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1 **TITLE:** A Randomized Controlled Trial of the Effects of Isolated Lumbar Extension Exercise on Lumbar
2 Kinematic Pattern Variability during Gait in Chronic Low Back Pain

3

4 **CONTRIBUTORSHIP STATEMENT**

5 All listed authors contributions include the conception and design, acquisition of data or analysis and interpretation
6 of data, drafting the article or revising it critically for important intellectual content, and final approval of the
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11 The authors have no conflicts of interest to declare.

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28 **ABSTRACT**

29 *Background:* Chronic low back pain (CLBP) is a multifactorial condition with a variety of symptoms; one being
30 abnormal gait. The lumbar spine and its musculature are important in controlling gait and in CLBP the lumbar
31 extensors are often deconditioned. Because of this specific isolated lumbar extension exercise is often
32 recommended. It was therefore of interest to examine its effects of upon gait variability.

33 *Objective:* To examine the effects of isolated lumbar extension resistance training upon lumbar kinematic
34 variability during gait in participants with CLBP.

35 *Design:* Randomized controlled trial.

36 *Setting:* University Health, Exercise and Sport Science Laboratory

37 *Participants:* Twenty four participants with non-specific CLBP

38 *Interventions:* Participants were randomly allocated to a 12 week isolated lumbar extension exercise intervention
39 (1x/week performing a single set to momentary muscular failure using a load equal to 80% max tested torque) or
40 non-training control period.

41 *Main Outcome Measurements:* Lumbar kinematics during gait including angular displacement, kinematic
42 waveform pattern (CV_p) and offset (CV_o) variability were examined using three dimensional analyses.

43 *Results:* No significant changes in displacement or CV_o were found as a result of the intervention. However, a
44 small but significant reduction in sagittal plane CV_p ($-20.90 \pm 43.53\%$, Effect Size = 0.48, $p = .044$) occurred
45 indicating improved motor pattern replication through this movement plane.

46 *Conclusions:* Considering the role of the lumbar extensors in gait, and their common deconditioning in CLBP, an
47 isolated lumbar extension resistance exercise intervention may reduce gait variability. These results suggest
48 isolated lumbar extension exercise may specifically reduce sagittal plane variability, indicating improved motor
49 pattern replication through this movement plane, perhaps due to the plane of movement utilized during the
50 exercise.

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58 **KEY WORDS:** Three Dimensional Analyses; Resistance Training; Strength; Disability; Motor Control

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88 **INTRODUCTION**

89 Chronic low back pain (CLBP) is highly prevalent [1-4] with considerable costs worldwide [5-13]. However, in
90 as much as 85% of LBP cases no specific patho-anatomical diagnosis is found [14]. More recently CLBP has been
91 noted a multifactorial condition with a variety of associated symptoms [15,16]. One of these symptoms being
92 abnormal gait [17-19]. Average movement amplitudes of the trunk and pelvis in CLBP participants do not usually
93 differ from asymptomatic participants [18,20,21]. However despite this, CLBP participants do present differently
94 in other aspects of lumbar spine movement; inability to adapt pelvis/trunk coordination phase differences during
95 increased walking velocity [20-26], and greater stride-to-stride variability lumbar spine movement relative to the
96 pelvis [18]. Lamothe and colleagues [24] suggest ability to deal with unexpected perturbations in movement is
97 likely reduced. It is also suggested that deficiencies in gait control produce excessive stresses to the lumbar spine,
98 perhaps contributing to CLBP [18]. However, recent review reports little evidence for walking itself being
99 causally associated with CLBP [27]; thus the gait observed in CLBP might be justifiably considered a symptom
100 instead.

