


**Please cite the Published Version**

Steele, J, Fisher, J, Perrin, C, Conway, R, Bruce-Low, S and Smith, D  (2019) Does change in isolated lumbar extensor muscle function correlate with good clinical outcome? A secondary analysis of data on change in isolated lumbar extension strength, pain, and disability in chronic low back pain. *Disability and Rehabilitation*, 41 (11). pp. 1287-1295. ISSN 0963-8288

**DOI:** <https://doi.org/10.1080/09638288.2018.1424952>

**Publisher:** Taylor & Francis

**Version:** Accepted Version

**Downloaded from:** <https://e-space.mmu.ac.uk/620347/>

**Usage rights:**  In Copyright

**Additional Information:** This is an Author Accepted Manuscript of a paper accepted for publication in *Disability and Rehabilitation*, published by and copyright Taylor & Francis.

**Enquiries:**

If you have questions about this document, contact [openresearch@mmu.ac.uk](mailto:openresearch@mmu.ac.uk). Please include the URL of the record in e-space. If you believe that your, or a third party's rights have been compromised through this document please see our Take Down policy (available from <https://www.mmu.ac.uk/library/using-the-library/policies-and-guidelines>)

Title: Does Change in Isolated Lumbar Extensor Muscle Function Correlate with Good Clinical Outcome? A Secondary Analysis of Data on Change in Isolated Lumbar Extension Strength, Pain and Disability in Chronic Low Back Pain

Authors: James Steele PhD<sup>a</sup>, James Fisher MSc<sup>a</sup>, Craig Perrin BSc<sup>a</sup>, Rebecca Conway BSc<sup>a</sup>, Stewart Bruce-Low PhD<sup>a</sup>, Dave Smith PhD<sup>c</sup>,

<sup>a</sup>School of Sport, Health and Social Science, Southampton Solent University, Southampton

<sup>b</sup>Department of Exercise and Sport Science, Manchester Metropolitan University, Manchester

<sup>c</sup>Anglo European Chiropractic College, Bournemouth

Statement of Institutional Review Board:

All studies reported in this manuscript had received ethical approval from the ethics committee at the author's institution.

Corresponding author:

James Steele ([james.steele@solent.ac.uk](mailto:james.steele@solent.ac.uk))

School of Sport, Health and Social Science

Southampton Solent University,

East Park Terrace,

Southampton

Hampshire,

SO14 0YN

## **Introduction**

Chronic low back pain (CLBP) is one of the most prevalent medical conditions in today's societies (NICE, 2009; ONS, 2000; Waddell & Burton, 2001; Walker, 2000; WHO, 1998) representing a total economic cost amounting to billions worldwide (Ekman et al., 2005; Freburger et al., 2009; Guo et al., 1999; Katz, 2006; Maniadakis & Graym 2000; NICE, 2009; Ricci et al., 2006; Stewart et al., 2003; Van Tulder et al., 1995; Waddell et al., 2002). Exercise is a common prescription for CLBP though previous Cochrane reviews have generally reported small effect sizes with respect to most exercise approaches, reflecting either low average outcomes or high variability in outcomes (Van Tulder et al., 2000; Hayden et al., 2005). However, these typically consider 'exercise' as a single class of treatment without consideration to the variation in exercise approaches that exist. In general, these reviews have not adequately described, defined and categorised the 'exercise' studies they have examined, potentially explaining the generally inauspicious conclusions drawn have been specifically criticised for this flaw and their wide-sweeping conclusions (Van Tulder et al., 2000; Hayden et al., 2005; Manniche & Jordan, 2001; Manniche & Jordan, 2001). Though, this may be because many empirical studies of exercise in CLBP lack an adequate description of the precise exercises used (Helmhouit et al., 2008; Mayer et al., 2008). A more recent systematic review has instead looked to examine broadly the impact of different exercise types, reporting that resistance training and motor control type exercise approaches appear to offer the greatest benefits (Searle et al., 2015).

