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ARTICLE

# The Relationship Between Lead Hip Rotation and Low Back Pain in Golfers– A Pilot Investigation

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Reduced lead hip mobility has been proposed to be associated with low back pain (LBP) in golfing populations, although the literature is contradictory. This study aimed to clarify this relationship by recording the magnitude of hip rotation during the golf swing and a range of clinical assessments. Sixteen recreational male golfers (8 with a history of LBP and 8 age and anthropometrically matched controls) performed their golf drive with their hip movement recorded. Hip range of motion was also recorded in sitting, lying prone and standing and correlated to hip rotation during the golf drive. The LBP group demonstrated reduced lead hip internal rotation (LHIR) compared to those in the no pain (NP) group when using the prone passive and standing clinical measures ($p < .01$), but not sitting. The prone passive measure of LHIR was moderate-strongly correlated to golf swing LHIR r = 0.560; $p = .024$. The LBP group had an average of 8° less LHIR during the golf swing than the NP group and were closer to end or range, although both were non-significant trends. These results may have implications for LBP prevention and rehabilitation strategies in golf populations.

***Keywords:*** back pain, golf, golf swing, kinematics

The most common pain problem affecting both amateur and professional golfers is low back pain (LBP) (Bulbulian, Ball, & Seaman, 2001), with the reported incidence ranging from 25% to 55% (Fradkin, Cameron, & Gabbe, 2005; Gluck, Bendo, & Spivak, 2008; Gosheger, Liem, Ludwig, Greshake, & Winkelmann, 2003; Lindsay & Horton, 2002; McHardy, Pollard, & Luo, 2007). While LBP is known to be a multi-factorial condition (Waddell & Burton, 2001), given the dynamic nature of the golf swing a mechanical aetiology is proposed, with several clinical measures, including reduced lead hip mobility, suggested to play a potentially important role (Kim, You, Kwon, & Yi, 2015; Tsai et al., 2010). Reduced internal rotation range

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of movement measures of the lead hip has been associated with LBP in golfers (Gulgin, Armstrong, & Gribble, 2010; Murray, Birley, Twycross-Lewis, & Morrissey, 2009; Vad et al., 2004). This has led to recommendations to improve hip internal rotation flexibility as a means of preventing and managing LBP in golfers. However, this may be premature given the number of uncertainties remaining regarding this relationship based on current evidence.

Firstly, the relationship in the literature, pertaining to reduced mobility in LHIR and LBP is both contradictory and limited. When hip rotation was measured using an inclinometer with participants lying prone, both amateur and professional male golfers with LBP were found to have reductions in passive and active LHIR compared to; (i) their non-lead hip internal rotation, (ii) their lead hip external rotation (LHER) and, (iii) the LHIR of a group of golfers with no LBP (Gulgin et al., 2010; Murray et al., 2009; Vad et al., 2004). A further study found contradictory results, reporting no difference in passive hip rotation recorded using a goniometer in sitting between golfers with no history of LBP and those with LBP (Tsai et al., 2010). These contradictory results might be explained by the different method of hip rotation assessment (i.e., sitting versus prone), although this has not been verified (Tsai et al., 2010).

Secondly, the direct mechanism explaining the relationship between reduced LHIR and LBP is unclear. Reduced LHIR may result in increased torsional and compressive forces on the lumbar spine during the golf swing, although to date, there is no evidence for this (Grimshaw & Burden, 2000; Vad et al., 2004). Further, the relationship between these clinical assessments of LHIR have not been compared to what occurs during the golf swing at the lead hip in golfers with a history of LBP. Quantifying this relationship is a necessary first step in understanding the potential mechanisms linking reduced LHIR and LBP in golfers.

The primary aim of this pilot study was to examine the relationship between LHIR during the golf swing between golfers with and without LBP. The second aim was to quantify the relationship between LHIR using a range of clinical measures, including weight bearing, and that measured during the golf swing. We hypothesized that golfers with a history of LBP would demonstrate reduced LHIR during the golf swing, and with clinical measures of hip rotation, compared to the group with no history of LBP.

## Materials and Methods

This study received approval from the relevant Human Research Ethics Committee (PT219/2012) and all participants provided written informed consent prior to data collection. Sixteen recreational golfers aged ≥18 years who currently played or practiced golf for an average of ≥4 hours per week over the past 24 months participated in this study. Participants were included in the LBP group if they had experienced two or more episodes of LBP that were aggravated by golf and prevented them from playing in the 24 months preceding the data collection, with their most recent LBP episode occurring in the six months prior to study commencement. Participants were included in the no LBP (NP) group if they had no history of LBP over the two years preceding the data collection. Participants were excluded if they presented with the following conditions: a specific spinal pathology (e.g.,

infection, inflammatory disorder, fracture, cauda equina syndrome) (Airaksinen et al., 2006); previous spinal fusion surgery; current or recent history of spinal radicular pain; current pain of intensity >3/10 anywhere in their body including LBP (Collins, Moore, & McQuay, 1997); taking pain medication for LBP; hip and knee arthroplasty; illness at the time of testing. There were no differences in age, height, mass, golf handicap and experience and weekly time spent playing golf between the two groups (Table 1).