101

102 Another common factor associated with CLBP is specific deconditioning (i.e. reduced strength/endurance,
103 atrophy, and excessive fatigability) of the lumbar extensor musculature [28] with evidence suggesting it may be
104 involved in abnormal gait in CLBP [20,23,29-35]. Healthy participants demonstrate relatively low stride-to-stride
105 variability in lumbar kinematic patterns during level and incline gait [36]. However, greater stride-to-stride
106 variability at the lumbar spine in all planes [18], greater frontal plane coordination variability of the pelvis/trunk
107 [20,21], and more rigid transverse plane coordination variability of the pelvis/trunk [20,25,37] is reported in CLBP
108 participants. This abnormal variability combines with poorer erector spinae activity adaptability to unexpected
109 perturbations [29], or velocity changes [23]. In fact, findings from numerous studies suggest lumbar extensor
110 dysfunction during gait in CLBP [20,23,29-31]. Hanada et al. [35] also report, though asymptomatic controls
111 activated their rectus abdominus and internal oblique's greater compared with their lumbar extensors, the opposite
112 was seen in symptomatic participants i.e. greater lumbar extensor activation compared to rectus abdominus and
113 internal oblique's. More recent work suggests greater lumbar extensor activity in CLBP participants compared
114 with controls [32], at a range walking velocities [33], and neither disability nor fear of movement is associated
115 with this activity [32]. However, different coping strategies may be associated with greater activity
116 (catastrophizing) or greater relaxation during double support (distraction) suggesting some cognitive influence
117 over control of motor patterns [34].

118

119 Gait is normally quite robust in the face of lower limb muscular weakness [38]. The lumbar spine, however, helps
120 drive human bipedal gait [39]. It is possible greater lumbar extensor activation, and altered lumbar spine
121 kinematics in CLBP, is a manifestation of the commonly associated lumbar extensor deconditioning [28]. Greater
122 activation in the face of fatigue due to deconditioning might be compensatory to maintain lumbar spine control
123 during gait. Hart et al. [40] demonstrate inducing lumbar extensor fatigue impacts lumbar kinematics during
124 running gait of healthy and CLBP participants. Arjunan et al. [41] also show greater lumbar extensor activity
125 during running gait in CLBP. Indeed, prospective evidence suggests lumbar extensor deconditioning as a risk
126 factor for low back injury and pain [28]. Thus, it may be responsible for development of abnormal gait variability
127 in CLBP.

128

129 Exercise programs have shown success in improving aspects of gait variability in older individuals with
130 improvement in part determined by strength gains [42]. Specific lumbar extensor exercise, however, is often used
131 in CLBP [43] and thus may affect the associated lumbar spine kinematic gait variability. Varied exercise based
132 interventions (Pilates, trunk extensions, stability exercise, transverse abdominus exercise) improve gait control
133 in CLBP participants [44-46]. However, more specific exercise for the lumbar extensors is isolated lumbar
134 extension (ILEX) [47]. ILEX significantly improves lumbar extensor strength, pain and disability in CLBP
135 participants [48-50]. Further, recent work reports improvement in ILEX strength from a strengthening program
136 predicts improved gait endurance in CLBP participants [51]. ILEX however has yet to be examined for effects
137 upon lumbar kinematics during gait. Taking this into consideration, the purpose of this study was to examine the
138 effects of an ILEX exercise intervention upon lumbar kinematic variability during gait in participants with CLBP.

139

140 **METHODS**

141 *Study Design*

142 A randomized controlled trial design was adopted with one experimental group and a control group. The study
143 was part of a wider investigation examining ILEX in CLBP participants published in part elsewhere [50]. Gait
144 data were also collected as part of this study, though it was not hypothesized the different training groups
145 (FULLROM & LimROM) would differ in this outcome. Data analysis revealed no differences between the two
146 intervention groups for these outcomes and variables found to significantly improve here were similar between
147 the two groups (see below). Thus here the two groups were combined to form a single training group. Strength,

148 pain and disability outcomes are reported elsewhere [50]. Here the gait data are described only. The study was
149 approved by the NHS National Research Ethics Service, Southampton & South West Hampshire Research Ethics
150 Committee B (REC Reference: 11/H0504/9).

151

152 ***Participants***

153 Thirty eight participants (males n = 21, females n = 17) were initially identified and recruited by posters, group
154 email and word of mouth from a University and the surrounding locality. Direct referral was also provided from
155 a local private chiropractor in addition to posters in their practice. A power analysis described previously [50]
156 showed that each group required 7 participants to meet the required power of 0.8 at an alpha value of $p \leq .05$. No
157 previous work has examined effect sizes of the kinematic variables considered here and so, though considered
158 adequately powered with respect to ILEX strength outcomes, there was possibility a type II error may result with
159 respect to kinematic data. To reduce this risk, 5 kinematic trials were performed per participant, considered
160 sufficient for adequate statistical power for kinematic data utilizing single subject statistical methods [52].