Both of these exercise approaches are commonly aimed at improving different functional deficits that are thought to present, and potentially play a role in, CLBP. Motor control exercise is aimed at improving the ability of the neuromuscular system to control specific movement quality and/or create stability, whereas resistance training approaches are often aimed at improving more general components of neuromuscular function such as strength and endurance. Indeed, theories regarding the mechanisms of action that are often offered to explain the benefits of exercise can be roughly grouped as being mechanical, such as those

described above, neural (e.g. desensitisation), or cognitive and/or operant conditioning based (Helmhout et al., 2008). Motor control and resistance exercise based approaches are effective in producing positive clinical outcomes in CLBP (Searle et al., 2015), though it has been questioned whether the changes in function they produce are indeed responsible or even related to the changes in pain and/or disability (Willeminck et al., 2012; Lederman, 2010; Steiger et al., 2012; Mannion et al., 2012). Indeed, Mannion et al. (2012) recently reported that changes in abdominal muscle function such as voluntary and anticipatory activation after a motor control based exercise intervention were not significantly correlated with changes in disability ( $r = 0.08$  and  $r = 0.16$  respectively). Further, a systematic review examining the relationships between changes in trunk mobility, strength, and endurance provided little support to the idea that improvements in these aspects of functional performance were related to improvements in pain and/or disability (Steiger et al., 2012). As such, many now favour the neural/cognitive theories of mechanism of action for exercise.

These findings might be expected as many prior reviews have found that the evidence for consistent associations between general decrease in functional performance (i.e. deconditioning) with respect to the development of presence of CLBP is lacking (Verbunt et al., 2003; Verbunt et al., 2010; Wittink et al., 2000; Smeets et al., 2006). However, these reviews lacked consideration of the specific component that was deconditioned (Steele et al., 2014). In fact, a more recent review has re-appraised the evidence regarding the specific role of deconditioning of the extensor muscles of the lumbar spine (lumbar extensor musculature i.e. thoracic and lumbar erector spinae, including the iliocostalis lumborum and longissimus thoracis, the multifidus, and also the quadratus lumborum when contracted bilaterally; Steele et al., 2014). This review concluded that there was consistent evidence that persons with CLBP generally present deconditioning of these muscles (reduced lumbar extension strength/endurance, atrophy, and excessive fatigability) and that this deconditioning may actually be involved in a range of multifactorial symptoms and dysfunctions present in CLBP

(Steele et al., 2014). Further, this relationship may find its origins in our evolutionary past (Steele, 2017).

Because of the presence of this specific deconditioning, the conclusion that changes in function are not related to changes in pain and/or disability has been contested as being potentially premature (Steele & Bruce-Low, 2012). In their review, Steiger et al. (2012) focused primarily upon measures of trunk function, which incorporates both hip and lumbar extension/flexion. In light of the relationship between specific lumbar extensor deconditioning and CLBP, it should be noted that measures of trunk extension are not good indicators of the specific isolated function of the lumbar extensors (Conway et al., 2016). As such, a lack of relationship between change in measures of 'trunk' function and pain/disability in the lower back are not indicative of a similar relationship in measures of 'lumbar' function.

A further issue is that many studies have utilised exercise interventions that, while likely improving elements of trunk function, are ineffective in improving lumbar extensor function. Numerous exercises are purported to specifically condition this musculature (i.e. develop strength, endurance and hypertrophy) including: bench and roman chair trunk extensions (TEX), use of free weights (e.g. deadlifts, squats, good mornings), floor and stability ball exercise (e.g. TEX, bridging, 4-point kneeling), and resistance machines including those with and without restraints for isolated lumbar extension (ILEX) exercise (Mayer et al., 2008). Many of these approaches lack evidence for efficacy in conditioning the lumbar extensors. However, resistance machines providing ILEX appear to be the exception (Steele et al., 2015). Further, a review of studies that have utilised ILEX resistance training in patients with CLBP suggests that it is also effective in improving ILEX strength and reducing pain and disability, the reductions of which are also consistently meaningful (Steele et al., 2015).

However, irrespective of the outcome measure for function, most studies have not reported correlations between functional and clinical changes (Steiger et al., 2012). In addition, there

are even fewer studies that have examined their association whilst utilising ILEX resistance training as an intervention, and also examined changes in specific lumbar extensor function (e.g. ILEX strength). Of the studies that have reported this, some suggest that there may in fact be a relationship between improvements in this specific outcome and clinical changes. Nelson et al. (1995) reported that change in ILEX strength and change in pain were significantly and moderately correlated ( $r = -0.318$ ) in 677 participants with CLBP who underwent a 9 week ILEX resistance training intervention. Steele et al. (2013) reported significant moderate correlations for change in pain ( $r = -0.464$  to  $-0.651$ ) and change in disability ( $r = -0.453$  to  $-0.522$ ) in 24 participants with CLBP undergoing 12 weeks of ILEX resistance training. In contrast, however, Rittweger et al. (2002) reported no significant correlations for change in ILEX strength and change in pain in 50 participants with CLBP after a 12 week ILEX resistance training intervention.

