrophy of paraspinal muscle in postoperative failed back syndrome. Spine 1993; 18(5): 575–581

95. Motosuneya T, Asazume T, Tsuji T, et al. Post-operative change of the cross-sectional area of back musculature after 5 surgical procedures as assessed by magnetic resonance imaging. J Spinal Disord Tech 2006; 19(5): 318–322

96. Cassisi JE, Robinson ME, O'Connor P, et al. Trunk strength and lumbar paraspinal muscle activity during isometric exercise in chronic low-back pain patients and controls. Spine 1993. 18(2), 245 – 251

97. Holmes B, Mooney V, Negri S, et al. Comparison of female geriatric lumbar extension strength: asymptomatic versus chronic low back pain patients and their response to active rehabilitation. J Spinal Disord 1996; 9(1): 17–22

98. Robinson ME, Cassisi JE, O'Connor PD, et al. Lumbar iEMG during isotonic exercise: chronic low back pain patients vs. controls. J Spinal Disord 1992; 5(1): 8–15

99. Nelson BW, O'Reilly E, Miller M, et al. The clinical effects of intensive, specific exercise on low back pain: A controlled study of 895 consecutive patients with a one year follow up. Orthopedics 1995; 18(10): 971–981.

100. Mooney V, Kron M, Rummerfield P, et al. The effect of workplace based strengthening on low back injury rates: A case study in the strip mining industry. J Occup Rehabil 1995; 5(3): 157–167

101. Mooney V, Gulick J, Perlman M, et al. Relationships between myoelectric activity, strength and MRI of lumbar extensor muscles in back pain patients and normal subjects. J Spinal Disord 1997; 10(4): 348–356

102. Boyce R, Boone E, Stallings J, et al. A multidisciplinary approach to a time-efficient low back exercise intervention in a small manufacturing plant: A case study. J Exerc Physiol 2008; 11(4): 12–24

103. Hides JA, Stokes MJ, Saide M, et al. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/sub-acute low back pain. Spine 1994; 19: 165–172

104. Hadar H, Gadoth N, Heifetz M. Fatty replacement of lower paraspinal muscles: normal and neuromuscular disorders. AJR Am J Roentgenol 1983; 141(5): 895–898

105. Lexell J, Downham D. What is the effect of aging on type 2 muscle fibres? J Neurol Sci 1992; 107(2): 250–251

106. Mannion AF, Kaser L, Weber E, et al. Influence of age and duration of symptoms on fibre-type distribution and size of the back muscles in chronic low back pain patients. Eur Spine J 2000; 9: 273–281

107. Mengiardi B, Schmid MR, Boos N, et al. Fat content of lumbar paraspinal muscles in patients with chronic low back pain and asymptomatic volunteers: Quantification with MR spectroscopy. Radiology 2006; 240(3): 786–792

108. Sirca A, Kostevc V. The fibre type composition of thoracic and lumbar paravertebral muscles in man. J Anatomy 1985; 141: 131–137

109. Ford D, Bagnall KM, McFadden KD, et al. Analysis of vertebral muscle obtained during surgery for correction of a lumbar disc disorder. Acta Anat 1983; 16(2): 152–157

110. Bagnall KM, Ford DM, McFadden KD, et al. The histochemical composition of human vertebral muscle. Spine 1984; 9(5): 470–473

111. Cooper RG, St. Clair Forbes W, Jayson MIV. Radiographic demonstration of paraspinal muscle wasting in patients with chronic low back pain. Br J Rheumatol 1992; 31: 389–394

112. Bouche KGW, Vanovermeire O, Stevens VK, et al. Computed tomographic analysis of the quality of trunk muscles in asymptomatic and symptomatic lumbar discectomy patients. BMC Musculoskelet Disord 2011; 12: 65

113. Alaranta H, Tallroth K, Soukka A, et al. Fat content of lumbar extensors muscles and low back disability: a radiographic and clinical comparison. J Spinal Disord 1993; 6(2): 137–140

114. Kader DF, Wardlaw D, Smith FW. Correlation between the MRI changes in the lumbar multifidus muscles and leg pain. Clin Radiol 2000; 55: 145–149

115. Barker KL, Shamley DR, Jackson D. Changes in cross-sectional area of multifidus and psoas in patients with unilateral back pain: the relationship to pain and disability. Spine 2004; 29(22): E515–519

116. Kjaer P, Bendix T, Sorensen JS, et al. Are MRI-defined fat infiltrations in the multifidus muscles associated with low back pain? BMC Med 2007; 5(2):

117. Hyun JK, Lee JY, Lee SJ, et al. Asymmetric atrophy of multifidus muscle in patients with unilateral lumbosacral radiculopathy. Spine 2007; 32(21): E598-E602

118. Kalichman L, Hodges P, Li L, et al. Changes in paraspinal muscle and their association with low back pain and spinal degeneration: CT study. European Spine Journal 2010; 19: 1136–1144

119. Hicks GE, Simonsick EM, Harris TB, et al. Cross-sectional association between trunk muscle composition, back pain, and physical function in the health, aging and body composition study. J Gerontol; 60(7): 882-887

120. Zhu XZ, Parnianpour M, Nordin M, et al. Histochemistry and morphology of erector spinae muscle in lumbar disc herniation. Spine 1989; 14(4): 391–397

121. Mannion AF, Weber BR, Dvorak J, et al. Fibre type characteristics of the lumbar paraspinal muscles in normal healthy subjects and in patients with low back pain. J Orthop Res 1997; 15(6): 881–887

122. Fidler MW, Jowett RL, Troup JDG. Myosin ATPase activity in multifidus muscle from cases of lumbar spinal derangement. J Bone Joint Surg Br 1975; 57(2): 220–227

123. Mattila M, Hurme M, Alaranta H, et al. The multifidus muscle in patients with lumbar disc herniation. A histochemical and morphometric analysis of intraoperative biopsies. Spine 1986; 11(7): 737–738

124. Zhao WP, Kawaguchi Y, Matsui H, et al. Histochemistry and morphology of the multifidus muscle in lumbar disc herniation: a comparative study between diseased and normal sides. Spine 2000; 25(17): 2191–2199

125. Bajek S, Bobinac D, Bajek G, et al. Muscle fiber type distribution in multifidus muscle in cases of lumbar disc herniation. Acta Med Okayama 2000; 54(6): 235–241

126. Yoshihara K, Shirai Y, Nakayama Y, et al. Histochemical changes in the multifidus muscle in patients with lumbar intervertebral disc herniation. Spine 2001; 26(6): 622–626

127. Danneels LA, Vanderstraeten GG, Cambier DC, et al. CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. European Spine Journal 2000; 9: 266–272

128. Kang CH, Shin MJ, Kim SM, et al. MRI of paraspinal muscles in lumbar degenerative kyphosis patients and control patients with chronic low back pain. Clin Radiol 2007; 62(5): 479-486

129. Luthi JM, Howald H, Claassen H, et al. Structural changes in skeletal muscle tissue with heavy resistance exercise. Int J Sports Med 1986; 7(3): 123–127

130. Hepple RT, Mackinnon SLM, Goodman JM, et al. Resistance and aerobic training in older men: effects on VO_2 peak and the capillary supply to skeletal muscle. J Appl Physiol 1997; 82: 1305-10.

131. Green H, Goreham C, Ouyang J, et al. Regulation of fiber size, oxidative potential, and capillarization in human muscle by resistance exercise. Am J Physiol 1999; 276: R591-596

132. Steele J, Fisher J, McGuff D, et al. Resistance training to momentary muscular failure improves cardiovascular fitness in humans: A review of acute physiological responses and chronic physiological adaptations. J Exerc Physiol 2012; 15(3): 53–80

133. Mannion AF, Dumas GA, Cooper RG, et al. Muscle fibre size and type distribution in thoracic and lumbar regions of erector spinae in healthy subjects without low back pain: normal values and sex differences. J Anatomy 1997; 190: 505–513

134. Haig AJ, Weismann G, Haugh LD, et al. Prospective evidence for change in paraspinal muscle activity after herniated nucleus pulposus. Spine 1993; 18(7): 926

135. Roy SH, De Luca CJ, Casavant DA. Lumbar muscle fatigue and chronic low back pain. Spine 1989; 14(9): 992–1001

136. Bouissou P, Estrade PV, Goubel F, et al. Surface EMG power spectrum and intramuscular pH in human vastus lateralis muscle during dynamic exercise. J Appl Physiol 1989; 67(3): 1245–1249

137. Vestergaard Poulsen P, Thomsen C, Sinkjaer T, et al. Simultaneous electromyography and 31P nuclear magnetic resonance spectroscopy - with application to muscle fatigue. Electroencephalogr Clin Neurophysiol 1992; 85(6): 402-411

138. Laurent D, Portero P, Goubel F, et al. Electromyogram spectrum changes during sustained contraction related to proton and diprotonated inorganic phosphate

accumulation: a 31P nuclear magnetic resonance study on human calf muscles. Eur J Appl Physiol 1993; 66(3): 263–268

139. Cooper RG, Stokes MJ, Sweet C, et al. Increased central drive during fatiguing contractions of the paraspinal muscles in chronic low back pain. Spine 1993; 18(5): 610-616

140. De Luca C. The Use of Surface Electromyography in Biomechanics. J Appl Biomech 1997; 13: 135–163

141. De Luca C, Merletti R. Surface myoelectric signal cross-talk among muscles of the leg. Electroencephalogr Clin Neurophysiol 1988; 69: 568–575

142. Wakeling JM, Pascual SA, Nigg BM, et al. Surface EMG shows distinct populations of muscle activity when measured during sustained submaximal exercise. Eur J Appl Physiol 2001; 86: 40–47

143. Roman-Liu D, Tokarski T. EMG of arm forearm muscle activities with regard to handgrip force in relation to upper limb location. Acta Bioengineering Biomech 2002; 4(2): 33–48

144. Farina D, Merletti R, Enoka RM. The extraction of neural strategies from the surface EMG. J Appl Physiol 2004; 96: 1486–1495

145. Semmler JG, Tucker KJ, Allen TJ, et al. Eccentric exercise increases EMG amplitude and force fluctuations during submaximal contractions of elbow flexor muscles. J Appl Physiol 2007; 103: 979–989

146. Roberts TJ, Gabaldon AM. Interpreting muscle function from EMG: lessons learned from direct measurements of muscle force. Integr Comp Biol 2008; 48(2): 312–320

147. Stokes IAF, Henry SM, Single RM. Surface electrodes do not accurately record from lumbar multifidus muscles. Clin Biomech 2003; 18: 9–13