161

162 Inclusion criteria were as follows; participants suffered from current non-specific low back pain having lasted
163 longer than 12 weeks [53] and had no medical condition for which resistance training would be contraindicated.
164 Exclusion criteria were as follows; participants must have no medical condition for which movement therapy
165 would be contraindicated. These included: acute (not re-occurring) low back injury occurring within the last 12
166 weeks, pregnancy, evidence of sciatic nerve root compression (sciatica), leg pain radiating to below the knee,
167 paresthesia (tingling or numbness), current tension sign, lower limb motor deficit, current disc herniation, previous
168 vertebral fractures or other major structural abnormalities. Participants were cleared as meeting the inclusion
169 criteria and not exhibiting any of the exclusion criteria prior to involvement in the study by either their General
170 Practitioner or the Chiropractor in the research group and provided written informed consent.

171

172 Figure 1 shows a CONSORT diagram highlighting participant numbers for enrolment, allocation, follow-up and
173 analysis. After initial drop outs thirty one participants were randomized using an randomization program
174 (Research Randomizer vs. 3.0) to one of three participant groups; FullROM training group who trained using a
175 full range of motion (n = 12), LimROM training group who trained using a limited range of motion (n = 10), and
176 a control group (n = 9). As noted, the two training groups were combined for analysis.

177

178 ***Equipment***

179 Isometric ILEX strength testing and training were performed using the MedX Lumbar Extension Machine (MedX,
180 Ocala, Florida; figure 2). The lumbar extension machine is reliable in asymptomatic [54] and symptomatic
181 participants [55], and valid in removal of gravitational effects [56] and pelvic movement [57]. Pain was measured
182 using a 100mm point visual analogue scale (VAS) [58], and disability measured using the revised Oswestry
183 disability index (ODI) [59]. Gait kinematic variables were captured at 500hz using a 10 MX T20 camera three
184 dimensional motion capture system (Vicon, Oxford) and analyzed using both Vicon Nexus software version
185 1.4.116 (Vicon, Oxford), MATLAB version R2012a (MathWorks, Cambridge) and Microsoft Excel version 2010
186 (Microsoft, Reading).

187

188 ***Participant Testing***

189 Isometric ILEX strength was tested twice, on separate days (at least 72 hours apart in order to avoid the effects of
190 residual fatigue or soreness) both before and after the intervention. The first test acted as familiarization and data
191 from the second test was used for analysis. Each test involved maximal voluntary isometric contractions. Details
192 of the full test protocol using the lumbar extension machine are documented elsewhere [54]. During the first and
193 second to last visit to the laboratory, before and after the intervention, participants completed the VAS and the
194 ODI. Gait data was collected using the Vicon system during the third visit and final visit to the laboratory both
195 before and after the intervention period. Gait data was collected at least one week after isometric ILEX strength
196 testing.

197

198 ***Three dimensional motion analyses***

199 A three dimensional approach was used for data collection. Ten cameras were set up and angled in a manner to
200 reduce hidden spots that might obscure data collection. The cameras identified reflective markers attached to the
201 participant and output three dimensional coordinates for each marker. Data were recorded for 5 walking trials
202 both pre and post intervention. Participants walked barefoot along a marked runway 8 meters in length at their
203 free walking speed. At least one full gait cycle was captured per trial.

204

205 ***Biomechanical Model***

206 The lumbar spine was considered from S1 to T12 relative to the pelvis and modelled as a rigid segment due to the
207 segments ranging S2 to T10 always bending laterally toward the support leg with little variation between segments

208 [60]. Lumbar spine data were collected using the model previously described by Schache et al. [61] shown to have
209 high overall repeatability of angular parameters [62].

210

211 ***Marker Set Up***

212 Reflective markers were placed over anatomical landmarks on the pelvis at both anterior superior iliac spines
213 (ASIS) and at the midpoint of the posterior superior iliac spine (PSIS). Reflective markers were also used upon a
214 thoracolumbar marker cluster similar to that used by Schache et al., [61,62]. As with the biomechanical model,
215 this marker set up has been previously described elsewhere [61,62]. The only alteration in this present study was
216 the use of a flexible based wand marker for the thoraco-lumbar cluster. Two additional markers were secured
217 equidistant either side of the midpoint of the wand markers base. This was placed over T12 with the mid-point of
218 the base located over the spinous process. The ASIS and PSIS were identified by palpation after identifying the
219 iliac crest and palpating along its length. T12 was first located and marked using the technique suggested in *Gray's*
220 *Anatomy for Students* [63]. This location was confirmed, whilst the participant was in a flexed standing position
221 supporting themselves upon a stool, by palpation and counting of the spinous processes from this marked point
222 down to the sacrum, and then double checked by counting back up to the marked spinous process. All markers
223 and the base of the thoracolumbar marker cluster were secured using double sided adhesive tape. Markers were
224 placed by the same investigator for all gait trials. Figure 3 shows the marker set-up used.

