

Time-course changes associated with PA Lumbar Mobilizations on Lumbar  
and Hamstring Range of Motion: A Randomized Controlled Crossover Trial

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## Abstract

**Objective:** To compare the post-intervention time-course changes in Active Knee Extension (AKE) and Active Lumbar Flexion (ALF) range of motion in response to unilateral posterior–anterior (UPA) mobilizations of the lumbar spine (L4/5 zygapophyseal).

**Methods:** Twenty-four asymptomatic participants (maleness: 0.58, age [mean  $\pm$  standard deviation]:  $32 \pm 8$  y, body mass index  $25.9 \pm 2.6$  kg·m<sup>2</sup>), were recruited to a fully controlled crossover trial. Following either the intervention (L4/5 zygapophyseal mobilizations) or control, participants immediately performed the AKE and ALF tests, which were also performed at baseline. Subsequent tests were made at intervals of 5, 10, 15, 20, 25, 30, 45 and 60 minutes.

**Results:** After adjustment for baseline (mean AKE: 37.2° from full extension, mean ALF: 14.37 cm), sex and age, UPA lumbar mobilizations had a most likely moderate effect on AKE (9.8° closer to full extension;  $\pm 1.9$ ) and a likely moderate effect on ALF (1.34 cm;  $\pm 90\%$  confidence limits 0.43). The magnitude of the AKE effect became most likely small 20-minutes post-treatment (5.3;  $\pm 1.7$ ) and possibly small/ possibly trivial 60-minutes post-treatment (2.1;  $\pm 1.4$ ). For ALF, the magnitude of the effect became most likely small 15-minutes post-treatment (0.76;  $\pm 0.25$ ), possibly small/ possibly trivial 25-minutes post-treatment (0.38;  $\pm 0.18$ ), and likely trivial 60-minutes post-treatment (0.26;  $\pm 1.8$ ).

**Discussion:** UPA lumbar mobilizations increased lumbar ROM and hamstring extensibility by a moderate magnitude, with the effect reducing after 10–20-minutes post-treatment. Clinicians should consider these time-course changes when applying UPA lumbar mobilizations.

**Clinical Trials Registry:** NCT03273400

**Evidence Level:** 2b

**Keywords:** Lumbar Vertebrae, Mobilizations, Hamstring Muscles,

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## **Introduction**

Hamstring strains continue to be one of the most common musculoskeletal injuries in athletes and patients of all age ranges, genders, sports, and levels of competition [1,2]. Hamstring muscle strain injuries are common in multidirectional sports, such as American football, Australian football, cricket and English rugby union [3]. Hamstring injuries also continue to be the most prevalent musculoskeletal diagnosis in soccer, with no decrease in incidence during the last 30 years [3,4]. The impact of such an injury is substantial resulting in lost playing time and monetary loss to both players and teams in professional sport. An average injury rate of 1.20 hamstring injuries per 1000 hours of play was recorded over a thirteen-year period, with 40% of all soccer muscle injuries occurring in the region [5,6]. As such, researchers and clinicians continue to seek the optimal hamstring rehabilitation program to minimise the impact of hamstring pathology.

Hamstring rehabilitation requires a multifactorial and potential individualized approach. Nevertheless, the lumbar spine has a direct anatomical and functional relationship with the hamstring complex and is therefore considered a fundamental element of clinical hamstring management [7,8,9,10]. Specifically, spinal joint mobility facilitates lumbopelvic control and is considered an important part of hamstring rehabilitation and prevention [11,12,13]. Therefore, the use of lumbar zygoapophyseal joint (z-joint) mobilizations has been advocated in both the regeneration and functional phase of the acute hamstring injury return- to- sport algorithm [8]. How the hamstring extends in relation to the lumbar region is reported as an important modifiable risk factor for injury [14]. Decreased passive stiffness of the hamstring, defined as the ability of the tissue to allow elongation, is associated with increased risk of injury [15,16,17]. The ability of the hamstring muscle to extend allows it to absorb greater applied forces. This is of further

importance in sports requiring the optimal use of the stretch-shortening cycles generally found in multi-directional sports including soccer [14]. As reduced hamstring extensibility is a feature following hamstring injury, treatment modalities that offer evidence-based solutions to this issue will be welcomed by the clinician.