## Clinical Measures

Participants attended the motion analysis laboratory at Curtin University. The sitting and prone protocols were performed according to previously described protocols (Murray et al., 2009; Tsai et al., 2010). In order to localize the measure to the hip joint in the prone protocol, the pelvis was stabilized with a belt at the level of the posterior inferior iliac spines. The measured hip was placed in 0° of abduction and knee flexed to 90°. The contralateral hip was abducted to 30°. An inclinometer was positioned on the participant's shin. The leg was then rotated both medially and laterally and the measure recorded at the when resistance was met or compensatory movement at the pelvis became evident (Murray et al., 2009).

The sitting protocol as used in (Tsai et al., 2010), involved the hip joint placed in 90° flexion in the neutral position and the knee joint adjusted to 90° flexion sitting over the edge of a plinth. The hip was passively and actively rotated for range of motion measurements. A goniometer was utilized to measure final position of the shin relative to the vertical. Measures were recorded when compensatory movement (i.e., hip lifting from the table) begun to occur or final resistance was met.

The standing assessment of hip rotation required each participant to stand on a large piece of plastic card with their back against the wall. Two lines were then marked on the plastic card that corresponded to the bilateral acromion processes (i.e., shoulder width). The participants then positioned their feet such that the lines bisected the midpoint of the heel and the space between the hallux and second toe (line 1). To achieve optimum hip internal and external rotation the participant was asked to lift their toes and swivel on the heel of their foot without changing any pressure in weight distribution from normal standing, with their hip and

**Table 1 Means and Standard Deviation for the Characteristics of the Low Back Pain and no Back Pain Groups**

| | LBP | NP | 95% CI | p Value |
|---|---|---|---|---|
| Age (yr) | 36 (15) | 26 (4) | −23 to 2 | .08 |
| Height (cm) | 180 (8) | 181 (7) | −7 to 9 | .76 |
| Mass (kg) | 81 (10) | 79 (10) | −14 to 8 | .60 |
| Handicap | 4.5 (4.2) | 1.5 (4.1) | −7 to 1 | .17 |
| Golf experience (yr) | 19 (9) | 14 (6) | −13 to 4 | .28 |
| Weekly golfing time (hr) | 19 (16) | 23 (15) | −12 to 21 | .58 |

Note. LBP = low back pain; NP = no pain; CI = confidence interval.

knee extended to move the hip into maximal internal and then external rotation positions. The tester stabilized the pelvis against the wall to ensure that only hip rotation was performed. When the participant achieved end of range rotation, a second line bisecting the midpoint of the heel and the space between the hallux and second toe was drawn (line 2). The angle between the starting and end of range lines was then measured using a goniometer. The order that the clinical measures were performed was randomized by leg (right or left) and by hip rotation (internal and external). Each measure was repeated three times. Finally, a subset of 7 participants (3 from the LBP group and 4 from the NP group) attended a second data collection 1 week following their initial attendance where the clinical measurement protocol was repeated.

## Laboratory Analysis

The participant's hip rotation during the golf swing was recorded using a 14 camera Vicon Nexus (Oxford Metrics, Inc.) motion analysis system operated at 250Hz. This system is considered a gold standard motion analysis system with reconstruction errors <1 mm (Ehara et al., 1997; Richards, 1999). Reflective markers were secured to each participant's pelvis (left and right anterior, superior iliac spines; and left and right posterior superior iliac spines) thighs (t-bar of 3 markers placed on the most lateral aspect of each thigh) and knees (medial and lateral femoral condyles) using low allergenic tape. The pelvis and thigh marker set followed a cluster based approach and was in accordance with the International Society of Biomechanics recommendations (Besier, Sturnieks, Alderson, & Lloyd, 2003; Wu et al., 2002). A static trial was utilized to calibrate the knee joint center (centroid of medial and lateral femoral condyle markers) and hip joint center (regression equation) using validated methods (Besier et al., 2003).

The data collection begun with each golfer completing a five-minute cycling warm-up using a stationary bike at a comfortable cadence with minimal resistance. They were encouraged to undertake any other self-directed exercise and stretches that they typically performed prior to playing golf (Gulgin et al., 2010). Each participant was then given the opportunity to adopt his usual golfing stance on a two meter square of synthetic grass turf and complete at least 10 practice/familiarization golf swings. Each participant used their own driver club to strike plastic practice balls (X-tech Inc.) from a rubber tee. In order to increase the ecological validity of the golf drive execution, a fairway was projected onto a screen secured three meters in front of the participant. Golfers received standardized instructions to perform their drive as they normally would on a golf course and were permitted to include any usual pre swing routine. Following familiarization, five golf swings were performed. Following the golf swing trials, the measures of hip rotation were recorded in standing, sitting and prone. The order of performing the clinical measures was intentionally varied between participants.