Despite the present lack of research reporting relationships between changes in ILEX strength and changes in pain and/or disability, there have been numerous studies utilising this outcome in addition to ILEX resistance training in participants with CLBP (Steele et al., 2015). As such, there is a rich body of data that exists that could be examined retrospectively for the presence of correlations between these variables. Therefore, the purpose of the present study was to conduct a secondary analysis of data from studies utilising ILEX resistance training interventions for correlations between changes in ILEX strength, pain, and disability. This will include pooling of data from the present group's prior studies in addition to the attempted acquisition of raw data for pooling from studies identified in a recent review of this literature (Steele et al., 2015).

## **Methods**

### **Study Selection**

Raw data from 4 prior studies conducted by the authors were included (Smith et al., 2011; Bruce-Low et al., 2012; Steele et al., 2013; Steele et al., 2017). In addition, 23 studies identified in a prior review (Steele et al., 2015) were examined for whether they met the following inclusion criteria: participants must suffer from CLBP (symptoms lasting >12 weeks), the intervention included ILEX resistance training and lasted for  $\geq 4$  weeks, and the outcomes reported included ILEX torque, pain using visual analogue scale (VAS), and/or disability using the Oswestry Disability Index (ODI). These inclusion criteria were chosen to facilitate pooling with the 4 studies examined from the authors. Of those examined, 10 studies met the inclusion criteria and the corresponding authors of these studies were contacted to request release of the raw data for synthesis and analysis. Of these 10 studies, raw data were available from only 2. Data not included were due to either lack of response from corresponding authors or due to the data no longer being available (study was conducted prior to keeping of electronic records and paper records were no longer available). As such, 6 studies including 272 participants in total were pooled for analysis (Rittweger et al., 2002; Helmhout et al., 2004; Smith et al., 2011; Bruce-Low et al., 2012; Steele et al., 2013; Steele et al., 2017).

### Data Synthesis and Analysis

Study characteristics including participant demographics (average reported age, sex, duration of CLBP, pain, and disability) in addition to the ILEX intervention used (duration, repetition number, load, set volume, and frequency) were extracted. Dependent upon how raw data were reported, ILEX torque was considered as the peak from testing of multiple angles throughout the range of motion, average of all angles tested throughout the range of motion, and as a 'strength index' which was calculated as the area under the curve for all angles tested throughout the range of motion. Where necessary, raw torque data was converted from ft·lbs to N·m for synthesis. Pain measured using VAS was converted to a value from 0 to 100 mm if applicable (i.e. if authors had used a 0 to 10 scale) for synthesis. Both data for individual studies, in addition to the pooled data, were examined for assumptions of normality of distribution using a Shapiro-Wilk test. Those data sets meeting assumptions of normality of

distribution were examined for correlations among change in ILEX peak/average/strength index torque, VAS and ODI using a Pearson's correlation. Those data sets violating assumptions of normality of distribution were examined for correlations among change in ILEX peak/average/strength index torque, VAS and ODI using a Spearman's correlation. Correlations were examined individually within included studies in addition to being included in pooled analysis. Correlation coefficients were interpreted as weak ( $r = 0.30$  to  $0.50$ ), moderate ( $r = 0.50$  to  $0.70$ ) or strong ( $r > 0.70$ ). Statistical analyses were performed using SPSS (version 22; IBM, Portsmouth, Hampshire, UK) and  $p \leq 0.05$  accepted as the limit for statistical significance.

## **Results**

### **Study Characteristics**

Data from a total of 272 participants were available from the 6 studies included. The average age of participants was reported as ~40 to 46 years. Sex ratio of participants was ~2:1 (male:female). The average reported duration of CLBP symptoms was ~11 to 15 years. Baseline average pain was ~41 to 46 mm, and average disability ~14 to 39 pts. The ILEX interventions reported in the studies included were all of 12 weeks in duration. They used repetition numbers ranging from 8 to 20, using loads ranging 20% to 80% of maximal voluntary contraction, all used a single set of repetitions, and were performed from 1x to 2x/week.