148. De Luca C. Use of the surface EMG signal for performance evaluation of back muscles. Muscle Nerve 1993; 16: 210–216

149. Pitcher MJ, Behm DG, MacKinnon SN. Reliability of electromyographic and force measures during isometric back extension in subjects with and without low back pain. Appl Physiol Nutr Metab 2008; 33(1): 52-60

150. Watson PJ, Booker CK, Main CJ. Evidence for the role of psychological factors in abnormal paraspinal activity in patients with chronic low back pain. J Musculoskelet Pain 1997; 5(4): 41–56

151. Roy SH, De Luca CJ, Emley M, et al. Spectral electromyographic assessment of back muscles in patients with low back pain undergoing rehabilitation. Spine 1995; 20(1): 38–48

152. Steele J. Intensity; in-ten-si-ty; noun. 1. Often used ambiguously within resistance training. 2. Is it time to drop the term altogether? Br J Sports Med 2013. Online First

153. Geisser ME, Ranavaya M, Haig AJ, et al. A meta-analytic review of surface electromyography among persons with low back pain and normal, healthy controls. The J Pain 2005; 6(11): 711–726

154. Mayer TG, Kondraske G, Mooney V, et al. Lumbar myoelectric spectral analysis for endurance assessment. A comparison of normal and deconditioned patients. Spine 1989; 14(9): 989–991

155. Peach JP, McGill SM. Classification of low back pain with the use of spectral electromyogram parameters. Spine 1998; 23(10): 1117–1123

156. Roy SH, De Luca CJ, Snyder-Mackler L, et al. Fatigue, recovery, and low back pain in varsity rowers. Med Sci Sports Exerc 1990; 22(4): 463–469

157. Biedermann HJ, Shanks GL, Forrest WJ, et al. Power spectrum analyses of electromyographic activity. Discriminators in the differential assessment of patients with chronic low back pain. Spine 1991; 16(10): 1179–1184

158. Klein AB, Snyder-Mackler L, Roy SH, et al. Comparison of Spinal Mobility and Isometric Trunk Extensor Forces with Electromyographic Spectral Analysis in Identifying Low Back Pain. Phys Ther 1991; 71(6): 445–454

159. Mannion AF, Connolly B, Wood K, et al. The use of surface EMG power spectral analysis in the evaluation of back muscle function. J Rehabil Res Dev 1997; 34(4): 427–439

160. Lariviere C, Gagnon D, Gravel D, et al. The assessment of back muscle capacity using intermittent static contractions. Part 1 – Validity and reliability of electromyographic indices of fatigue. J Electromyogr Kinesiol 2008; 18(6): 1006-1119

161. Hill AB. The environment and disease: association or causation. J R Soc Med 1965; 58: 295–300

162. Mooney V, Andersson GBJ. Controversies. Trunk strength testing in patient evaluation and treatment. Spine 1994; 19(21): 2483–2485

163. Rissanen A, Heliovaara M, Alaranta H, et al. Does good trunk extensor performance protect against back-related work disability. J Rehabil Med 2002; 34: 62-66

164. Bemben MG, Grump KJ, Massey BH. Assessment of technical accuracy of the Cybex II dynamometer and analog recording system. J Orthop Sports Phys Ther 1988; 10: 12–17

165. Mooney V. On the dose of therapeutic exercise. Orthopedics 1992; 15(5): 653–656

166. Murray D. Optimal filtering of constant velocity torque data. Med Sci Sports Exerc 1986; 18: 603–611

167. Newton M, Thow M, Somerville D, et al. Trunk strength testing with iso-machines. Part 2. Experimental evaluation of the cybex II back testing system in normal subjects and patients with chronic low back pain. Spine 1993; 18(7): 812 – 824

168. Reimer DS, Halbrook BD, Dreyfuss PH, et al. A novel approach to preemployment worker fitness evaluations in a material handling industry. Spine 1994; 19(18): 2026–2032

169. Batt'ie M, Bigos SJ, Fisher LD, et al. Isometric lifting strength as a predictor of industrial back pain reports. Spine 1989; 14(8): 851–856

170. Lee JH, Hoshino Y, Nakamura K, et al. Trunk muscle weakness as a risk factor for low back pain. A 5 year prospective study. Spine 1999; 24(1): 54–57

171. Kujala UM, Taimela S, Viljanen T, et al. Physical loading and performance as predictors of back pain in healthy adults. A 5-year prospective study. Eur J Appl Physiol 1996; 73: 452–458

172. Chaffin DB, Herrin GD, Keyserling WM. Preemployment strength testing. An updated position. J Occup Med 1978; 20(6): 403–408

173. Keyserling WM, Herrin GD, Chaffin DB. Isometric strength testing as a means of controlling medical incidents on strenuous jobs. J Occup Med 1980; 22(5): 332–336

174. Salminen JJ, Erkintalo M, Laine M, et al. Low back pain in the young. A prospective three year follow up study of subjects with and without low back pain. Spine 1995; 20(19): 2101–2107

175. Sjolie AN, Ljunggren AE. The significance of high lumbar mobility and low lumbar strength for current and future low back pain in adolescents. Spine 2001; 26(23): 2629–2636

176. Adams MA, Mannion AF, Dolan P. Personal risk factors for first-time low back pain. Spine 1999; 24(23): 2497–2505

177. Mostardi RA, Noe DA, Kovacik MW, et al. Isokinetic lifting strength and occupational injury. A prospective study. Spine 1992; 17(2): 189–193

178. Cady LD, Bischoff DP, O'Connell ER, et al. Strength and fitness and subsequent back injuries in firefighters. J Occup Med 1979; 21(4): 269–272

179. Mooney V, Kenney K, Leggett S, et al. Relationship of lumbar strength in shipyard workers to workplace injury claims. Spine 1996; 21(17): 2001–2005

180. Guo HR, Tanaka S, Halperin WE, et al. Back pain prevalence in US industry and estimates of lost workdays. Am J Public Health 1999; 89: 1029–1035

181. Stevenson JM, Weber CL, Smith JT, et al. A longitudinal study of the development of low back pain in an industrial population. Spine 2001; 26(12): 1370-1377

182. Heydari A, Nargol AVF, Jones APC, et al. EMG analysis of lumbar paraspinal muscles as a predictor of the risk of low-back pain. Eur Spine J 2010; 19: 1145-1152

183. Pollock ML, Leggett SH, Graves JE, et al. Effect of resistance training on lumbar extension strength. Am J Sports Med 1989; 17(5): 624–629

184. Carpenter DM, Nelson BW. Low back strengthening for the prevention and treatment of low back pain. Med Sci Sports Exerc 1999; 31(1): 18–24

185. Jones A. The lumbar spine, the cervical spine and the knee. Ocala, Florida: MedX Corporation, 1993

186. Smith D, Bissell G, Bruce-Low S, et al. The effect of lumbar extension training with and without pelvic stabilization on lumbar strength and low back pain. J Back Musculoskelet Rehabil 2011; 24: 1–9

187. Van Dieen JH, Kuijer PPFM, Burdorf A, et al. Non-specific low back pain. Lancet 2012; 379(9829): 1874

188. Balague F, Mannion AF, Pellise F, et al. Non-specific low back pain – Authors' reply. Lancet 2012; 379(9829): 1874–1875

189. Mooney V, Matheson L, Holmes D, et al. Effect of focused strength training after low back injury. North American Spine Society Annual Meeting; 1993; San Diego, California

190. Deutsch FE. Isolated lumbar strengthening in the rehabilitation of chronic low back pain. J Manipulative Physiol Ther 1996; 19(2): 124–133

191. Park YJ, Choi KS, Lee SG. Effect of lumbar extensor strengthening in chronic low back pain patients. J Korean Acad Rehabil Med 2000; 24(2): 295–300

192. Lee KW, Kwon JY, Kim HS, et al. Back exercise program with lumbar extension resisting exercise in patients with chronic low back pain. J Korean Acad Rehabil Med 2000; 24(3): 536–541

193. Choi G, Raiturker PP, Kim M, et al. The effect of early isolated lumbar extension exercise program for patients with herniated disc undergoing lumbar discectomy. Neurosurgery 2005; 57: 764–772

194. Bruce-Low S, Smith D, Burnet S, et al. One lumbar extension training session per week is sufficient for gains and reductions in pain in patients with chronic low back pain ergonomics. Ergonomics 2012; 55(4): 500–507

195. Risch SV, Norvell NK, Pollock ML, et al. Lumbar strengthening in chronic low back pain patients. Spine 1993; 18(2): 232–238

196. Leggett S, Mooney V, Matheson LN, et al. Restorative exercise for clinical low back pain: a prospective two-center study with 1-year follow up. Spine 1999; 24(9): 889–898

197. Costa K. Effects of a trunk strengthening program on pain perception, strength and flexibility on patients with non-specific low back pain. Thesis, Pindara Physiotherapy and Sports Medicine, 2010

198. Carlson J, MacKay G. Impact of specific muscular strength therapy on patients with chronic lower back pain. Thesis, Functional Physio, 2010

199. Al-Obaidi SM, Beattie P, Al-Zoabi B, et al. The relationship of anticipated pain and fear avoidance beliefs to outcome in patients with chronic low back pain who are not receiving workers compensation. Spine 2005; 30(9): 1051–1057

200. Steele J, Bruce-Low S, Smith D, et al. A Randomised Controlled Trial of Limited Range of Motion Lumbar Extension Exercise in Chronic Low Back Pain. Spine 2013. 38(15), pp 1245-1252

201. Mooney V, Kron M, Rummerfield P, et al. The effect of workplace based strengthening on low back injury rates: A case study in the strip mining industry. J Occup Rehabil 1995; 5(3): 157–167

202. Matheson L, Mooney V. Employment screening and functional capacity evaluation. In. Liebenson C. Rehabilitation of the Spine: A Practitioners Manual. New York: Lippincott Williams & Wilkins, 1993: 276

203. Dreisinger TE. Does prevention work. In: San Diego Comprehensive Care Symposium. San Diego CA, 2000

204. Lovejoy CO. Evolution of the human lumbopelvic region and its relationship to some clinical deficits of the spine and pelvis. In. Vleeming A, Mooney V, Stoeckart R. Movement, Stability and Lumbopelvic Pain. Integration of Research and Therapy. New York: Churchill Livingstone, pp 141 – 158, 2007