Spinal mobilizations have been shown to increase hamstring extensibility, the ability of the muscle tissue to lengthen or stretch beyond resting length, in both a general [18, 19,20,21] and elite soccer population [22]. The acute increase in hamstring extensibility, gained from lumbar mobilizations, together with reduced surface muscle electromyographic activity of the bicep femoris muscle [20] may offer a brief time-period to provide therapy to attenuate progression through rehabilitation. Unilateral Posterior Anterior (UPA) mobilizations have been found to provide superior increases in extensibility of the hamstring compared to centrally applied mobilizations [21]. However, the duration of this timeframe has not been adequately investigated. The duration of any effect from spinal mobilizations will provide the clinician with a wider appreciation of the effects this treatment modality may offer within an evidenced informed clinical reasoning framework. If clinicians are to utilize lumbar mobilizations within a multifactorial approach to hamstring management, knowledge of the intervention's duration, initially in a healthy control population is required to provide data for evaluation of its value.

Whilst there is evidence to suggest that neurophysiological effects following spinal mobilization subside after ~ 5 min [23], there is a paucity of evidence assessing time course changes in hamstring extensibility following lumbar mobilizations. Previous authors [24] have demonstrated a prolonged elevation in hamstring extensibility

immediately, and 24 h post mobilization. It is not clear how the authors controlled for confounding variables within this timeframe, or the rationale for choosing this timeframe.

Whilst the mechanisms of action are different, static stretching of the hamstring has been shown to result in prolonged increases in extensibility up to 30 min post intervention [25]. Therefore, a similar timeframe for elevated extensibility following UPA may also exist. Moreover, multiple time points should be measured to increase sampling frequency of data points to determine where the effects of the intervention may begin to subside, allowing greater accuracy for clinician decision making.

The duration of improved hamstring extensibility in the hours ensuing spinal mobilizations has yet to be fully elucidated. If those immediate improvements are indeed found to be transient, then the clinician may wish to consider the value of following such return to play treatment guidelines which incorporate lumbar manual therapy. Therefore, the primary aim of our investigation was to investigate the effect of UPA lumbar z-joint mobilizations on the time-course changes in lumbar ROM and hamstring extensibility.

## **Methodology**

### ***Study Design***

A fully controlled randomized crossover design was used to investigate the time-course changes in Active Knee Extension (AKE) and Active Lumbar Flexion (ALF) following UPA lumbar mobilizations [26]. This design was chosen to suit both the research question and constraints [26]: because our aim was to compare changes in AKE and ALF between treatment and control conditions; and the acute effects of the treatment are likely to washout in an acceptable time also the outcome measures are reliable over the washout

period (see subsequent sections), and; subjects and resources are not limited a fully controlled crossover was selected. The report of this trial is conducted within the recommendations of CONSORT for publishing non-pharmacologic intervention studies [27].

### ***Participants***

A priori estimation of sample size for magnitude-based inference in a pre–post crossover design using AKE and ALF as outcome measures yielded a minimum requirement of 24 participants (see *Statistical Analysis* for details). Participants were recruited, via means of a study flyer, from a population of students and staff at \*\*\* University, United Kingdom, between September and December 2017. Inclusion criteria included adults over eighteen without current spinal or lower limb pathology. Participants with current symptomatic low back or hamstring pain, neurological symptoms, history of spinal surgery or any contraindication to spinal mobilization were excluded [28]. All participants were considered moderately active; defined as performing moderate intensity (3-6 metabolic equivalents; METs) leisure time, and sporting (recreational) activities [29]. Given the frequency and intensity our participants engaged with per week, no participant performed an exercise intensity likely to induce delayed onset muscle soreness (DOMS) that might confound the main outcome variables. From those participants who volunteered to take part only one was excluded based on current lumbar pain. No changes were made to the methods after trial commencement. All participants provided written informed consent. Ethical approval was received from \*\*\* University’s ethics committee (Ethics Number: SSSBLREC061), in accordance with the Declaration of Helsinki. The trial was registered with clinicaltrials.gov (NCT03273400).