225

226 ***Kinematic Data***

227 Variability of angular kinematics of the lumbar spine about the three described axes relative to the pelvic segment
228 was of primary interest (i.e. movement of the thoraco-lumbar marker cluster with respect to the pelvic markers).
229 The Vicon Nexus software was used to run a Bodybuilder (Vicon, Oxford) code pipeline to calculate joint angles
230 as outputs using Cardan (Euler) angles. The angles were calculated in the following order; 1) sagittal, 2) frontal,
231 and 3) transverse. As with the biomechanical model, the Bodybuilder code used was the same as used by Schache
232 et al. [61,62]. Data were filtered using a low pass Butterworth filter (fourth order, cutoff frequency determined
233 for each individual participant as sum of residuals closest to zero using 4Hz, 6Hz, 8Hz, 10Hz, and 12Hz) and
234 normalized to percentage gait cycle corresponding to initial right heel contact (0%) and subsequent right heel
235 contact (100%). Heel contacts were identified as the lowest vertical displacement of a right heel marker. Stride
236 duration and length was also calculated using the horizontal displacement of the right heel marker from initial
237 right heel contact and subsequent right heel contact. Intra-subject variability in mean ensemble average was

238 calculated using coefficient of variation with pattern (CV_p) and offset (CV_o) variability calculated separately to
239 account for the different information they provide; CV_o being the variability in the mean offset of the waveform
240 determined by the reference frame used, identification of anatomical landmarks, markers and their configuration,
241 whereas CV_p represents the variability in the waveform pattern and is more representative of repeatability of motor
242 performance [64].

243

244 ***Participant Training***

245 Training was conducted 1x/week for 12 weeks. This frequency of training significantly improves ILEX strength
246 whereas overtraining can occur at greater frequencies for ILEX [65], and that 2x/week training offers no greater
247 improvements [48]. Twelve weeks was chosen as strength improvement from ILEX training occurs largely within
248 the first 12 weeks [66]. Both groups performed one set of variable resistance ILEX exercise. FullROM group used
249 their full ROM while LimROM group only used the mid 50% of their individual ROM [50]. Load was 80% of
250 max recorded ILEX strength and repetitions performed until momentary muscular failure to control intensity of
251 effort [67] using a duration of at least 2 seconds concentric phase, 1 second hold in full extension and at least 4
252 seconds eccentric phase. Load was increased 5% next session once the participant could continue for over 105
253 seconds using their current load before failure.

254

255 ***Data Analysis***

256 Eligibility for analysis required completion of 75% of the intervention. Twenty four participants' data (Males, n
257 = 13; Females, n = 11) were available after attrition. This number combined with 5 trials per participant was
258 sufficient for statistical power. Mean values for angular displacements, stride-to-stride intra-subject variability
259 using CV_p and CV_o were calculated for lumbar spine kinematics relative to the pelvis across all three planes of
260 movement. Baseline demographic data and changes in VAS, ODI and ILEX strength met assumptions of
261 normality and homogeneity of variance and thus were compared between groups using an independent samples t-
262 test. Kinematic data did not meet assumptions of normality or homogeneity of variance as is typical [68]. Thus
263 non-parametric analysis was used and baseline data compared between groups using Mann Whitney-U exact test
264 to check randomization succeeded for these variables. Examining the effects of the intervention, the independent
265 variable was participant group (i.e. Combined training or Control) and dependent variables the absolute change
266 from pre to post for kinematic variables. Wilcoxon Signed Ranks Exact test compared across the independent
267 conditions. Perceived pain and disability were compared to consensus standards for minimal clinically important

268 change [69] (MCIC). Ostelo et al [69] proposed the MCIC for VAS as 15mm and for ODI 10 points. Further,
269 effects sizes were calculated using Cohens d [70]. Statistical analysis was performed using SPSS statistics
270 computer package (vs.20) and $p \leq .05$ set as the limit for statistical significance.