205. Moffroid MT, Haugh LD, Haig AJ, et al. Endurance training of trunk extensor muscles. Phys Ther 1993; 73(1): 10–17

206. Graves JE, Webb DC, Pollock ML, et al. Pelvic stabilization during resistance training: Its effect on the development of lumbar extension strength. Arch Phys Med Rehabil 1994; 75: 210–215

207. Mayer JM, Graves JE, Udermann BE, et al. Development of lumbar extension strength: Effect of pelvic stabilization during resistance training. J Back Musculoskelet Rehabil 2002; 16: 25–31

208. Verna JL, Mayer JM, Mooney V, et al. Back extension endurance and strength: the effect of variable-angle roman chair exercise training. Spine 2002; 27(16): 1772-1777

209. Johnston BD. The need for direct, isolated exercise in spinal strength and fitness: the effects of the medx lumbar extension machine on three case subjects. J Appl Fitness 2005; 1 – 9

210. Fisher J, Bruce-Low S, Smith D. A Randomized Trial to Consider the Effect of Romanian Deadlift Exercise on the development of Lumbar Extension Strength. Phys Ther Sport 2012. IN PRESS

211. Helmhout PH, Staal JB, Maher CG, et al. Exercise Therapy and Low Back Pain: Insights and proposals to improve the design, conduct, and reporting of clinical trials. Spine 2008; 33(16): 1782–1788

212. Mayer J, Mooney V, Dagenais S. Evidence informed management of chronic low back pain with lumbar extensor strengthening exercises. Spine J 2008; 8: 96–113

213. Van Tulder MW, Malmivaara A, Esmail R, et al. Exercise therapy for low back pain. Cochrane Database Syst Rev 2000; 2: CD00335

214. Hayden J, Van Tulder MW, Malmivaara A, et al. Exercise therapy for no-specific low back pain. Cochrane Database Syst Rev 2005; 3: CD000335

215. Manniche C, Jordan A. Letter to the editor. Spine 2001; 26(7): 842–843

216. Manniche C, Jordan A. Letter to the editor. Spine 2001; 26(8): 994

217. Steele J, Bruce-Low S. Steiger et al. 2011: relationships and specificity in CLBP rehabilitation through exercise. Eur Spine J 2012; 21(9), 1887

218. Steele J, Bruce-Low S, Smith D. Controlling Resistance Training Variables in Interventions for Chronic Nonspecific Low Back Pain: Letter to the Editor regarding "The Effects of Dynamic Isolated Lumbar Extensor Training on Lumbar Multifidus Functional Cross-Sectional Area and Functional Status of Patients with Chronic Nonspecific Low Back Pain." Spine 2013. In Press

219. Steele J, Bruce-Low S, Smith D. A review of the specificity of exercises designed for conditioning the lumbar extensors. Br J Sports Med. Online First

Figure Ledgends

Figure 1. Disuse Syndrome Model, from [2]

Figure 2. Deconditioning Syndrome Model – Adapted* Disuse Syndrome Model, adapted from [2]

Figure 1

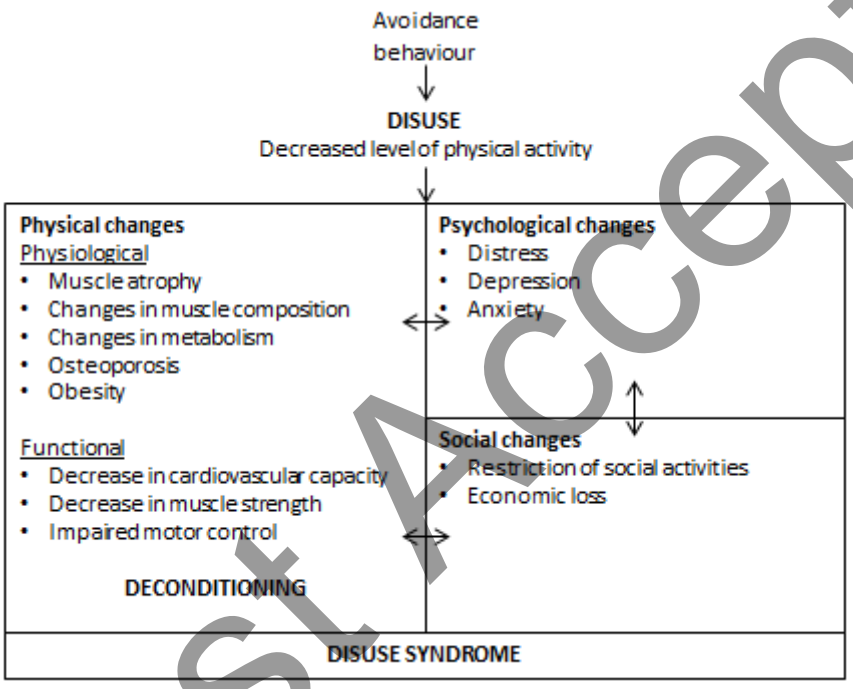

Figure 2

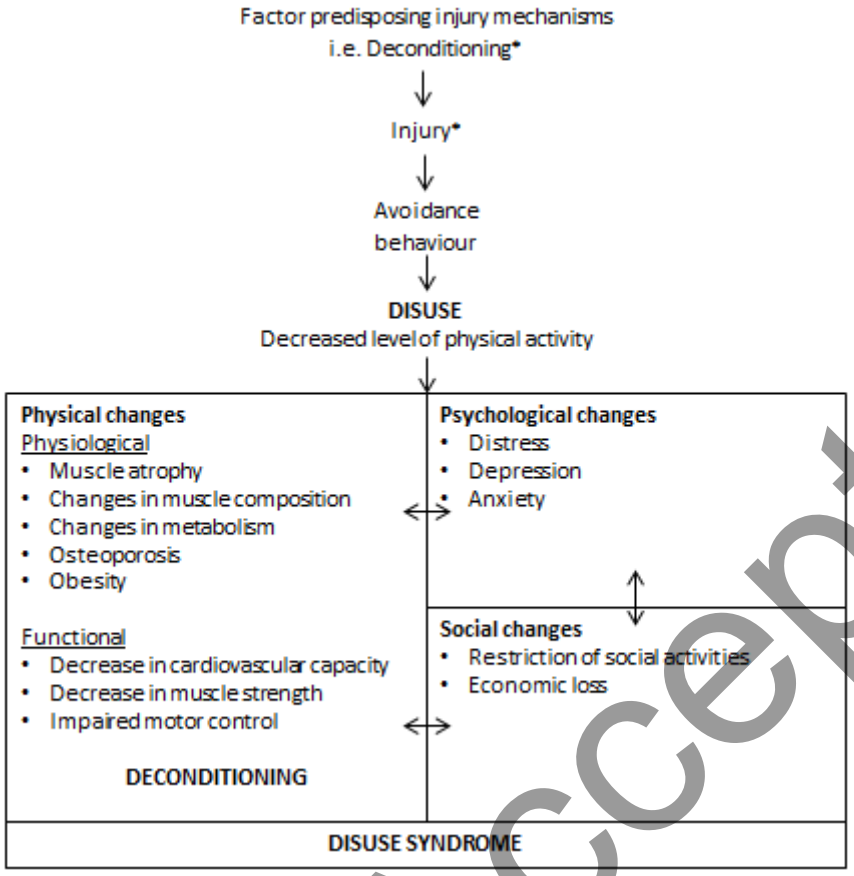

Table 1. Summary of studies testing strength and endurance of the lumbar extensor musculature in LBP

Reference	*Participants*	*Testing*	*Results*	*Comments*
Trunk Extension Studies				
Kankaanpaa et al. [37]	Healthy controls without history of LBP, *n* = 15 Middle aged women with CLBP, *n* = 20	Isometric MVC and isometric endurance to failure at 50%MVC during seated (knees 90^{o}) restrained trunk extension	Significantly lower MVC and time endurance time to exhaustion in CLBP (both $p <$ 0.05)	Those with previous lumbar surgery were excluded Age, height, body mass and BMI similar between groups
Alaranta et al. [48]	Never any pain, *n* = 116 Pain more than 12 months ago, *n* = 46 Pain during previous 12 month, no disability, *n* = 166 Disabling pain during previous	Biering-Sorensen test	Significantly lower endurance time in those with history of LBP ($p < 0.05$)	

	12 months, *n* = 147			
McNeil et al. [62]	Healthy controls, *n* = 57 CLBP patients, *n* = 40	Standing trunk extension/flexion MVC with pelvis restrained at top of iliac crest on superior edge of backboard using a belt across the anterior superior iliac spine, and bilateral restraints upon the iliac crests.	Both extension/flexion were lower in CLBP, however extension was reduced to a significantly greater degree shown by significantly lower extension/flexion ratios ($p < 0.01$)	Participants with sciatica & CLBP had significantly lower extension strength compared to both just CLBP participants and healthy controls ($p < 0.01$) – with the exception of comparison to females with CLBP (*ns)*
Addison & Schultz [63}	Healthy controls, *n* = 57 CLBP patients, *n* = 33	Standing trunk extension/flexion MVC with pelvis restrained at top of iliac crest on superior edge of backboard using a belt across the anterior superior iliac spine, and bilateral restraints upon the iliac crests.	Both extension/flexion were lower in CLBP, however extension was reduced to a significantly greater degree shown by significantly lower extension/flexion ratios ($p < 0.001$)	No differences between CLBP and an outpatient CLBP group suggesting common physical deficit despite differences in treatment seeking behaviour
Takemasa et al. [64]	Healthy controls without past	Isometric MVC during seated	Both flexion/extension were	No differences CLBP with or

	history of LBP, *n* = 126 CLBP with or without organic lumbar lesions, *n* = 123	(knees 90°) restrained flexion/extension	significantly lower in CLBP ($p < 0.05$), however extension was reduced to a significantly greater degree shown by significantly higher flexion/extension ratios in lesion group ($p < 0.01$)	without organic lumbar lesions suggesting common physical deficit despite differences symptoms Age, height, body mass and BMI similar between groups
Handa et al. [65]	Healthy controls without past history of LBP, *n* = 60 CLBP patients, *n* = 52	Isometric MVC during seated (knees 90°) restrained flexion/extension	Isometric flexion did not significantly differ between groups, isometric extension was significantly lower in CLBP group ($p < 0.05$)	Age, height, body mass and BMI similar between groups
Suzuki & Endo [67]	Healthy controls without past history of LBP, *n* = 50 CLBP patients with or without root impairment, *n* = 90	Prone trunk extension MVC and flexion with legs both straight and bent at hips and knees with restraint belts across lower extremities	Both straight leg flexion, and trunk extension were significantly weaker in the CLBP group ($p < 0.001$)	Age weight and height similar between groups