### ***Outcome Measures***

Two main outcome measures were assessed pre- and post-intervention and control. These measurements were taken by a qualified physiotherapist, with 22 years post graduate experience, who was blinded to the participant's condition. Active hamstring extensibility was measured by the AKE test (Figure 1). Our pilot test-retest analysis indicated excellent reliability of AKE and ALF ROM (see *Statistical Analysis* for details), which is in agreement with previous research [30,31]. The test has also been suggested to be the gold standard for hamstring muscle length, displaying good intra-rater reliability (0.87-0.94) [32]. Participant's laid supine, with one mobilization belt across the anterior superior iliac spine preventing pelvic and lumbar movement and another placed 20 cm above the tibial tuberosity of the non-dominant/non-testing leg preventing potential movement [33]. Belt positions were marked for re-measurement purposes. The hip of the dominant/testing leg was held at a 90° flexed angle by a purpose made wooden wedge. During testing the knee was extended until maximal range was achieved as determined by the participant [30]. An inclinometer (Dr Rippstein, Zurich, Switzerland), measured the degrees from full extension positioned on the anterior tibial border halfway between the inferior pole of the patella and the line between the malleoli [34]. Ankle plantar grade was maintained by a medical brace. Test performance (range of motion change from pre to post-test) was measured as the degrees (°) from full active knee extension, where full active knee extension would equal 0°.

*Figure 1 – Testing position of the Active Knee Extension Test*

\*\*\*Insert Figure 1 about here\*\*\*

The modified Schober test (mSchober) was used to measure ALF range [35,36]. This test has been demonstrated to have excellent reliability in both symptomatic and

asymptomatic populations [36, 37] and recommended for use in clinical trials [39]. Each participant was stood on a 60 cm wooden box, feet positioned 8 cm apart, indicated by tape. A skin marker was placed 5 cm below and 10 cm above the lumbosacral junction, determined by a passive physiological intervertebral movement and lumbar palpation [28,36]. Verbal instructions informed all participants to actively flex forward whilst maintaining knee extension (Figure 2). Lumbar range was recorded as the change in distance (cm) between the two skin markers measured by a tape measure (seca Germany).

*Figure 2 – Testing position of Active Lumbar Flexion*

\*\*\*Insert Figure 2 about here\*\*\*

### **Intervention**

The lumbar UPA mobilizations were applied by a physiotherapist with twelve years clinical experience and postgraduate qualifications in spinal mobilization. Throughout the application participant's laid prone on a plinth. Grade 3 UPA lumbar mobilizations, defined as large amplitude oscillations into resistance, were applied to the L4/5 unilateral z-joint for two minutes, three times [28]. Mobilizations were applied to the same side of the dominant limb identified by kicking preference. Spinal level was determined by passive physiological intervertebral movement and spinal palpation by the same physiotherapist. Mobilizations were applied at a frequency of 2 Hz maintained by a metronome, as previously evidenced to provide sympathetic nervous system excitability [39].

### **Procedure**

Participants visited a biomedical sciences laboratory on two separate occasions and received either UPA mobilizations or no mobilization (CON). The order of treatment (UPA or CON) was counterbalanced to mitigate potential order effects, conducted via electronic software (Microsoft Excel©), by an individual independent and therefore blinded to the study. Each participant attended on the same day at the same time, one week apart. Following either UPA or CON, participants immediately performed a test of AKE and ALF. During the CON arm of the trial participant's lie prone on a plinth for a ten-minute period, the time it took for the clinician to explain, identify and perform the lumbar mobilizations. To mitigate the effect of repeated assessment causing natural variations in tissue extensibility five AKE and four ALF were conducted prior to the initial recorded assessment [20,21,22]. At repeated re-measurements the AKE and ALF were tested only once so not to influence tissue extensibility and measurement outcome. Subsequent tests were made at intervals of 5, 10, 15, 20, 25, 30, 45 and 60 minutes. The 5 min intervals were chosen to coincide with the diminishing returns reported from neurophysiological responses [24], to provide an adequate sampling frequency for investigating time-course changes (i.e. identify any substantial change with an accuracy of 5 minutes), and to avoid any confounding effects from subsequent intervals. AKE and ALF assessments were performed in a counterbalanced order both within- and between-participants at each time point.