271

272 **RESULTS**

273 *Participant Demographics*

274 Participant demographics, pain, disability and ILEX strength data are shown in Table 2 for groups. Comparison
275 between groups revealed similar demographic variables at baseline and only showed a significant difference in
276 VAS score ($t_{(22)} = 2.420, p = .024$).

277

278 *Effects of Intervention upon VAS, ODI, and ILEX Strength*

279 Table 2 shows mean changes in VAS, ODI and ILEX strength in addition to effects sizes and 95% Confidence
280 Intervals. The training group showed significant changes in VAS ($t_{(22)} = -3.651, p = .001$), ODI ($t_{(22)} = -4.831, p <$
281 $.001$ and ILEX strength ($t_{(20)} = 3.641, p = .002$) compared with the control group. Effect sizes were also considered
282 larger for the training group and VAS and ODI both met MCICs.

283

284 *Effects of Intervention upon Kinematic Variables*

285 Table 3 shows pre and post group data for displacement, CV_p and CV_o . Wilcoxon Signed Ranks Exact test revealed
286 significant changes after the intervention only for sagittal plane CV_p ($W_{(16)}, Z = -1.728, p = .044$) in the training
287 group only (The FullROM and LimROM groups made similar average improvements individually of -20.32%
288 and -21.72% respectively) suggesting improvement in stride to stride waveform pattern replication. Figure 4
289 presents an example of the pre and post kinematic waveforms for one training group participant for both individual
290 gait trials and also the mean ensemble average showing a reduced stride to stride variability (evidenced by the
291 narrower standard deviations about the mean ensemble average).

292

293 **DISCUSSION**

294 A 12 week ILEX resistance training intervention produced significant reduction in sagittal plane variability during
295 gait in CLBP participants. These findings potentially offer further understanding regarding the relationships
296 between CLBP, gait variability and lumbar extensor deconditioning.

297

298 Lumbar kinematic variability during gait in CLBP participants may be a consequence of the lumbar extensor
299 deconditioning frequently associated with this population [28]. This potential link is emphasized by the fact that
300 lumbar extensor fatigue affects lumbar kinematics during gait [40]. It seems reasonable to conclude that
301 deconditioning of the musculature associated with controlling gait in patients with CLBP might be partially
302 responsible for altered motor control. [39,71-73] Our findings in this study tend to support this conclusion.
303 Previous studies offer support for exercise interventions improving aspects of gait variability including muscle
304 activation [46], ground reaction force parameters [45] and displacements [44]. However, none have examined
305 lumbar kinematic variability during gait, nor utilized specific exercise to isolate the lumbar extensors. Within the
306 present study an intervention employing a highly specific form of exercise (ILEX) evidenced as most effective
307 for conditioning the lumbar extensors was used [47]. The results indicate ILEX resistance training produced
308 significant reduction in sagittal plane CV_p suggesting improved ability to replicate motor patterns in this plane
309 during gait. Because ILEX may be an optimal approach for conditioning the lumbar extensors [47] it appears
310 reasonable the results produced may be the result of addressing the specific deconditioning seen in CLBP [28].

311

312 However, the improvement in sagittal CV_p may suggest a specific intervention effect due to the plane of motion
313 ILEX exercise is performed through. An exercise device similar to that used for ILEX also exists, which allows
314 pelvic restraint for torso rotation through the transverse plane to be performed in isolation (Torso Rotation
315 Machine, MedX, Ocala, Florida). Mooney et al. [74], demonstrated the latissimus dorsi and contralateral gluteus
316 maximus follow a reciprocal activity relationship during gait, presumably contributing to control about the
317 transverse plane. Mooney et al. [74] also examined activation during torso rotation exercise reporting abnormal
318 activation patterns in symptomatic participants compared with controls. After a training intervention of
319 progressive resistance training using the torso rotation device activation returned to normal levels of activity seen
320 in asymptomatic participants. However, despite reporting EMG results for the latissimus and gluteus to clarify
321 their role during gait, Mooney et al. [74] did perform pre and post intervention gait measurements to identify if
322 any change had occurred in muscular control during gait. In light of the results of the present study it is suggested
323 future research examine whether plane of movement specific training produces consequent plane of movement
324 specific changes in lumbar spine control during gait i.e whether torso rotation improves transverse CV_p .