Leino et al. [69]	*Baseline participants* Participants with "Good" low back status, *n* = 578 Participants with "Intermediate" low back status, *n* = 260 Participants with "Bad" low back status, *n* = 64 *Follow-up participants* Participants with "Good" low back status, *n* = 239 Participants with "Intermediate" low back status, *n* = 203	Standing dynamic (baseline) and isometric (follow-up) trunk extension/flexion MVC with buttock and thighs against a supporting plate and ankles tied by a belt	At baseline dynamic flexion was significantly weaker in those with worse low back status ($p < 0.01$) however dynamic extension was significantly weaker only in women ($p < 0.05$) At follow-up isometric flexion was significantly weaker in only men with worse low back status ($p = 0.01$) however isometric extension was significantly weaker in both men and women ($p < 0.05$)

	Participants with "Bad" low back status, *n* = 210			
Bayramoglu et al. [70]	Healthy controls with no history of LBP past 2 years, *n* = 20 CLBP patients, *n* = 25	Standing trunk extension/flexion MVC with stabilised knees and lower back	Both flexion and extension were significantly weaker in CLBP group ($p < 0.05$)	Age and height similar between groups
Nicholaisen & Jorgensen [71]	(Group 1) LBP that made work impossible, *n* = 17 (Group 2) LBP but not that hindered work, *n* =28 (Group 3) No history of LBP, *n* = 32	Standing trunk extension/flexion MVC a and isometric extension endurance to exhaustion at 60%MVC with stabilised knees and lower back Biering-Sorensen test	No difference in extension/flexion strength between groups. Isometric endurance significantly lower for Group 1 compared to 2 3 in females and males ($p < 0.05$) Endurance time significantly lower females for Biering-	Age, weight, height and fat free mass similar between groups except for age being higher in group 1 and weight and fat free mass higher in group 1 for females

			Sorenson test ($p < 0.05$)	
Holmstrom et al. [72]	(Group A) Healthy controls with no history of LBP, $n = 42$	Standing trunk extension/flexion MVC unrestrained lower extremities	No difference in extension/flexion strength between groups	Age, weight and height similar between groups
	(Group B) CLBP patients with uncertain or negative clinical assessment, $n = 75$	Biering-Sorensen test	Extension/flexion ratio was significantly lower in Group C compared to A ($p < 0.05$)	
	(Group C) CLBP patients with positive clinical assessment, $n = 86$		Endurance time significantly lower in both Group C and B compared to A for Biering-Sorenson test ($p < 0.01$)	
Salminen et al. [73]	Healthy children, $n = 38$	Biering-Sorensen test	Both flexion and extension endurance times were significantly lower in LBP groups ($p < 0.05$)	No differences CLBP with or without sciatica suggesting common physical deficit despite differences symptoms
	Children with LBP, $n = 31$	Sit up isometric test with knees at 90^0		

	Children with LBP and sciatica, $n = 7$		No difference between LBP and LBP with sciatica was found.	Age, sex, school matched between groups
Hultman et al. [74]	Healthy controls without history of LBP, $n = 36$ Patients with intermittent LBP, $n = 91$ CLBP patients, $n = 21$	Seated isokinetic/isometric trunk extension/flexion with thighs restrained Biering-Sorensen test	All variables, except isokinetic/isometric trunk flexion, were significantly lower in CLBP compared to healthy controls and intermittent LBP patients ($p < 0.05$)	Those with previous lumbar surgery were excluded Age, height, body mass and body composition similar between groups
Parkkola et al. [75]	Healthy controls, $n = 60$ CLBP patients suitable for active rehabilitation, $n = 38$ CLBP patients with serious back problems suitable for moderate	Standing isometric trunk extension/flexion MVC with chest, thighs and hips restrained	Extension/flexion MVCs showed a gradient between the three groups from higher to lower.	No statistical data reported Incidence of disc degeneration significantly higher in CLBP patients ($p < 0.05$) Age, sex, employment and

	rehabilitation only, *n* = 10			profession matched between groups and BMI similar
Mayer et al. [76]	Healthy controls without history of previous LBP, *n* = 19 Postoperative spinal disc surgery patients, *n* = 46	Isokinetic trunk extension/flexion peak torque unrestrained lower extremities	Both extension and flexion were significantly lower in the postoperative group ($p < 0.05$) with the greatest decrease being in extension strength	There was a significant correlation between trunk extensor strength and muscle density in postoperative patients No information on whether demographic characteristics differed between groups
Crossman et al [77]	Healthy controls without lasting >3 days in previous 12 months, *n* = 32 CLBP patients, *n* = 35	Standing trunk extension/flexion isometric MVC unrestrained lower extremities Biering-Sorensen test	MVC and endurance time significantly lower in CLBP group ($p < 0.05$)	Those with previous lumbar surgery were excluded Age, gender and all anthropometric characteristics similar between groups
Paasuke et al. [78]	Healthy controls, *n* = 12	EMG recorded bilaterally from	Endurance time was	Those with previous lumbar

		lumbar paraspinal muscles at L3 level 3cm from midline during Biering-Sorenson test to failure	significantly lower in the CLBP group ($p < 0.05$)	surgery were excluded Age and gender matched between participant groups Age, height, body mass and BMI similar among participant groups
Humphrey et al. [79]	Healthy controls without history of LBP in previous 5 years, n = 175 CLBP patients, n = 145 Participants with past history of LBP but no attack within previous 2 years, n = 30	Back lift MVC	MVC significantly lower in CLBP patients compared to controls ($p < 0.01$)	Those with previous lumbar surgery were excluded CLBP group was significantly older and had higher body mass and BMI than controls

Suuden et al. [80]	Healthy controls, *n* = 20 CLBP patients, *n* = 20	Biering-Sorenson test	Endurance time significantly lower for CLBP patients compared to controls ($p < 0.05$)	Those with previous lumbar surgery were excluded Age, height and weight and BMI similar between groups
Lariviere et al. [81]	Healthy controls without history of LBP in previous year, *n* = 18 CLBP patients, *n* = 18	Dynamic roman chair trunk extensions to failure	Number of repetitions to failure were significantly less in CLBP patients compared to controls ($p < 0.001$)	Those with previous lumbar surgery were excluded Age, height, weight and BMI similar between groups
Demoulin et al. [82]	Healthy controls without history of LBP in previous year, *n* = 10 CLBP participants, *n* = 10	Isometric MVC during seated (knees 90^{o}) restrained trunk extension	Extension strength significantly weaker in CLBP ($p < 0.05$)	Those with previous lumbar surgery were excluded
Balague et al. [86]	Children (10-16yrs) without history of LBP, *n* = 79	Standing isokinetic trunk extension/flexion peak torque	No significant differences for flexion or extension between	

		unrestrained lower extremities	groups at any age	
	Children (10-16yrs) with history of LBP, *n* 38			
Suter & Lindsay [87]	Healthy controls, *n* = 16	Biering-Sorenson test	No significant difference in endurance time between groups	Age, height and weight similar between groups
	Golfers with CLBP, *n* = 25			
Da Silva et al. [88]	Healthy controls without history of LBP in previous year, *n* = 15	Standing trunk extension, prone trunk extension and back lift MVC	No differences between groups	Those with previous lumbar surgery were excluded
	CLBP patients, *n* = 13			Age, height and weight similar between groups
Lariviere et al. [89]	Healthy controls without LBP lasting 1 wk in previous year, *n* = 31	Standing trunk extension/flexion MVC and repetitions to failure (endurance time) with stabilised knees and lower back	No significant difference between health controls and CLBP patients for strength or endurance time	Those with previous lumbar surgery were excluded
				Age, height, weight and BMI

	CLBP patients, *n* = 27			similar between groups
			Low predicted endurance time was associated with high pain catastrophising in CLBP patients ($p < 0.01$)	
Renkawitz et al. [90]	Healthy tennis players without LBP, *n* = 36 Tennis players with CLBP, *n* = 48	Standing isometric trunk extension MVC with shoulders, pelvis and thighs hips restrained	No association between presence of CLBP and trunk extension strength in either univariate or multivariate logistic regressions	
Isolated Lumbar Extension				
Lariviere et al. [40]	Healthy controls without LBP lasting 1 wk in previous year, *n* = 18 CLBP patients, *n* = 18	Isolated lumbar extension MVC and number of repetition to failure at 60%MVC using customised dynamometer	No significant difference between groups	Those with previous lumbar surgery were excluded Age, body mass, height, BMI, body % and physical activity

				levels were similar between groups
Cassisi et al. [96]	Healthy controls without history of LBP, *n* = 12 CLBP patients, *n* = 21	Isolated lumbar extension MVC using MEDX	Lumbar extension significantly weaker in CLBP ($p = 0.01$)	13 CLBP patients had undergone previous surgery though no effect upon lumbar extension strength was observed Age and height were similar between groups though body mass was greater in CLBP group
Holmes et al. [97]	Healthy geriatric female controls, *n* = 20 CLBP geriatric female patients, *n* = 18	Isolated lumbar extension MVC using MEDX	Lumbar extension significantly weaker in CLBP ($p < 0.05$)	Age, height and weight similar between groups
Robinson et al. [98]	Healthy controls, *n* = 12 CLBP patients (53% having had	Isolated lumbar extension MVC using MEDX was performed and 60%MVC determined at full	Absolute load used during isotonic trial was significantly lower in the CLBP group	10 CLBP patients had undergone previous surgery

	previous surgery), *n* = 16	extension for further EMG analysis during isotonic trial (see table 3)	compared with the asymptomatic controls ($p < 0.05$)	Age, height and weight similar between groups
Nelson et al. [99]	CLBP patients, *n* = 895	Isolated lumbar extension MVC using MEDX	CLBP baseline data was compared graphically to healthy norms from [44] and shown to considerably weaker.	Patients diagnoses included non-specific CLBP, degenerative disc/arthritic disease, lumbar disc syndrome or spondylolisthesis/spondylolysis
Mooney et al. [100]	Strip mine workers (90% reported prior LBP), *n* = 197	Isolated lumbar extension MVC using MEDX	Baseline data was compared graphically to healthy norms from [44] and shown to considerably weaker.	
Mooney et al. [101]	Healthy controls, *n* = 8 CLBP patients, *n* = 8	Isolated lumbar extension MVC using MEDX	CLBP baseline data was compared graphically to both healthy participants in the study and healthy norms from [44] and shown to be considerably weaker.	Patients showed evidence of degenerative disc disease

Boyce et al. [102]	Small manufacturing plant workers (53% reported LBP), *n* = 20	Isolated lumbar extension MVC using MEDX	Baseline data was compared graphically to healthy norms from [44] and shown to considerably weaker.	