### **Correlations between ILEX torque, VAS, and ODI**

For ILEX average torque (see table 1) and VAS significant moderate correlations were found for 3 of 3 studies ( $r = -0.526$  to  $-0.560$ ;  $p = 0.016$  to  $<0.001$ ) with pooled data showing a significant moderate correlation ( $r = -0.539$ ;  $p < 0.001$ ) and for ODI significant moderate correlations were found for 2 of 4 studies ( $r = -0.503$  to  $-0.510$ ;  $p = 0.033$  to  $<0.001$ ) with pooled data showing a significant moderate correlation ( $r = -0.386$ ;  $p < 0.001$ ). For ILEX peak torque (see table 2) and VAS significant weak to moderate correlations were found for 4 of 4 studies ( $r = -0.298$  to  $-0.483$ ;  $p = 0.050$  to  $0.011$ ) with pooled data showing a significant moderate



correlation ( $r = -0.391$ ;  $p < 0.001$ ) and for ODI significant weak to moderate correlations were found for 3 of 4 studies ( $r = -0.235$  to  $-0.522$ ;  $p = 0.047$  to  $<0.001$ ) with pooled data showing a significant moderate correlation ( $r = -0.349$ ;  $p < 0.001$ ). For ILEX strength index torque (see table 3) and VAS significant weak to moderate correlations were found for 4 of 4 studies ( $r = -0.285$  to  $-0.624$ ;  $p = 0.045$  to  $<0.001$ ) with pooled data showing a significant moderate correlation ( $r = -0.415$ ;  $p < 0.001$ ) and for ODI significant moderate correlations were found for 2 of 3 studies ( $r = -0.405$  to  $-0.564$ ;  $p = 0.015$  to  $<0.001$ ) with pooled data showing a significant moderate correlation ( $r = -0.470$ ;  $p < 0.001$ ). Figures 1 and 2 show scatter graphs for all pooled correlations examined.

## **Discussion**

It has recently been reported that there is an absence of relationship between change in most function or performance outcomes and improvements in clinical outcomes for lower back exercise (Steiger et al., 2012). However, the data reported herein suggests that change in ILEX strength may be associated with a positive clinical outcome. Indeed, the results of this secondary analysis show relatively consistent significant relationships between increases in ILEX strength and reductions in both VAS and ODI. It is interesting then to consider why change in ILEX strength might be uniquely related to clinical outcomes whereas other function and performance measures are not.

As noted, the theories regarding the mechanisms of action for exercise in CLBP can be broadly grouped as mechanical, neural, and cognitive (Helmhout et al., 2008). The lack of relationship between changes in many function- and performance- outcomes with changes in clinical outcomes has been suggested as arguing against the mechanical theory of exercise and being more supportive of the neural/cognitive theories (Steiger et al., 2012; Lederman, 2010). Indeed, the biopsychosocial model incorporates these components and has been adopted to better explain the complex relationships between nociception, pain and suffering (Engel, 1980, Turk & Okifuji, 2002; Gatchel et al., 2007). However, it is peculiar that despite

the lack of relationships between most function and performance outcomes and clinical outcomes, ILEX function stands out. This may be related to the specific role that deconditioning of the lumbar extensor musculature might play in the initiation and development of CLBP (Steele et al., 2014; Steele, 2017). As such, where interventions effective in conditioning this musculature are used, such as ILEX resistance training, they may be addressing a key causative mechanism. However, the evidence suggests that the deconditioning of the lumbar extensors in and of itself is not responsible for the initiation of pain causing mechanisms but instead likely leads to mechanisms responsible for injury, such as poor motor control and movement performance (Steele et al., 2014). Indeed, the improvements seen with ILEX resistance training, and the relationships between change in ILEX strength and pain and disability, may be due to the positive impact it has upon these mechanisms.