### ***Statistical Analysis***

Prior to the main experimental trials, we performed a pilot study in which participants (n = 15) visited the laboratory on two occasions, separated by one week, and performed assessments of AKE and ALF. A pairwise analysis of consecutive trials was then performed, using a custom-made spreadsheet [40], to assess the reliability of AKE and

ALF. Typical error, the pure between-participant standard deviation (SD), and the intraclass correlation coefficient was  $3.3^\circ$  from full extension (90% confidence limits [CL]  $2.7$  to  $4.4^\circ$  from full extension),  $10.4^\circ$  from full extension ( $7.1$  to  $12.8^\circ$  from full extension), and  $0.92$  ( $0.84$  to  $0.96$ ) for AKE, and  $0.72$  cm ( $0.58$  to  $0.96$  cm),  $1.70$  cm ( $1.37$  to  $2.25$  cm), and  $0.83$  ( $0.68$  to  $0.91$ ) for ALF. Subsequently, we estimated the minimum sample size required to produce acceptable error rates and adequate precision, defined by 90% confidence interval, for a difference in changes in means in a pre–post crossover trial evaluated with non-clinical magnitude-based inference [41]. Using the aforementioned statistics and with a smallest important standardized difference of 0.20 multiplied by the between-participant SD [41], sample sizes of at least 15 and 24 participants were deemed appropriate for AKE and ALF, respectively.

Prior to analysis, assumptions of normality were checked using visual inspection of the raw data via histograms and Q-Q plots. Raw data was seen to follow a normal distribution and is presented as the mean  $\pm$  SD. We used linear mixed models (SPSS V23, Armonk, NY: IBM Corp.) with fixed (condition [UPA or CON]; with intercept) and random effects (participant; without intercept) to examine the pre–post, treatment–control differences in AKE and ALF. The analysis of covariance approach was adopted whereby change scores were treated as the dependent variable and the baseline (i.e. ‘pre’) value was specified as a covariate [42]. Effects were also adjusted for sex and age. Uncertainty in the estimates was expressed as 90% CL. Standard deviations for individual differences in response to the UPA treatment (vs control) were estimated via the model’s random effects (variance components). Negative SDs (i.e. more variation in response to CON) were manually calculated using standard errors of the change score estimated marginal means [42]. This novel method identifies responders by accounting for variability in the change scores in

the control group rather than inappropriately using the change scores from the treatment group alone [43].

Evaluation of the size and uncertainty of the pre-post, treatment-control differences in AKE and ALF made using the magnitude-based inferences [41]. Prior to analysis, we performed an exhaustive search of the literature to obtain known reference values for minimum clinically important differences/changes in AKE and ALF with respect to health and/or performance. We were unable to find any research providing such data. Therefore, in the absence of clinically meaningful reference values, standardized thresholds of 0.2, 0.6, and 1.2 multiplied by the baseline between-participant SDs were calculated to anchor small, moderate and large effects, respectively [41]. Baseline between-participant SDs were pooled from both conditions (control and treatment) then adjusted for small sample bias. Inference was then based on the probability distribution of the true effect in relation to these thresholds using a custom-made spreadsheet [44]. The probability (percentage chance) that observed effects were at least greater than their nearest lower thresholds were evaluated using the following scale: 25.0–74.9% possibly; 75.0–94.9% likely; 95.0–99.4% very likely;  $\geq 99.5\%$  most likely [41]. All effects were evaluated mechanistically, whereby a difference was deemed unclear if its chance of being both substantially positive and negative was  $\geq 5\%$  (based on the threshold for a small effect [0.2 SDs]). Finally, SDs representing individual responses to UPA mobilizations were double before interpreting their magnitude against the above standardized thresholds [45].