Table 2. Summary of imaging and histochemical studies of the lumbar extensor musculature in LBP

Reference	***Participants***	***Testing***	***Results***	***Comments***
Imaging Studies				
Hultman et al. [74]	Healthy controls without history of LBP, *n* = 24 Patients with intermittent LBP, *n* = 40	CSA and density of erector spinae using CT at L3 level	Muscle density was significantly lower in CLBP patients compared to both other groups ($p < 0.05$) CSA did not significantly differ	Those with previous lumbar surgery were excluded Age, height, body mass and body composition similar between groups

	CLBP patients, *n* = 21		between groups	
Parkkola et al. [75]	Healthy controls, *n* = 60	CSA, fat content and grading status graded using 4 classification system of psoas and back muscles (erector spinae and multifidus) using MRI at L4/L5 level	CSA was significantly lower in both CLBP groups compared with controls ($p < 0.001$)	Incidence of disc degeneration significantly higher in CLBP patients ($p < 0.05$)
	CLBP patients suitable for active rehabilitation, *n* = 38		Back muscle status showed a gradient between the three groups from better to worse. It was significantly worse in severe CLBP patients compared with mild CLBP patients ($p < 0.05$) and healthy controls ($p < 0.001$), and was significantly worse in mild CLBP patients compared with controls also ($p < 0.05$)	Age, sex, employment and profession matched between groups
	CLBP patients with serious back problems suitable for moderate rehabilitation only, *n* = 10		Psoas muscles did not differ between groups	

Mayer et al. [76]	Healthy controls without history of previous LBP, *n* = 19 Postoperative spinal disc surgery patients, *n* = 46	CSA and muscle density of psoas, erector spinae, rectus abdominus and obliques using CT at L3	Non-significant trends towards reduced CSA in psoas and erector spinae were found in the postoperative group Muscle density of psoas and erector spinae was significantly lower in the postoperative group ($p < 0.001$)	There was a significant correlation between trunk extensor strength and muscle density in postoperative patients No information on whether demographic characteristics differed between groups
Kamaz et al. [91]	Healthy controls without LBP or leg pain, *n* = 34 CLBP patients, *n* = 36	CSA of total paraspinal, multifidus, quadratus lumborum, psoas and gluteus maximus muscles using CT at L4 upper and lower plates	CSA was significantly reduced in only paraspinal and multifidus at the lower plate in CLBP ($p < 0.01$) CSA was significantly reduced in only multifidus, psoas and quadratus lumborum at the upper plate in CLBP ($p = 0.05$)	Those with previous lumbar surgery were excluded Age and BMI similar in both groups.

			No significant differences between CSA of gluteus maximus	
Sihvonen et al. [94]	LBP patients who underwent surgery for lumbar spinal stenosis and/or disc herniation 2-6 years prior with good recovery, *n* = 14 LBP patients who underwent surgery for lumbar spinal stenosis and/or disc herniation 2-6 years prior regarded as post-operatively failed, *n* = 21	Paraspinal muscle density at L4-L5 level using CT	Muscle density was significantly greater in the group with good recovery compared with the post-operatively failed group ($p < 0.01$)	Lumbar spinal stenosis and/or disc herniation confirmed by CT Age similar between groups
Mooney et al. [101]	Healthy controls, *n* = 8 CLBP patients, *n* = 8	Fatty infiltration and CSA of lumbar paraspinal musculature using MRI from L3 endplate to lower endplate of L5 and graded	CLBP patients showed evidence of fatty infiltration compared with controls 5/8 showing severe	No statistical data reported Patients showed evidence of degenerative disc disease

		using 4 classification system		
			All patients showed greater fatty infiltration of paraspinal muscles compared with any other lumbar muscles	
			No difference in CSA between groups	
Hides et al. [103]	Healthy controls, *n* = 51 First episode acute LBP patients, *n* = 26	CSA of multifidus on left and right sides using real-time ultrasound at L2, L3, L4, L5 and S1	Asymmetry was significantly greater corresponding to level of symptoms in LBP patients compared with normal participant between-side differences ($p < 0.001$)	Only comparisons of between side differences were reported between LBP patients and normal participants. Manual extraction of data on CSA from figure 2 in ref [96] suggests that average CSA of asymptomatic side in LBP patients did not differ significantly from healthy participant's largest side.

				Age, height and weight similar between groups
Mannion et al. [106]	CLBP patients, *n* = 59	CSA of erector spinae, quadratus lumborum and psoas using MRI at L3/L4 and L4/L5 levels	CSA showed association with lean body mass and age, but no association with symptom duration	No healthy control group for comparisons Those with previous lumbar surgery were excluded
Mengiardi et al. [107]	Healthy controls without history of LBP in previous 2 years, *n* = 25 CLBP patients, *n* = 25	CSA of multifidus and longissimus fat content and semi-quantitative grading using 5 classification system using MRI at L4-L5 level	CLBP patients showed significantly greater fat content in the multifidus ($p < 0.05$) No difference found using semi-quantitative system	Those with previous lumbar surgery were excluded Age, sex and BMI matched between participant groups
Cooper et al. [111]	Recent onset LBP patients (symptoms less than 18 months), *n* = 43	CSA of paraspinal and psoas muscles using CT at L4 normalised to L4 bone CSA	Normalised paraspinal and psoas CSAs significantly reduced in CLBP compared to recent onset group ($p < 0.05$)	All participants technically chronic as defined by Frymoyer [108]

	CLBP patients (symptoms more than 18 months), *n* = 44			Lumbar surgery in preceding 18 months were excluded, though most CLBP patients included (*n* = 31) had undergone prior surgery
Bouche et al. [112]	Post-discectomy patients pain free, *n* = 18 Post-discectomy patients with LBP, *n* = 18	Muscle CSA and fat CSA of total paraspinal, erector spinae, multifidus and psoas+iliac muscle using CT at L3, L4, and L5 normalised to L3 bone CSA	Muscle CSA of erector spinae and multifidus significantly smaller in pain patients ($p < 0.05$) Fat CSA significantly greater in psoas of pain patients ($p < 0.05$)	CLBP participants also significantly older Level of operation was not found to be a significant factor and so suggests a general deconditioning of the lumbar musculature independent of surgery Age and BMI similar between groups
Danneels et al. [127]	Healthy controls without history of previous LBP, *n* = 23	Total CSA and muscle CSA of total paraspinal, erector spinae,	Total CSA of paraspinal and multifidus muscles significantly	Those with previous lumbar surgery were excluded in

		multifidus and psoas muscles using CT at upper L3, and upper and lower L4 normalised	smaller at lower L4 in CLBP ($p < 0.05$) No significant difference for erector spinae or psoas	addition to those who had participated in training for the lower back muscles in the previous 3 months Age, height, weight and activity similar between groups
Alaranta et al. [113]	CLBP patients, $n = 39$	Fat content of lumbar paraspinal musculature using CT at three lowest levels and 4 level classification system	Fat content was moderately positively associated with disability score on Oswestry index ($p < 0.05$) but not with age, sex, body mass, BMI, degree of disc degeneration, or facet joint osteoarthritis	Those with previous spinal fusion surgery were excluded however 16 patients had undergone previous surgery for lumbar disc herniation >1 year prior.
Kader et al [114]	CLBP patients, $n = 75$	Atrophy of the multifidus compared with normal results from [106] using MRI and 3 level classification system	80% of participants showed moderate of severe multifidus atrophy	Those with previous lumbar surgery were excluded Significant association between multifidus atrophy and leg pain

				($p < 0.01$)
Barker et al. [115]	CLBP patients with unilateral pain, $n = 50$	CSA of left and right multifidus and psoas muscles using MRI at level of symptoms and one level above and below	CSA of both multifidus and psoas significantly smaller on symptomatic side at all levels ($p < 0.05$)	Those with previous lumbar surgery were excluded Multifdus atrophy consistently relatively greater than psoas atrophy at all levels Significant association between psoas atrophy and pain, nerve root compression and symptom duration. Significant association between multifidus atrophy and symptom duration
Kjaer et al. [116]	Adults aged 40 years, $n = 409$ (85% reporting LBP ever, 70%	Fat content of multifidus using MRI at 3 lower lumbar levels	Association between fat content of multifidus for LBP ever	Associations increased when controlling for effect moderators

	reporting LBP in previous year) Adolescents aged 13 years, *n* = 439 (41% reporting LBP ever, 22% reporting LBP in previous year)	using 3 level classification system	(Odds Ratio = 7.2) and LBP in previous year (Odds Ratio = 3.6) in adults. No association between fat content of multifidus in adolescents	including gender, BMI, physical workload, leisure and sports activities.
Hyun et al. [117]	Healthy controls without lumbosacral radiculopathy or disc herniation, *n* = 19 LBP patients with unilateral lumbosacral radiculopathy, *n* = 14 LBP patients with disc herniation but no lumbosacral radiculopathy, *n* = 25	Total CSA and muscle CSA of mulitifidus using MRI at L3/L4, L4/L5, and L5/S1	Total CSA, muscle CSA and ratio of the two were significantly reduced in both LBP groups involved sides compared to controls at most levels ($p < 0.05$) and ratio at L3/L4 ($p < 0.05$) No difference between LBP groups for total CSA, muscle CSA and ratio of the two	Those with previous lumbar surgery were excluded from LBP control group

			Ratio of involved side CSA to uninvolved side CSA was significantly different in radiculopathy patients compared to both controls and the other LBP patients ($p < 0.01$ to 0.05)
Kalichman et al. [118]	Healthy controls without LBP in previous year, $n = 150$	Density of erector spinae and multifidus muscles using CT at L3, L4, and L5	Muscle density was not associated with LBP
	Patients who had suffered from LBP of at least 1 month within previous year, $n = 37$		Reduced muscle density was significantly associated with presence of facet joint osteoarthritis, spondylolisthesis and disc narrowing ($p < 0.05$)
Hicks et al. [119]	Controls aged 70-79 years without LBP in previous year, $n = 861$	Total CSA and density of paraspinal, and lateral abdominal muscles using CT at L4-L5 level	Both non-adjusted and adjusted means for muscle density showed significant associations with the presence and severity of