ILEX strength has been shown to be associated with lifting capacity (Reyna et al., 1995; Matheson et al., 2002) which improves as a result of an ILEX resistance training intervention in persons with CLBP (Mooney et al., 1993). Fisher et al. (2013) even found in recreationally trained males an ILEX intervention increased deadlift one repetition maximum. ILEX strength is also associated with lumbar kinematic pattern variability during gait in CLBP participants (Steele et al., 2014). This also improves as a result of an ILEX resistance training intervention (Steele et al., 2016) and other work has shown that change in ILEX strength is predictive of improvements in walking endurance in obese older persons with CLBP (Vincent et al., 2014). Thus, the relationship between improved ILEX strength and clinical outcomes as a result of ILEX resistance training may be due to the mechanism of action of improved motor control and movement performance.

However, the role of changes falling under the neural/cognitive theories of mechanisms of action may also be involved in the relationships presented here between changes in ILEX strength and clinical outcomes. High baseline fear-avoidance beliefs and disability have been

found to be predictive of poor outcomes resulting from ILEX resistance training interventions in persons with CLBP (Al-Obaidi et al. 2005; Helmhout et al., 2010), although ILEX resistance training interventions have been shown to improve such psychosocial outcomes (Risch et al., 1993). It is therefore possible that changes in ILEX function are acting as a 'surrogate' indicator of improvements in other elements of psychosocial function and that these are responsible for the clinical improvements. Indeed, measures of physical performance may in some cases be measures of pain-related behaviour (Huijen et al., 2013). However, the initial deconditioning of the lumbar extensors identified through ILEX strength tests in CLBP participants is also corroborated with other physiological measures such as atrophy and fatigability identified through electromyography analysis (Steele et al., 2014). Further, if it were indeed the case that ILEX strength changes were acting as a surrogate for improvements in other psychosocial factors affecting clinical outcomes, then we would expect to see similar relationships between other function and performance measures and clinical outcomes. Though this does not rule out that part of the mechanism of action for ILEX may be neural/cognitive, it does suggest that there may also be some influence of mechanical mechanisms of action through improved ILEX strength.

It is worth noting that there was some heterogeneity in the ILEX resistance training interventions employed in the studies included. Studies from the authors' group (Smith et al., 2011; Bruce-Low et al., 2012; Steele et al., 2013; Steele et al., 2017) in addition to Rittweger et al. (2002) all used a similar manipulation of resistance training variables, most notably characterised by having participants train to momentary failure and thus producing maximal effort (Steele, 2014; Steele et al., 2017). The study of Helmhout et al. (2004) contrastingly had participants train with low loads not to failure, and thus producing a relatively lower effort. This was the only study included in the current analysis that did not support any significant relationship between changes in ILEX strength and clinical outcome ( $r = 0.031$ ,  $p = 0.793$  for change in ILEX and change in ODI). A further study from this group using a similarly low load and low effort ILEX resistance training intervention reported improvements in disability, but no

change in multifidus cross sectional area (Willemink et al., 2012). Evidently, clinical improvement can occur as a result of any exercise as noted and this might be a result of neural/cognitive mechanisms (Steiger et al., 2012; Lederman, 2010). However, it has been argued that these studies employed ILEX resistance training interventions that were in fact lacking in efficacy with respect to addressing the condition of the lumbar extensors (Steele, 2014; Steele et al., 2013). Contrastingly, studies from the present authors group (Smith et al., 2011; Bruce-Low et al., 2012; Steele et al., 2013; Steele et al., 2017) and Rittweger et al. (2002) employed higher effort ILEX resistance training interventions that are better evidenced to produce improvements in muscular condition, including strength (Fisher et al., 2011), hypertrophy (Fisher et al., 2013), and aerobic capacity (Steele et al., 2012). As such, the presence of significant relationships between improvements in ILEX strength and clinical outcomes in these studies might suggest that although any exercise can produce improvement, exercise addressing the mechanical mechanism of improved muscular condition may optimise outcomes. Indeed, low volume, low frequency, yet high effort ILEX resistance training is shown to consistently produce significant improvements in pain and disability that meet minimal clinical important change criteria (Steele et al., 2015).