## **Results**

### ***Descriptive Data & Main effects***

*Figure 3 – Descriptive (mean ± standard deviation) AKE (A) and ALF (B) data at each time point for UPA mobilizations and CON*

*\*\*\* insert Figure 3 about here \*\*\**

A total of twenty-four participants (maleness: 0.58, age [mean ± standard deviation]: 32 ± 8 y, body mass: 81.6 ± 8.0 kg, stature: 177 ± 10 cm, body mass index 25.9 ± 2.6 kg·m<sup>2</sup>) were recruited to and completed the study. Descriptive data for AKE and ALF in response to CON and UPA mobilizations are presented in Figure 3. The time-course pre–post, treatment–control differences in AKE and ALF are presented in Figure 4. Differences are adjusted to sex, a mean age of 32, a baseline AKE of 37.2° from full extension and a baseline ALF of 14.37 cm. UPA mobilizations had a most likely moderate effect on AKE (Figure 4A) and a likely moderate effect on ALF (Figure 4B). For AKE, the effects of UPA mobilizations remained most likely to likely moderate 5- and 10-minutes post-treatment and became: possibly moderate/most likely small 15-minutes post-treatment, most likely and very likely small 20- to 25-minutes post-treatment, likely small at 30- and 45-minutes post-treatment, and possibly small/ possibly trivial at 60-minutes post-treatment (Figure 4A). For ALF, the effect of UPA mobilizations remained likely moderate 5-minutes post-treatment and became: possibly moderate/most likely small 10-minutes post-treatment, most likely small 15- and 20-minutes post-treatment, possibly small/ possibly trivial 25- and 45-minutes post-treatment, and likely trivial 60-minutes post-treatment (Figure 4B).

*Figure 4 – Time-course changes in AKE (A) and ALF (B) following UPA mobilizations. Data are presented as the treatment-control differences for each time point change from*

*baseline (i.e. 'pre'). Data points are presented with 90% confidence limits and standard deviations for the interindividual responses to UPA mobilizations versus control*

*\*\*\* insert Figure 4 about here \*\*\**

### ***Individual Responses***

For AKE, SD representing interindividual responses to UPA mobilizations were moderate immediately and up to 10-minutes post-treatment, and small at all time points from 15- to 30-minutes post-treatment (Figure 4A). AKE Individual response SD were negative for 45- (-1.6° degrees closer to full extension) and 60-minutes (-3.5° degrees closer to full extension) post-treatment, indicating greater variance following CON when compared with UPA. For ALF, interindividual response SDs were large immediately and 5-minutes post-treatment, moderate at 10- and 15-minutes post-treatment, and small at all time points from 20- to 60-minutes post-treatment (Figure 4B).

### **Discussion**

The hamstring complex continues to be a problematic region to prevent and treat injury. The value of treating the hamstring region proximally via the lumbar spine has been advocated by researchers and is included in management algorithms [8,11-13]. Specially, z-joint mobilizations have been advocated as the mobilization technique of choice to increase ROM in both the lumbar and hamstring regions [21]. The duration of these observed changes is yet to be adequately investigated. To date, this is the first study to investigate the magnitude of the time-course changes in ROM for both the lumbar and hamstring region.

The key findings from our study in healthy, recreationally active controls were: 1) the application of UPA mobilizations resulted in moderate improvements to AKE and ALF, 2) the magnitude of the effect substantially reduced 20- and 15-minutes post-treatment for AKE and ALF, respectively, with further reductions in effect magnitudes and uncertainty evident until 60-minutes post-treatment, and 3) moderate and moderate-to-large individual responses to UPA were evident up to 10- and 15-minutes post-treatment for AKE and ALF, respectively, with the magnitude of individual responses at all subsequent time points being small to trivial.