	Patients with mild LBP in previous 12 months, *n* = 244 Patients with moderate LBP in previous 12 months, *n* = 299 Patients with severe/extreme LBP in previous 12 months, *n* = 111		LBP for the paraspinal muscles ($p \leq 0.0001$), and lateral abdominals ($p < 0.05$).	
Kang et al.[128]	CLBP patients with lumbar degenerative kyphosis undergoing corrective surgery, *n* = 54 CLBP control patients, *n* = 54	CSA and muscle to disc CSA ratio of psoas, erector spinae and multifidus was assessed at L4/L5 level and fatty infiltration of psoas, erector spinae and multifidus assessed at L3/L4 using three grade classification using MRI	CSA and muscle to disc CSA ratios for all muscles were significantly lower in the lumbar degenerative kyphosis group compared with controls ($p < 0.001$) with regression analysis showing multifidus wasting to be most strongly associated ($p < 0.001$)	No healthy control group for comparisons Those with previous lumbar surgery were excluded from CLBP control group Age and sex matched between groups and symptom durations

				were similar
			Severe fatty infiltration was significantly more common in lumbar degenerative kyphosis compared to CLBP controls ($p < 0.05$)	Body mass and BMI was significantly higher in CLBP controls No difference in degenerative changes (degenerative disc disease, herniation's, stenosis or spondylolithesis) between groups
Histochemical Studies				
Crossman et al [77]	Healthy controls without LBP lasting >3 days in previous 12 months, $n = 32$	Percutaneous biopsy of paraspinal muscle (specific location not noted) for fibre CSAs and fibre typing.	No significant differences between groups for any fibre histochemical comparisons	Those with previous lumbar surgery were excluded Age, gender and all

	CLBP patients, *n* = 35			anthropometric characteristics similar between groups
Weber et al. [92]	LBP patients undergoing posterior surgery, *n* = 61 (posterior surgery for persistent pain Op1 *n* = 43, posterior surgery for removal of internal fixation Op2 *n* = 32)	Biopsy of multifidus at L3, L4, L5 or S1 level for fibre diameter, fibre typing and pathological changes	Pathological changes were common in biopsy specimens from Op1 Type II atrophy was associated with age and severity of pain in biopsy specimens from Op1 Patients undergoing Op2 showed significantly greater pathological changes compared with biopsy specimens from Op 1 ($p = 0.05$) Biopsy specimens were taken from 14 patients at the same level in both Op1 and Op2 with	No healthy control group for comparisons Muscular alterations were present in patients undergoing Op1 however surgery may have caused further alterations as presence of changes were increased in Op2

			70% of normal biopsies at Op1 showing alterations at Op2	
Rantanen et al [93]	Patients from ref [123] who underwent surgery for lumbar disc herniation 5 years prior, *n* = 18	Biopsy of multifidus taken 1cm laterally from spinous process of the level immediately below the previously herniated disc (L4/L5 and/or L5/S1) for fibre narrow diameter, fibre typing, atrophy/hypertrophy and pathological changes	No changes in fibre type distribution, atrophy/hypertrophy factors were noted compared with baseline Type I fibre size significantly increases	Level of herniation and thus biopsy did not influence results Patients with both 'positive' and 'negative' outcomes from original surgery were compared showing decreased pathological changes in 'positive' group compared with their persistence in 'negative' group
Sihvonen et al. [94]	LBP patients who underwent surgery for lumbar spinal stenosis and/or disc herniation 2-6 years prior with good recovery, *n* = 14	Biopsy of paraspinal muscle taken from site of abnormal myelogram finding for fibre atrophy	Local denervation atrophy observed in all but one post operatively failed patients	Lumbar spinal stenosis and/or disc herniation confirmed as absent by CT Age similar between groups
	LBP patients who underwent			No statistical data reported

	surgery for lumbar spinal stenosis and/or disc herniation 2-6 years prior regarded as post-operatively failed, *n* = 21			
Mannion et al. [106]	CLBP patients, *n* = 59	Biopsy of belly of lateral tract of left erector spinae at L3/L4 level for fibre CSA, fibre typing and pathological changes	Symptom duration was a strong predictor of both fibre type changes towards a more type IIx phenotype Pathological changes were common and significantly associated with age and showed a trend to association with symptom duration	No healthy control group for comparisons Those with previous lumbar surgery were excluded
Ford et al. [109]	Patients undergoing surgery for lumbar disc herniation reporting LBP duration between 3 and 52 weeks, *n* = 18	Biopsy of erector spinae (sacrospinalis) 1cm lateral to tip of spinous process and multifidus 1cm from inferior border of lamina at L5 level for	No differences between left and right sides Pathological changes were common but varied and not	No healthy control group for comparisons Side of herniation did not affect results

		fibre typing, fibre narrow diameter and pathological changes	impacted by side of herniation	
Zhu et al. [120]	Patients undergoing surgery for lumbar disc herniation, *n* = 22	Biopsy of erector spinae from side and level of herniation 1cm lateral to top of spinous process for fibre typing, atrophy/hypertrophy and pathological changes	Proportion of fibres types for type I, type IIa and type IIb were 68%, 10.6% and 21.4% respectively Type II atrophy was common with type IIb most frequent and severe 18 patients showed evidence of pathological changes	No healthy control group for comparisons
Mannion et al. [121]	Healthy controls without history of LBP requiring time of work or doctors attention, *n* = 29 CLBP patients undergoing	Biopsy of belly of lateral tract of left erector spinae at L3 level for fibre narrow diameter, fibre typing and pathological changes	Smaller proportion of type I and greater proportion of type IIb fibres as both % and % fibre type area were found in CLBP patients compared to healthy	Age, sex and body mass matched between participant groups

	posterior surgery, *n* = 31 (First time operation *n* = 22, patients undergoing second operation *n* = 9)		controls ($p < 0.05$) Pathological changes did not differ between groups	
Fidler et al. [122]	Patients with LBP, *n* = 17 Cadavers within 24 hours of death, *n* = 3	Biopsy of multifidus from separated muscle cut transversely, taken during operation	Grouping of slow fibres appeared in addition to reduced CSA of fast fibres in LBP	No details on nature of operation No statistical data reported
Mattila et al [123]	Patients undergoing first time surgery for lumbar disc herniation, *n* = 41 Control participants without history of LBP undergoing autopsy within 48 hours of death, *n* = 12	Biopsy of multifidus taken during operation or autopsy at L4/L5 and L5/S1 levels for fibre narrow diameter, fibre typing, atrophy/hypertrophy and pathological changes	Relative numbers of type I and type II fibres did not correlate with age nor differ significantly between groups Pathological changes were significantly more frequent in patients compared to controls ($p < 0.01$)	Biopsy taken from deltoid to rule out systemic congenital myopathy Level of herniation and thus biopsy did not influence results
Zhao et al. [124]	LBP patients undergoing first	Biopsy of multifidus taken	CSAs and diameters of both	No healthy control group for

	time surgery for lumbar disc herniation, *n* = 19	during operation from transversospinal corner on both left and right sides at the level of herniation (L4/L5 or L5/S1) for fibre CSA, fibre narrow diameter, fibre typing and pathological changes	type I and type II fibres were significantly smaller on the side of herniation ($p < 0.05$) Strength factor (%fibre type x fibre CSA) of type II fibres was also lower on side of herniation ($p < 0.05$) Pathological changes were present in both sides but more severe on the side of herniation	comparisons Location of pain symptoms was associated with muscle alterations
Bajek et al. [125]	Patients undergoing surgery for lumbar disc herniation, *n* = 76 Control participants without history of neuromuscular disease undergoing autopsy within 48 hours of sudden death, *n* = 41	Biopsy of multifidus on side of herniation and at level of herniation in patients (L3/L4, L4/L5, or L5/S1) and L4/L5 level in controls 1cm lateral from midline deeper than the aponeurosis of erector spinae for	Greater proportion of type I and smaller proportion of type IIa type IIb fibres in patients compared with controls in males only ($p < 0.05$) Fibre diameter in type I fibres	Age was similar between groups

		fibre typing and fibre diameter	was significantly greater in paitents compared to controls ($p < 0.05$) and for type IIa and type IIb was significantly greater than controls for males only ($p < 0.05$)	
Yoshihara et al. [126]	LBP patients undergoing first time surgery for lumbar disc herniation, $n = 29$	Biopsy of multifidus taken during operation immediately after start of surgery dissected from L4 and L5 muscle bands on both sides for fibre typing, fibre size and pathological changes	Fibre size of type 2 fibres was significantly smaller than type I at all biopsy sites Fibre size did not differ between sides at L4 for type I or type II fibres but fibre size was significantly smaller at L5 on side of herniation for both type I and type II fibres ($p < 0.01$) No difference in fibre type proportions	No healthy control group for comparisons No difference between level of biopsy

			Pathological changes were present at all biopsy sites but only significantly different between sides, with greater frequency on side of herniation at L5	

Table 3. Summary of studies testing fatigability with EMG of the lumbar extensor musculature in LBP

Reference	***Participants***	***Testing***	***Results***	***Comments***
Kankaanpaa et al. [37]	Healthy controls without history of LBP, *n* = 15 Middle aged women with CLBP, *n* = 20	EMG recorded bilaterally from gluteus muscles and lumbar paraspinal muscles at L3/L4 and L5/S1 levels 2cm laterally from midline of spinous process during isometric MVC and isometric endurance to failure at 50%MVC during seated (knees	Neither EMG amplitude or fatigue indices data differed between groups for the paraspinal muscles	Those with previous lumbar surgery were excluded Age, height, body mass and BMI similar between groups

		90°) restrained trunk extension		
Lariviere et al. [40]	Healthy controls without LBP lasting 1 wk in previous year, *n* = 18 CLBP patients, *n* = 18	EMG recorded bilaterally from gluteus maximus, biceps femoris and vastus medialis muscles and lumbar paraspinal muscles at L4, L3, L1, and T10 levels during isolated lumbar extension MVC and repetitions to failure at 60%MVC using customised dynamometer	None of the EMG fatigue indices data differed between groups for the paraspinal muscles	Those with previous lumbar surgery were excluded Age, body mass, height, BMI, body % and physical activity levels were similar between groups
Crossman et al [77]	Healthy controls without lasting >3 days in previous 12 months, *n* = 32 CLBP patients, *n* = 35	EMG recorded biltaerelly from lumbar paraspinal muscles at L4-L5 level during standing isometric trunk extension for 60 seconds at 60%MVC and during the Biering-Sorensen test	EMG fatigue indices were similar between groups for the Biering-Sorenson test and also the 60%MVC test	Those with previous lumbar surgery were excluded Age, gender and all anthropometric characteristics similar between groups