It is worth noting that there was evidently some variability in the individual relationships between change in ILEX strength and change in pain and disability in response to ILEX resistance training, as can be seen in Figures 1 and 2. Nelson et al. (1995) found that specific sub-grouping did not appear to affect group outcomes despite all participants receiving the same ILEX resistance training intervention. Although, considering the heterogeneity of CLBP, it might be expected that there would be at least some degree of variability in the responsiveness of individuals to different treatments. Nelson et al. (1995) also asked their participants to rate pain changes after an ILEX intervention on a 5-item scale ('worse', 'no change', 'slight decrease', 'decreased', 'substantially decreased'), reporting 64% rated a substantial decrease, 14% rated a decrease, 6% rated a slight decrease, 12% rated no change, and 3% rated a worsening of symptoms. It seems likely that considering the

multifactorial nature of CLBP, though in general improvements in ILEX strength may be related to clinical improvements, there may be instances whereby certain symptoms and dysfunctions present might impact that relationship. Numerous models attempting to explain, predict and integrate the multifactorial nature of CLBP have emerged within the literature (Hodges et al., 2015; Langevin & Sherman, 2007; Richmond, 2012). Indeed, due to the multifactorial nature of CLBP, sub-grouping (i.e. splitting of the larger heterogeneous population of LBP into smaller more homogenous groups) has been argued to be valuable in directing treatment to be more effective (Ford et al., 2007; Hepple & Robertson, 2006; Lebouef-Yde et al., 1997). Further studies should consider the prognostic factors that might help practitioners discern *a priori* whether a person is likely to be either a good or bad responder to ILEX resistance training in terms of clinical outcome (Helmhout et al., 2004).

### Conclusion

The results presented here suggest that improvements in ILEX strength may be related to positive clinical outcomes. Considering the absence of relationships between many other function or performance changes and clinical outcomes, conditioning of the lumbar extensor musculature may be a mechanism of action affecting symptom improvement. The precise nature of this relationship and how this mechanism of action specifically works is still unclear. However, these results suggest that specific conditioning of the lumbar extensor musculature could be considered an important outcome to focus upon in clinical practice in persons suffering from CLBP.

## **References**

1. National Institute for Health and Clinical Excellence. Low back pain: early management of persistent non-specific low back pain. London: Royal College of General Practitioners; 2009
2. Van Tulder MW, Malmivaara A, Esmail R, et al. Exercise therapy for low back pain. Cochrane Database Syst Rev. 2000;2:CD00335
3. Hayden J, Van Tulder MW, Malmivaara A, et al. Exercise therapy for no-specific low back pain. Cochrane Database Syst Rev. 2005;3:CD000335
4. Searle A, Spink M, Ho A, et al. Exercise interventions for the treatment of chronic low back pain: A systematic review and meta-analysis of randomised controlled trials. Clin Rehabil. 2015;29(12):1155-1167

5. 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
6. Willemink MJ, van Es HW, Helmhout PH, et al. 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*. 2012;37(26):E1651-1658
7. 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 the targeted aspect(s) of performance? A systematic review. *Eur Spine J*. 2012;21:575-598
8. Mannion AF, Caporaso F, Pulkovski N, et al. Spine stabilisation exercise in the treatment of chronic low back pain: a good clinical outcome is not associated with improved abdominal muscle function. *Eur Spine J*. 2012;21:1301-1310
9. Verbunt JA, Smeets RJ, Wittink HM. Cause or effect? Deconditioning and chronic low back pain. *Pain*. 2010;149:428-430
10. Smeets RJEM, 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
11. Steele J, Bruce-Low S, Smith D. A reappraisal of the deconditioning hypothesis in low back pain: review of evidence from a triumvirate of research methods on specific lumbar extensor deconditioning. *Curr Med Res Opin* 2014;30(5):865-911
12. Steele J. An evolutionary hypothesis to explain the role of deconditioning in low back pain prevalence in humans. *J Evo Health* 2014;2(2):DOI: 10.15310/2334-3591.1007  
Changes in Strength, Pain, and Disability