Our results support previous research indicating that lumbar z-joint mobilizations produce similar responses to increase lumbar and hamstring ROM. The mean effects of 9.8 degrees AKE for a baseline of -37.15 is around 26%. For ALF, the effect of 1.3 cm on a baseline of 14.4 is ~9.4%. Szlezak et al [19] have demonstrated similar increases in hamstring extensibility using the straight-leg raise (SLR) test, with a mean difference of 8.5 degrees post mobilization. Chesterton et al [20-22] have reported small to moderate effects of increased ROM following mobilizations similar to those found within this study. These similarities are likely due to the healthy populations and mobilization protocols used.

There is limited research that has investigated the time course changes of lumbar mobilizations. As such, our study provides novel data to suggest that the moderate effects lumbar mobilizations have on AKE and ALF ROM appear to last up to 20- and 15-minutes post-intervention, respectively. The magnitude of the effect substantially reduces following these time points, with AKE ROM remaining increased by at least a possibly small magnitude (versus pre-intervention) through 60-minutes post-intervention, and

ALF ROM remaining increased by at least a possibly small magnitude through 45-minutes post-intervention before returning to near baseline (likely trivial difference) at 60-minutes post-intervention.

Ganesh et al [24], replicated Szlezak's study [19] protocol with a 24 h re-test of the straight-leg raise following UPA mobilizations. The authors utilised a different protocol with multiple levels of mobilizations (L1-S1) and measured hamstring length via the neurally biased straight-leg raise test. Improvements were reported both immediately following application and at the 24 h follow-up measure. It is unclear why Ganesh et al [24] have reported prolonged elevation in ROM up to 24 h where we have demonstrated effects subside after 15-20 minutes. Due to the lack of data points it is difficult to draw conclusions as to where the effects of the intervention begin to subside. Furthermore, it is not clear on why a 24 h time point was chosen given the transient and short-lived changes observed in neurophysiological responses. Finally, it is not clear how the authors controlled for confounding variables (e.g. activity levels) in the proceeding 24 h timeframe that may contribute to greater ROM seen at 24 h.

Neurophysiological responses to mobilizations have been reported to subside after a shorter timeframe than the ROM changes. Perry and Green [39] reported that skin conductance increased for a period of less than five minutes in a population of 45 healthy subjects. However, further measurements were not taken beyond 5 minutes. Whilst in this study, UPA mobilizations applied to the L4/5 region resulted in side specific changes in the sympathetic nervous system (SNS), it is not clear how these changes translate into observed biomechanical changes to facilitate clinician decision making on further treatment programmes.

There is a lack of current research into the time course changes of spinal manual therapy for comparison to this study. Hatano et al, [25], reported static stretching of the hamstring can maintain length changes at 30 min post intervention. This study similarly used an asymptomatic population, with measurements of hamstring extensibility taken at 10, 20 or 30 minutes. Increased extensibility was maintained for approximately 30 minutes post intervention like the acute response found in our study. The authors failed to report individual differences or control for gender, which further limits direct comparison to our results.

We report for the first time moderate to large individual responses in AKE and ALF ROM following UPA mobilizations, lasting up to 10–15-minutes post-intervention. These can be considered real interindividual responses to UPA mobilizations in healthy, recreationally active participants, since we removed any source of error arising from measurement inaccuracy or biological variation by using a control condition [46]. Interestingly, these seemingly moderate to large individual responses were observed despite controlling for age, sex and baseline ROM (specific to each test). It may therefore be of value and interest for clinicians and researchers to consider other factors that may reasonably moderate the response to UPA mobilizations, inclusive of the central nervous system [46]. Previously, individual differences were once considered tissue related however the paradigm shift has led us to acknowledge the factors associated with the central nervous system may be important [46].