Paasuke et al. [78]	Healthy controls without history of LBP or LBP in previous year, *n* = 12 CLBP patients, *n* = 12	EMG recorded bilaterally from lumbar paraspinal muscles at L3 level 3cm from midline during Biering-Sorenson test to failure	EMG indices of fatigue showed significantly greater fatigue in the CLBP group compared to controls ($p < 0.05$)	Those with previous lumbar surgery were excluded Age and gender matched between participant groups Age, height, body mass and BMI were similar between participant groups
Humphrey et al. [79]	Healthy controls without history of LBP in previous 5 years, *n* = 175 CLBP patients, *n* = 145 Participants with past history of LBP but no attack within previous 2 years, *n* = 30	EMG recorded bilaterally from lumbar paraspinal muscles at L4/L5 during a back lift test with 66.66%MVC for 30 seconds	EMG indices of fatigue showed significantly greater fatigue in the CLBP compared to controls ($p < 0.05$) Logistic regression showed high sensitivity (0.65) and specificity (0.75) in classifying CLBP patients	Those with previous lumbar surgery were excluded CLBP group was significantly older and had higher body mass and BMI than controls

			Past history participants could not be adequately discriminated from either group	
Suuden et al. [80]	Healthy controls, *n* = 20 CLBP patients, *n* = 20	EMG recorded bilaterally from lumbar paraspinal muscles at L3 3 cm from midline during Biering-Sorenson test to failure	No significant differences in EMG indices of fatigue between groups	Those with previous lumbar surgery were excluded Age, height and weight and BMI similar between groups
Lariviere et al. [81]	Healthy controls without history of LBP in previous year, *n* = 18 CLBP patients, *n* = 18	EMG recorded bilaterally from gluteus maximus, biceps femoris and lumbar paraspinal muscles at L4, L3, L1, and T10 levels during dynamic roman chair trunk extensions to failure	No significant differences in EMG indices of fatigue between groups	Those with previous lumbar surgery were excluded Age, height, weight and BMI similar between groups
Suter & Lindsay [87]	Healthy controls without history of LBP, *n* = 16	EMG recorded bilaterally from lumbar paraspinal muscles at T12 and L4-L5 level 3cm from	No significant difference in EMG fatigue indices between groups	Age, height and weight similar between groups

	Golfers with CLBP, *n* = 25	midline during Biering-Sorenson test to failure		
Da Silva et al. [88]	Healthy controls without history of LBP in previous year, *n* = 15	EMG recorded bilaterally from lumbar paraspinal muscles at T10, L1, L3, and L5 levels	No difference in EMG fatigue indices between groups	Those with previous lumbar surgery were excluded
	CLBP patients, *n* = 13	during standing trunk extension and back lift at 50%MVC for 60 seconds, and during Biering-Sorenson test for 60 seconds		Age, height and weight similar between groups
Lariviere et al. [89]	Healthy controls without LBP lasting 1 wk in previous year, *n* = 31	EMG recorded bilaterally from gluteus maximus, biceps femoris and lumbar paraspinal muscles at L5, L3, L1, and T10 levels	EMG indices of fatigue showed significantly greater fatigue in CLBP patients with high catastrophising compared with CLBP patients with low catastrophising ($p < 0.01$)	Those with previous lumbar surgery were excluded
	CLBP patients, *n* = 27	during standing trunk extension/flexion MVC and repetitions to failure (endurance time) with stabilised knees and lower back		Age, height, weight and BMI similar between groups
Robinson et al. [98]	Healthy controls never treated	EMG recorded bilaterally from	EMG amplitude in millivolts	Age, height and weight similar

	for LBP and without LBP in previous year, *n* = 12 CLBP patients (53% having had previous surgery), *n* = 16	lumbar paraspinal muscles at L1-L2 level during isolated lumbar extension at 60%MVC in full extension for 12-13 repetitions	decreased across repetitions in asymptomatic participants compared with a significantly flatter curve in the CLBP group ($p < 0.05$)	between groups
Roy et al. [135]	Healthy controls, *n* = 12 CLBP patients, *n* = 12	EMG recorded bilaterally from lumbar paraspinal muscles at L1, L2 and L5 levels during standing isometric trunk extension for 60 seconds at 40%MVC, 60%MVC and 80%MVC	Discriminant analysis of EMG fatigue indices successfully classified 92% controls, 82% CLBP at 40%MVC, 67% controls, 75% CLBP at 60%MVC and 84% controls, 91% CLBP at 80% MVC	Those with previous lumbar surgery were excluded Age, height and weight similar between groups
Roy et al. [151]	Healthy controls without history of LBP, *n* = 42 CLBP patients (43% having had previous surgery), *n* = 28	EMG recorded bilaterally from lumbar paraspinal muscles at L1, L2 and L5 levels during standing isometric trunk extension for 30 seconds at 40%MVC and 80%MVC	Discriminant analysis of EMG fatigue indices successfully classified 85% CLBP patients and 86% healthy controls	CLBP patients heterogeneous with respect to symptoms and history (75% had disc herniation and 43% had undergone previous surgery)
Mayer et al. [154]	Healthy controls, *n* = 11	EMG recorded bilaterally from	EMG indices of fatigue showed	Those with previous lumbar

	CLBP patients, *n* = 10	lumbar paraspinal muscles at L3 level 3cm from midline during 10 isometric trunk extension holds on a roman chair lasting 15 seconds each and with 10 seconds rest between each hold	significantly greater fatigue in the CLBP group compared to controls ($p < 0.01$)	surgery at level of EMG placement were excluded Age and torso weight similar between groups
Peach & McGill [155]	Healthy controls without history of LBP in previous 2 years, *n* = 18 CLBP patients, *n* = 21	EMG recorded from lumbar paraspinal muscles at T9 level 5cm from midline, L3 level 3cm from midline, and L5 level 1-2cm from midline respectively during semi-standing isometric trunk extension for 30 seconds at 60%MVC and then after a 60 second rest during a further 10 second extension at 60%MVC	EMG indices of fatigue showed significantly greater fatigue in the CLBP compared to controls ($p < 0.05$) Discriminant analysis of EMG fatigue indices successfully classified 100% controls and 93.75% CLBP patients Logistic regression was equally powerful using two parameters	Age, height and weight similar between groups

			with concordance of 92.4%	
Roy et al. [156]	Varsity rowers without LBP, *n* = 17 Varsity rowers with LBP in past year, *n* = 6	EMG recorded bilaterally from lumbar paraspinal muscles at L1, L2 and L5 levels during standing isometric trunk extension for 30 seconds at 80%MVC and then after a 60 second rest during a further 5 second extension at 80%MVC	Discriminant analysis of EMG fatigue indices successfully classified 93% controls and 100% LBP participants	Age, height and weight similar between groups
Biedermann et al. [157]	Healthy controls without history of LBP, *n* = 22 CLBP patients, *n* = 27	EMG recorded bilaterally from lumbar paraspinal muscles at L2-L3 and L4-L5 levels during standing with a 11.6 pound dumbbell held in outstretched arms for 45 seconds followed by a 5 minute recovery and the repetition of the 45 second trial – all adjusted for arm length differences	CLBP patients were classified into 'avoiders' or 'confronters' Discriminant analysis of EMG fatigue indices successfully classified 88.9% 'avoiders', 66.7% 'confronters' and 59.1% controls	Age, height, weight and arm length similar between groups Continuum of fatigue seen between avoiders>confronters>controls, however pain duration differed significantly between avoiders and confronters (8.57±6.22 years and 1.60±0.76 years

				respectively)
Klein et al. [158]	Varsity rowers without LBP, *n* = 17 Varsity rowers with LBP in past year, *n* = 8	EMG recorded bilaterally from lumbar paraspinal muscles at L1, L2 and L5 levels during standing isometric trunk extension for 30 seconds at 80%MVC and then further 10 second extensions at 80%MVC at 1 minute, 2 minutes, 5 minutes, 10 minutes and 15 minutes into recovery	Discriminant analysis of EMG fatigue indices showed most successful classification at 1 and 2 minute recovery, classifying for 1 and 2 minutes respectively 88% and 100% of LBP participants and 100% and 88% of controls	Age, height and weight similar between groups
Mannion et al. [159]	Healthy controls without history of LBP, *n* = 10 LBP patients, *n* = 12	EMG recorded bilaterally from lumbar paraspinal muscles at T10 and L3 level 3-4cm from midline during Biering-Sorenson test for 60 seconds	MFS was greater in LBP group indicating greater fatigue but just failed to achieve significance ($p = 0.10$)	Age, height and weight similar between groups Mean values for MFS were similar to those in prospective study which did achieve significance in predicting first time LBP

Table 4. Summary of prospective studies of lumbar extensor musculature deconditioning in LBP

Reference	*Participants*	*Testing*	*Results*	*Comments*
Biering-Sorenson [68]	Men aged between 30, 40, 50, and 60 years old, *n* = 449 Women aged between 30, 40, 50, and 60 years old	Biering-Sorensen test conducted at baseline 1 year follow-up with questionnaire concerning first time occurrence, recurrence or persistence of LBP	First time occurrence was significantly associated with low endurance time	
Leino et al. [69]	*Baseline participants* Participants with "Good" low back status, *n* = 578 Participants with "Intermediate" low back status, *n* = 260 Participants with "Bad" low back status, *n* = 64	Standing dynamic trunk extension/flexion maximum repetitions performed over 30 seconds with buttock and thighs against a supporting plate and ankles tied by a belt conducted at baseline Standing isometric trunk extension/flexion MVC with	Trunk strength was not predictive of low back symptoms or status at follow up.	

		buttock and thighs against a supporting plate and ankles tied by a belt conducted at 10 year follow-up in addition to questionnaire and assessment of low back symptoms and status	
	Follow-up participants Participants with "Good" low back status, *n* = 239 Participants with "Intermediate" low back status, *n* = 203 Participants with "Bad" low back status, *n* = 210		
Luoto et al. [83]	Healthy participants without history of LBP in previous year at baseline, *n* = 167	Biering-Sorensen test and questionnaire regarding previous and present LBP conducted at baseline 75% of participants were available for follow-up at 1 year	Endurance time was significantly associated with first time occurrence of LBP when adjusted for age, sex and occupation ($p < 0.05$) Endurance time broken into

		with the same questionnaire, *n* = 126	tertiles (poor, medium, good) showed a non- linear dose-response relationship with first time occurrence of LBP ($p < 0.04$)
			Relative odds ratio compared to 'good' for 'medium' and 'poor' were1.4 (95% CI 0.4 - 4.2) and 3.4 (95% CI 1.2 – 10.0) respectively
Gibbons et al. [84]	Healthy participants without history of LBP in previous year at baseline, *n* = 43	Isokinetic back lift MVC, psychophysical back lift test, Biering-Sorensen test, CSA, proton-density weighted signal, and T2-weighted signal of erector spinae, quadratus lumborum, psoas major and total paraspinal muscle using	Neither back lift, psychophysical back lift or endurance time differed between those with and without LBP at follow-up, nor where they associated with frequency of LBP at follow-up