325

326 Though it seems reasonable the lumbar extensor conditioning effect from ILEX [47] might be the responsible for
327 the sagittal CV_p changes reported, the effect of reduced pain or disability should also be considered. In a previous

328 report [50] and others [48,49] we show the ILEX intervention used produces significant and meaningful reductions
329 in pain and disability. Thus this may be a factor responsible for the gait improvements. However, other evidence
330 suggests pain presence may not be associated with gait variability [26,29,75]. Lumbar spine kinematics during
331 gait appear complex and developed over time, with patterns evident before pain is experienced [75] and both
332 induced pain and fear of pain produce little change in muscle activity in CLBP patients [29]. Recent studies have
333 shown that, even those with previous history of CLBP who are currently asymptomatic, demonstrate abnormal
334 gait patterns [21,76]. Considering the multifactorial nature of CLBP, this evidence suggests gait variability may
335 be a symptom associated with CLBP that results in consequence of lumbar extensor deconditioning. However, it
336 is possible that pain might not be primarily responsible for this findings, but it might be caused by the
337 consequences of pain. Though neither disability nor fear of movement is associated with greater lumbar extensor
338 activity during gait in CLBP [32], different cognitive strategies may be associated with greater activity
339 (catastrophizing), or greater relaxation during double support (distraction), suggesting influence of pain
340 consequences [34]. This consideration requires further investigation.

341

342 *Study Limitations*

343 The limitations of the present investigation should be noted. The clinical value of the significant change in sagittal
344 CV_p ($-20.90 \pm 43.53\%$) is not wholly clear due to the large variability. If the effect size is calculated (0.48), the
345 magnitude of change is considered small [70]. In addition, we are unaware of whether any data on asymptomatic
346 participants exists for these outcomes, thus making the determination of clinical significance difficult. Finally,
347 though Schache et al. [62] have shown high reliability for angular data for the model adopted in this study, we did
348 not conduct our own reliability analysis.

349

350 **CONCLUSIONS**

351 The results of this study provide novel information on lumbar spine kinematic variability during gait in CLBP. A
352 12 week ILEX resistance exercise intervention significantly reduced sagittal plane CV_p suggesting improved
353 motor pattern replication. These findings are important as they demonstrate that improvements may be possible
354 in various factors typically associated with CLBP through use of ILEX exercise.