It is beyond the scope of our study to understand the mechanisms for the observed duration of ROM changes. However, the SNS changes described by previous authors [39,

47-50] suggest that spinal mobilizations stimulate the dorsal peri-aqueductal (dPAG) region of the brain which in turn produces a SNS response. It is this response which produces the proposed benefits of manual mobilization including analgesia, sympathoexcitation and motor facilitation [51]. A paradigm shift has taken place over recent years with evidence suggesting the benefits of manual mobilization may not purely be due to a biomechanical mechanism but a neurophysiological one. However, if the neurophysiological effects return to baseline after 5-10 min, the mechanism for longer duration effects in ROM reported in our study and by Ganesh et al [24] require further investigation.

This study utilised active tests rather than passive to assess the influence of the intervention on functional outcomes measures. As well as being appropriate outcomes measures the AKE and ALF are both feasible for clinicians to apply in practice. Both outcome measures are considered reliable and valid [30,31]. Furthermore, it is worth noting that AKE was measured from full knee extension classed as zero degrees. Normative values in literature [32, 52] have been collected using different measurement methods, Youdas et al [52] with full extension as 180 degrees and Neto et al [32] as full extension measured from the 90-degree starting position. When comparing our results to normative values exact AKE measurement should be considered.

Recent hamstring injury treatment algorithms [8] have proposed the progression of rehabilitation from the sub-acute to functional phase when full hamstring extensibility has been restored. As the changes evident from our study are only short-term clinicians may want to use this short-time period to apply additional therapeutic interventions. For example, exercise therapy could be performed in functional positions that may not have

been achievable without the increased ROM in the hamstring and lumbar spine. Making use of this ‘window of opportunity’ following manual therapy has also been proposed by Piekarz and Perry [53] who suggested that the clinician could attempt to restore joint range of movement and pain free movement following spinal manual therapy. A broader appreciation of the effects of manual therapy should include the possible placebo effect experienced which can have an effect on motor performance in addition to pain modulation [54]. Advanced neurobiological testing procedures have led to a greater understanding of the physical performance changes associated with a placebo response which may be explained by a top down modulation of sensory and motor systems [55]. Whilst the placebo response is unlikely to be the only mechanism responsible for the extensibility gains reported in this study the placebo response should be a considered when explaining the effects of manual therapy [56].

### Limitations and Future Research

All measures were conducted on asymptomatic individuals to understand the magnitude of effect, and duration of the intervention, in a healthy population. Therefore, the findings are not transferable to individuals experiencing pain. Now this proof-of concept has been established, further research should be conducted specifically in patients and athletes with lumbar and hamstring symptoms to determine if similar timeframes are still evident. The use of a default smallest worth change was used in the absence of a minimal clinically important difference. Therefore, we cannot be certain that these small increases in ROM will lead to positive meaningful outcomes in return to play. Knowledge of the MCID could be derived from well-designed and robust validity, cohort, or case control studies, as well as prognostic-type studies in which AKE and ALF is the predictor and injury risk or athletic performance, for example, is the outcome [57]. Currently, no data exists to

provide reference values for lumbar/hamstring extensibility in relation to injury risk.

While we acknowledge this as a potential limitation to our present research, it is also a broader limitation within the discipline of sports medicine. Further study is therefore required to establish MCID values for outcome measures used in research and practice.

## **Conclusion**

Hamstring injuries continue to be a challenging injury to prevent and manage in the sporting population. Whilst we acknowledge that the management of these injuries should be multifactorial, spinal mobilizations have an important role in early hamstring injury rehabilitation. However, the magnitude of effect and underlying mechanisms has not been fully established. This study supports previous findings demonstrating that the lumbar and hamstring flexibility is increased following unilateral mobilization. The main and novel finding of our study is that the moderate effects of UPA mobilizations on lumbar and hamstring ROM are brief, lasting up to 15–20 minutes, with substantial individual responses apparent. Therefore, it is possible that clinicians could use this timeframe appropriately to prescribe any subsequent exercises in which applying load through greater outer ranges. However, it is important to consider these results based on the healthy, asymptomatic population recruited for this study, and further research is warranted to further elucidate the effectiveness of this intervention on a symptomatic population.

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