		MRI, and interview regarding previous and present LBP conducted at baseline Interviews regarding LBP were conducted at 1 year follow-up	Neither CSA, proton-density weighted signal, or T2-weighted signal differed between those with and without LBP at follow-up, however, total paraspinal CSA, and proton-density weighted signal andT2-weighted signal of erector spinae, quadratus lumborum, psoas major were significantly associated with frequency of LBP at follow-up ($p < 0.05$)
Mannion et al. [159]	Healthy nurses without history of LBP, $n = 200$	EMG recorded bilaterally from lumbar paraspinal muscles at T10 and L3 level 3-4cm from midline during Biering-Sorenson test and maintenance of 80%MVC for 28 seconds at	13% developed serious first time LBP during the follow-up period EMG indices of fatigue during Biering-Sorenson showed

		baseline Postal questionnaire regarding LBP conducted at 1 year follow-up	greater fatigue was significantly associated with development of first time LBP at follow-up ($p < 0.05$) however endurance time was not associated with first time LBP
Rissanen et al. [163]	Participants from the Mini-Finland Health Survey, $n = 535$	Dynamic trunk extension/flexion maximum repetitions performed over 30 seconds with buttock and thighs against a supporting plate and ankles tied by a belt conducted at baseline Average 12 year follow-up to time until retirement due to work disablement, death or end of observation period for	At follow-up of 56 incident cases 15 were due to back disorders Adjusted relative risks in multiple models showed trunk extension performance significantly predicted back disorder disability risk ($p = 0.04 – 0.002$)

		primary diagnosis as cause of work disability		
Newton et al. [167]	Healthy participants without history of LBP, *n* = 70	Isokinetic trunk extension, flexion, rotation, and back lift MVC and psychophysical lift conducted at baseline	23% developed LBP during the follow-up period, yet at least 6 months after initial assessment in all cases	Those with previous lumbar surgery were excluded
		1 year follow-up with questionnaire concerning first time occurrence, recurrence or persistence of LBP	None of the isokinetic measures differed between those who did and those who did not develop LBP	
Reimer et al. [168]	Healthy prospective order selector employees for 1989, *n* = 122 Healthy prospective order selector employees for 1990, *n* = 122	Dynamic lift capacity, isokinetic trunk extension, flexion, rotation, and back lift MVC and psychophysical lift conducted at baseline to determine placement in employment as an order selector in a warehouse grocery distributor	After implementation of prospective evaluation for employment placement in 1989, incidence of low back injuries were significantly reduced by 32% in 1990 and 41% in 1991 ($p < 0.001$)	

	Healthy prospective order selector employees for 1991, *n* = 122	2 year follow-up with questionnaire concerning first time occurrence, recurrence or persistence of LBP		
Batt'ie et al. [169]	Employees working for a large aircraft manufacturer (n = 497 reporting LBP in previous 10 years), *n* = 2178	Isometric MVC for torso, arm and leg lift was conducted at baseline 4 year follow-up conducted for claims related to low back injuries or LBP	Participants with higher MVC for arm, leg and torso lift were at higher risk for LBP and low back injury (p = 0.01, 0.03, and 0.26 respectively). When adjusted for age and sex however no association was present.	Due to an injury rate of 0.6% during torso lift testing it was discontinued. *n* = 495 participants completed torso lift testing, *n* = 2158 completed arm lift testing, and *n* = 2102 completed leg lift testing
Lee et al. [170]	Healthy student participants without history of LBP, *n* = 67	Isokinetic trunk extension, flexion, and rotation MVC conducted at baseline. 5 year follow-up concerning	27% developed first time LBP during the follow-up period Ratio of extension/flexion strength at baseline was	Age, height, weight and smoking habits similar between groups

		LBP incidence	significantly lower in participants who developed first time LBP, ($p < 0.05$)	
Kujala et al. [171]	Healthy participants without history of LBP, $n = 262$	Standing isometric trunk extension/flexion MVC was conducted at baseline 5 year follow-up with questionnaire was conducted regarding type, frequency, severity and functional limitations of LBP	47% developed first time LBP during the follow-up period, 11% of these reporting it as being of monthly frequency, 17% reporting radiating limb pain, and 2% having been hospitalised due to LBP Trunk extension/flexion was not associated with development of first time LBP	Age, weight and BMI similar between groups Height, occupational physical demands, and occupational musculoskeletal loading was significantly associated with first time LBP ($p < 0.05$)
Chaffin [172]	Pre-employed plant workers in a variety of jobs involving manual lifting, $n = 551$	Isometric MVC for torso, arm and leg lift in addition to job specific demands was conducted at baseline	As job strength requirements exceeded participant strength the incidence and severity of low back injuries increased at a	

			ratio of 3:1 across the tertiles	
		Preventative effectiveness of strength relative to job demands were evaluated by examining incidence and severity of low back injuries over an 18 month follow-up period Participants were grouped into tertiles relating to their individual strength relative to their job demands		
Keyserling [173]	Pre-employed plant workers applying for a range of 20 varied jobs, *n* = 71	Isometric MVC for torso and arm lift, and push in/out in addition to job specific demands was conducted at baseline Preventative effectiveness of	During the follow-up period the control group experienced 19 incidences of musculoskeletal injuries compared to 0 in the experimental group	Age, weight and height similar between groups

		strength relative to job demands evaluated by placing of experimental (n = 20) group into jobs matching strength whereas control group (n = 51) were not Incidence of musculoskeletal injuries were evaluated over a 1 year follow-up period		
Salminen et al. [174]	Healthy children, n = 38 Children with LBP, n = 31 Children with LBP and sciatica, n = 7	Biering-Sorensen test, sit up isometric test with knees at 90^0 and MRI conducted at baseline 3 year follow-up period evaluating LBP ever, LBP in past 12 months, and recurrent/continuous LBP	Both flexion and extension endurance times were significantly lower in LBP groups ($p < 0.05$) at baseline and follow-up yet endurance time was not predictive of development of first time LBP	Age, sex, school matched between groups

Sjolie & Ljunggren [175]	Healthy adolescents, *n* = 86	Biering-Sorensen test and questionnaire regarding LBP conducted at baseline 3 year follow-up period with questionnaire was completed	High mobility /endurance time ratios were significantly associated with development of LBP at follow-up when adjusted for gender, LBP at baseline, and well-being and physical activity at follow-up (OR 1.5 - 1.9, 95% CI 1.1 – 3.2, $p < 0.05$)
Adams et al. [176]	Healthy nurses without history of LBP, *n* = 262 Healthy nurses who had previously suffered with 'non-serious' LBP, *n* = 141	Biering-Sorenson tets, isometric back lift MVC and back lift at 80%MVC for 20 seconds while EMG recorded from T10 and L3 conducted at baseline 3 year follow-up (every 6 months) conducted using questionnaire regarding LBP in previous 6 months	Endurance time at 3 year follow-up was significantly associated with development of serious LBP ($p < 0.01$) and approached significance for any LBP ($p < 0.058$) Neither back lift nor indices of fatigue were associated with development of LBP

Mostardi et al. [177]	Healthy nurses without history of LBP, *n* = 171	Isokinetic back lift MVC conducted at baseline Injury reports used to examine incidence of low back injury over 2 years follow-up	9% sustained low back injuries during the follow-up period There was no significant difference in strength at baseline between those who reported low back injury during follow-up and those who did not	
Cady et al. [178]	Healthy fire-fighters without LBP, *n* = 1652	Isometric back lift MVC conducted at baseline Incidence of prior low back injuries examined subsequent to baseline measurements – no specific follow-up duration was noted	7.14% sustained low back injuries in the 'Least Fit' group, 3.19% sustained low back injuries in the 'Middle Fit' group, and 0.77% sustained low back injuries in the 'Most Fit' group	Mean age increased with decreasing fitness levels between the three groups

		Participants were split into percentiles for 'Most Fit' (84-100 percentile), 'Middle Fit' (17-83 percentile) and 'Least Fit' (0-16 percentile)		
Mooney et al. [179]	Workers without history of LBP in a ship-building firm in the 3 highest Physical Demand Characteristic categories across 32 jobs, *n* = 152	Isolated lumbar extension MVC using MEDX 2 year follow-up of low back injury and LBP claims	9% sustained low back injuries during the follow-up period the majority occurring in the heavy PDC category (64%) Isolated lumbar extension strength was not predictive of low back injuries and only 2 of those participants injured had below normal strength	Age, height and weight was similar amongst PDC categories and in those injured and uninjured Low back injury rates were significantly higher in heavy and very heavy PDC categories ($p < 0.0001$)
Stevenson et al. [181]	Spinning operators from DuPont without history of LBP, *n* = 72	EMG recorded bilaterally from lumbar paraspinal muscles at T10 and L3 level 3-4cm from	EMG indices of fatigues entered final model and were significantly predictive of LBP	Other factors in final predictive model included age, peak thoracic acceleration, leg

	Spinning operators from DuPont suffering from LBP in previous 2 years, *n* = 46	midline during Biering-Sorenson test	(p = 0.035)	strength/ endurance, however psychosocial factors were largely absent.
	Spinning operators from DuPont suffering from LBP in previous year, *n* = 31	2 year follow-up period at 6 month intervals for LBP experiences in previous 6 months		
Heydari et al. [182]	Healthy participants classified as either 'No History of LBP', 'CLBP' or 'Past History of LBP', *n* = 105	EMG recorded bilaterally from lumbar paraspinal muscles at L4/L5 level during back lift test maintaining 2/3MVC for 30 seconds at baseline and follow-up	At follow-up 76 classified themselves as 'the same', 13 'better' and 16 'worse'	
		2 year follow-up participants were asked to classify themselves as 'worse', 'better, or 'the same'	EMG indices of fatigue showed greater fatigue was significantly associated with development of first time LBP and with self-classification at follow-up (p < 0.05)	