355

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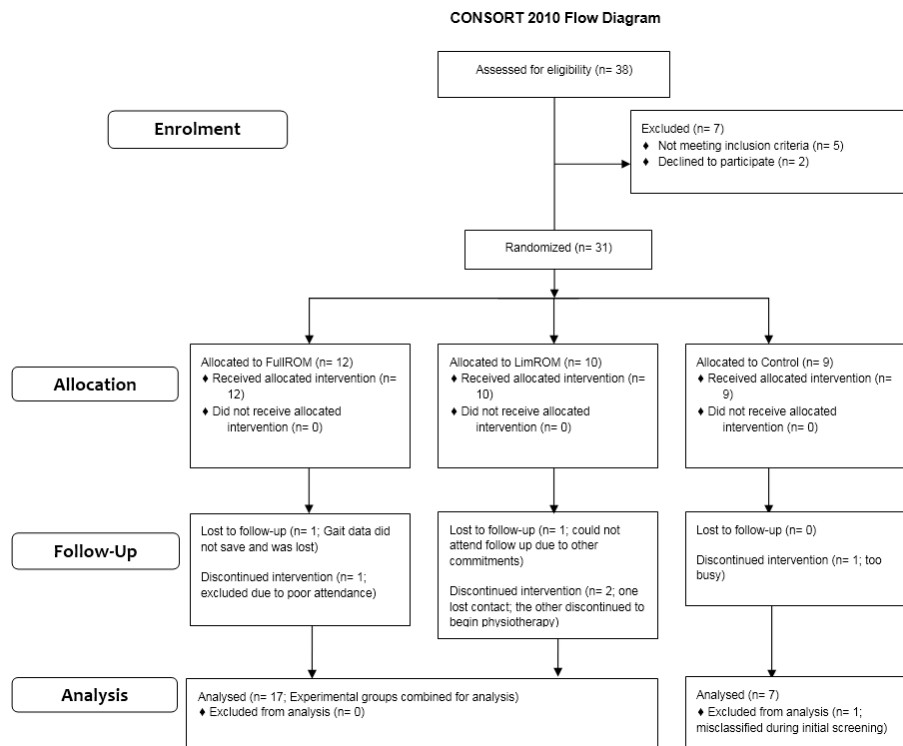
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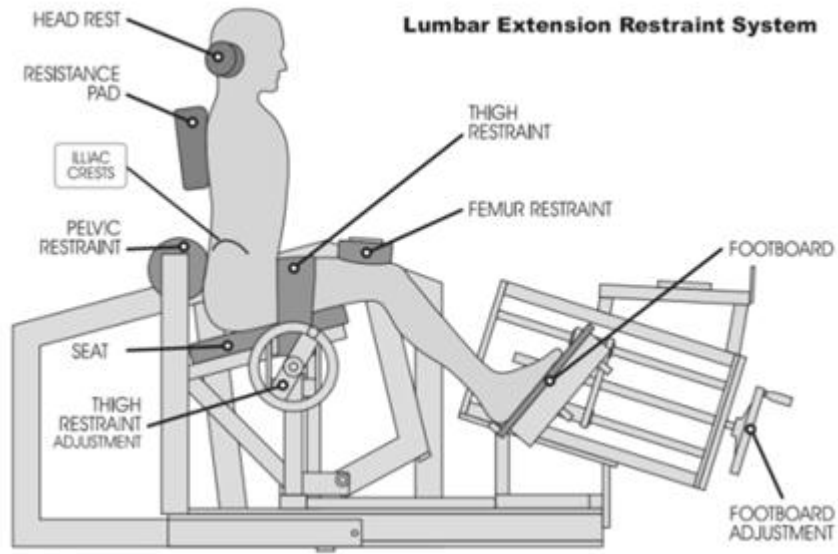
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FIGURES



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518 Figure 1. CONSORT diagram showing flow of participants through the study



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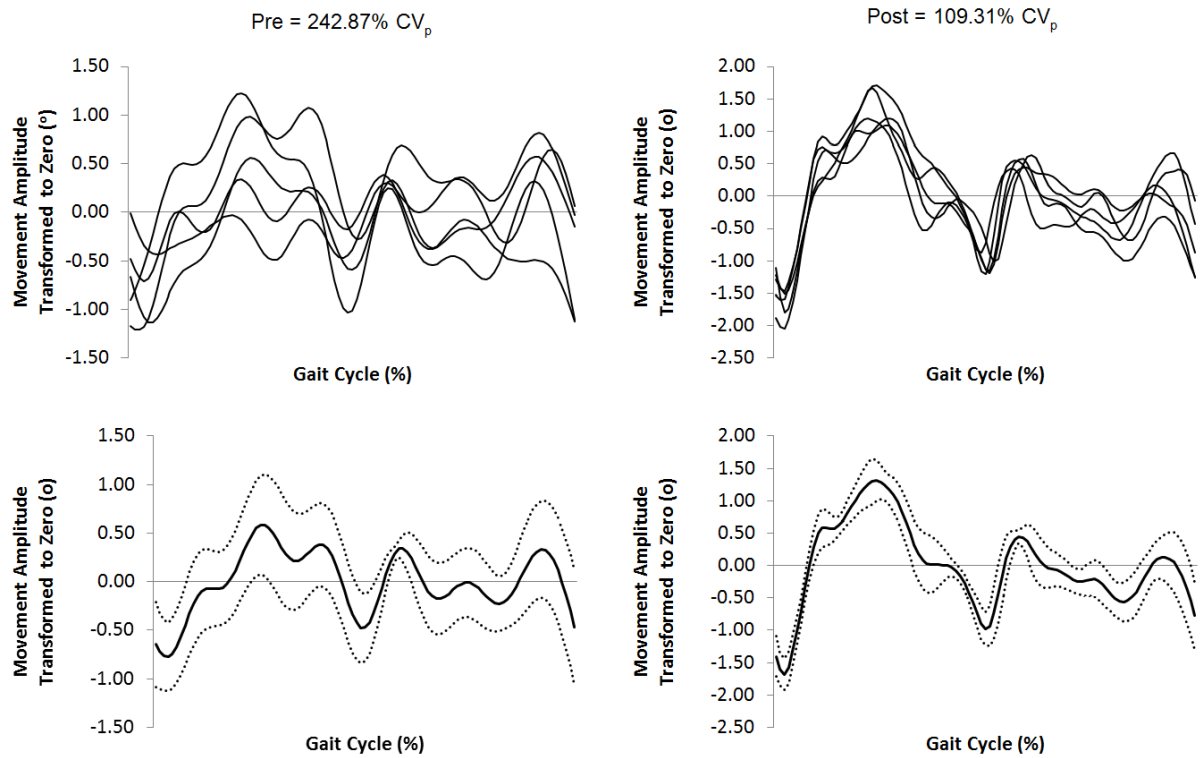
520 Figure 2. MedX Lumbar Extension Machine Restraint System (Reproduced with permission from MedX