13. Steele J, Bruce-Low S, Steiger et al. 2011: relationships and specificity in CLBP rehabilitation through exercise. *Eur Spine J.* 2012;21(9):1887
14. Conway R, Behennah J, Fisher J, et al. Associations between trunk extension endurance and isolated lumbar extension strength in both asymptomatic participants and those with chronic low back pain. *Healthcare.* 2016;4(3):e70
15. Mayer J, Mooney V, Dagenais S. Evidence-informed management of chronic low back pain with lumbar extensor strengthening exercises. *Spine J.* 2008;8(1):96-113
16. Steele J, Bruce-Low S, Smith D. A review of the specificity of exercise designed for conditioning the lumbar extensors. *Br J Sports Med.* 2015;49(5):291-297
17. Steele J, Bruce-Low S, Smith D. A review of the clinical value of isolated lumbar extension resistance training for chronic low back pain. *PM R.* 2015;7(2):169-187
18. Nelson BW, O'Reilly E, Miller M, et al. The clinical effects of intensive, specific exercise on chronic low back pain: a controlled study of 895 consecutive patients with 1-year follow up. *Orthopedics.* 1995;18(10):971-981
19. Steele J, Bruce-Low S, Smith D, et al. A randomized controlled trial of limited range of motion lumbar extension exercise in chronic low back pain. *Spine* 2013;38(15):1245-1252
20. Rittweger J, Just K, Katzsch K, et al. Treatment of chronic lower back pain with lumbar extension and whole-body vibration exercise: a randomized control trial. *Spine.* 2002;27(17):1829-1834
21. 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 Musculoskeletal Rehabil.* 2011;24(4):241-249



22. Bruce-Low S, Smith D, Burnet S, et al. One lumbar extension training session per is sufficient for strength gains and reductions in pain in patients with chronic low back pain. *Ergonomics*. 2012;55(4):500-507
23. Steele J, Bruce-Low S, Smith D, et al. Isolated lumbar extension resistance training improves strength, pain, and disability, but not spinal height or shrinkage ("creep") in participants with chronic low back pain. *Cartilage*. 2017;Epub ahead of print  
[doi.org/10.1177/1947603517695614](https://doi.org/10.1177/1947603517695614)
24. Helmhout PH, Harts CC, Staaal JB, et al. Comparison of a high-intensity and a low-intensity lumbar extensor training program as minimal intervention treatment in low back pain: a randomized trial. *Eur Spine J*. 2004;13(6):537-547
25. Ostelo RW, Deyo RA, Stratford P, et al. Interpreting change scores for pain and functional status in low back pain: towards international consensus regarding minimal important change. *Spine*. 2008;33(1):90-94
26. Gatchel RJ, Peng YB, Peters ML, et al. The biopsychosocial approach to chronic pain: scientific advances and future directions. *Psychol Bull*. 2007;133(4):581-624
27. Reyna JR jr, Leggett SH, Kenney K, et al. The effect of lumbar belts on isolated lumbar muscle. Strength and dynamic capacity. *Spine*. 1995;20(1):68-73
28. Matheson LN, Leggett S, Mooney V, et al. The contribution of aerobic fitness and back strength to lift capacity.. *Spine*. 2002;27(11):1208-1212
29. Mooney V, Matheson L, Holmes D, et al. Effect of focused strength training after low back injury. Paper presented at: North American Spine Society Annual Meeting; 1993; San Diego, California
30. 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*. 2013;14(3):139-145

31. Steele J, Bruce-Low S, Smith D, et al. Lumbar kinematic variability during gait in chronic low back pain and associations with pain, disability and isolated lumbar extension strength. *Clin Biomech.* 2014;29(10):1131-1138
32. Steele J, Bruce-Low S, Smith D, et al. A randomized controlled trial of the effects of isolated lumbar extension exercise on lumbar kinematic pattern variability during gait in chronic low back pain. *PM R.* 2016;8(2):105-114
33. Vincent HK, Vincent KR, Seay AN, et al. Back strength predicts walking improvement in obese, older adults with chronic low back pain. *PM R.* 2014;6(5):418-426
34. Helmhout PH, Staal JB, Heymans MW, et al., Prognostic factors for perceived recovery or functional improvement in non-specific low back pain: secondary analyses of three randomized clinical trials. *Eur Spine J.* 2010;19(4):650-659
35. Risch SV, Norvell NK, Pollock ML, et al. Lumbar strengthening in chronic low back pain patients. Physiologic and psychological benefits. *Spine.* 1993;18(2):232-238
36. Huijen IPJ, Verbunt JA, Wittink HM, et al. Physical performance measurement in chronic low back pain: measuring physical capacity or pain-related behaviour? *Eur J Physiother.* 2013;15:103-110
37. Fisher J, Steele J, Bruce-Low S, et al. Evidence based resistance training recommendations. *Medicina Sportiva.* 2011;15(3):147-162
38. Fisher J, Steele J, Smith D. Evidence based resistance training recommendations for muscular hypertrophy. *Medicina Sportiva.* 2013;17(4):217-235
39. 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