521 Corporation)



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523 Figure 3. Marker set-up



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525 Figure 4. Example of Training Group Pre (left) and Post (right) Lumbar Kinematic Pattern Variability; top

526 graphs show individual trials kinematic waveform patterns and bottom graphs shows mean ensemble average (\pm

527 standard deviation; dotted line) for these trials; CV_p = Waveform pattern variability.

528

529 **TABLES**

530 Table 1. Baseline group demographics.

	Training (n = 17)	Control (n = 7)	<i>p</i>
Age (years)	47 \pm 13	42 \pm 15.	.645
Stature (cm)	171.90 \pm 9.26	180.82 \pm 7.70	.076
Body Mass (Kg)	75.00 \pm 15.49	85.48 \pm 18.26	.324
BMI (Kg/m ²)	25.12 \pm 3.10	25.94 \pm 4.41	.899
Symptom Duration (years)	14 \pm 11	12 \pm 11	.800
VAS (mm)	47.26 \pm 24.09	19.2 \pm 15.51	.024
ODI (pts)	34.71 \pm 12.69	26.2 \pm 7.27	.158
ILEX Strength (Nm)	177.80 \pm 83.80	192.21 \pm 67.60	.691

531 BMI = Body Mass Index; VAS = Visual Analogue Scale; ODI = Oswestry Disability Index; ILEX = Isolated

532 Lumbar Extension

533 Table 2. Changes in VAS, ODI, and ILEX strength as a result of the ILEX resistance training intervention.

Outcome		Change	95% CIs	Effect Size
VAS (mm)	Training	-23.65±21.59	-35.82 to -10.58	-1.10
	Control	10.29±18.11	-6.46 to 27.03	0.57
ODI (pts)	Training	-17.06±6.71	-20.13 to -12.67	-2.54
	Control	-1.71±7.95	-9.07 to 5.64	-0.22
ILEX Strength (Nm)	Training	41.49±30.51	24.60 to 58.39	1.36
	Control	10.29±18.11	-15.25 to 9.67	-0.21

534 95% CIs = 95% Confidence Intervals; VAS = Visual Analogue Scale; ODI = Oswestry Disability Index; ILEX =

535 Isolated Lumbar Extension

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Table 3. Pre and post ILEX resistance training intervention kinematic data

	Displacement (degrees)			CV _p (%)			CV _o (%)		
	Frontal	Sagittal	Transverse	Frontal	Sagittal	Transverse	Frontal	Sagittal	Transverse
Training									
Pre	10.61±3.74	3.92±1.20	8.85±2.72	41.95±16.62	111.99±42.64	46.49±20.57	27.48±18.34	103.94±52.78	41.69±28.15
Post	10.80±2.88	4.31±1.37	9.41±3.26	39.35±12.72	91.09±28.27*	48.20±24.02	25.87±15.02	87.95±41.10	42.35±25.28
Control									
Pre	8.15±1.94	4.13±1.78	6.91±7.87	52.65±19.23	92.95±27.07	33.41±11.74	32.30±29.09	66.33±69.07	14.15±5.46
Post	7.25±2.31	3.80±1.54	8.86±2.32	56.45±11.82	89.51±26.63	40.25±20.83	44.59±46.13	85.91±39.78	31.66±27.27

*Denotes significant change from pre to post ($p = .044$); CV_p = Waveform pattern variability; CV_o = Waveform offset variability