## Data Management of Golf Shots and Clinical Measures of Hip ROM

The Vicon system data was labelled and inspected for breaks as a result of marker occlusion. Breaks of <15 frames during the time period of interest were corrected

with cubic spline interpolation using the Vicon Nexus software (Oxford Metrics, Inc., Oxford, U.K). No breaks larger than this occurred. A residual analysis was performed prior to the data being filtered using a Woltring filtering routine and a mean square error of 1 (Woltring, 1986). A mathematical model was then utilized to calculate hip rotation (the thigh relative to the pelvis) throughout the golf swing (Besier et al., 2003).

In brief, the thigh anatomical coordinate system was created first from the y axis (knee joint center to hip joint center), followed by right hand z axis (medial femoral condyle to lateral femoral condyle). The cross product of these two axes was used to calculate the positive x axis. The pelvis anatomical coordinate system was calculated first using the mid point of the markers on the left and right anterior superior iliac spines and the mid point of the markers placed on the left and right posterior superior iliac spines to calculate the x axis. The z axis was then defined from the left anterior superior iliac spine to the right anterior superior iliac spine. Finally, the y axis was calculated from the cross product between the x and z vectors. Hip rotation was then calculated from thigh anatomical segment rotation relative to pelvis segment rotation using a ZXY Eular angle decomposition.

A customized LabVIEW 2011 SP1 program (National Instruments, Inc.) then output the peak internal and external hip rotation during the golf swing. The golf swing was defined as beginning the instant the club moved away from the ball in the backswing, until the end of follow through. The mean of the measures collected during the first three trials was used for analysis (Mullineaux et al., 2001). In the instance that the data from one of these trials was corrupted, the fourth then the fifth trial data was utilized. For all clinical measures of hip ROM, the mean of the three trials was applied.

## Statistics

Data were transferred to the Statistical Package for the Social Sciences (SPSS version 21; SPSS, Inc., Chicago, IL, USA) for analyses. The distribution of the data was assessed using Shapiro-Wilk test and frequency histograms. All data approached a normal distribution and all outliers were removed > 2 SD's from the mean. In order to confirm the reliability of the clinical measures intra-class correlation coefficients (ICCs) were calculated from between day testing on a subset of the population ($N = 7$). In order to answer the primary research question, a series of independent sample t-tests were used to compare the measures of LHIR between the LBP and no pain groups during the golf swing and clinical measures. The second research question was addressed by calculating Pearson's correlation coefficients between the clinical measure and golf swing measures of LHIR. A correlation of 0.1 to 0.3 was considered weak, 0.3 to 0.5 moderate and above 0.5 considered strong.(Bosco, Aguinis, Singh, Field, & Pierce, 2015) Finally, the results of the lead hip rotation during the golf swing were calculated relative to end of range as determined during the prone passive assessment of hip rotation, for both groups. These results were compared in a between groups t-test.

A sample size calculation indicated that 8 participants per group was sufficient to detect a difference of 15° ± 10° ($1\text{-}\beta = 0.8$; $\alpha = 0.05$) in LHIR between the LBP

and NP groups. Given the large number of comparisons, the α value for statistical significance was adjusted such that $p \leq .01$ was used to denote statistical significance.

## Results

The lead hip internal and external rotation and non-lead hip internal and external rotation measurements recorded during the golf swing were not significantly different between the LBP and NP groups (Table 2). Notwithstanding these non-significant findings, it is noteworthy that the LBP group had an average of 8° less peak LHIR than the NP group.

The comparisons of each of the clinical measures of hip rotation revealed that the LBP group internally rotated their lead hip on average 20° less during the active standing assessment and on average 16° less for the passive prone assessment (Table 3). There were no differences between groups for the clinical measures in the sitting position.

The novel assessment of standing hip rotation demonstrated strong between day reliability, although other established methods were stronger (Table 4).

Neither the standing (r = 0.325; $p$ = .220: Figure 1a) nor prone (r = 0.560; $p$ = .024; Figure 1b) measure of hip rotation were significantly correlated to the magnitude of LHIR during the golf swing, although the correlation for the prone assessment was moderate-strong.

Finally, when peak LHIR during the swing was calculated relative to each participant's end of range (prone passive assessment), there was no significant difference between groups (pain 16° ± 11; no pain 22° ± 11, 95% CI – 6 to 17, $p$ value .370). Although it is noteworthy that the pain group were on average 6° closer to end of LHIR range during the golf swing.

## Discussion and Implications

This is the first study to measure LHIR in male golfers with and without LBP, during the golf swing. The results of this pilot study provide some clarity regarding the relationship between LHIR and LBP in golfers**.** The results of this pilot study

**Table 2 Peak Internal and External Range of Hip Movement During the Golf Swing between the LBP and NP Groups**

| Variable | LBP Mean (SD)° | NP Mean (SD)° | 95% CI for the Difference | p Value |
|---|---|---|---|---|
| Lead External | 30 (9) | 27 (11) | −14 to 8 | .54 |
| Lead Internal | 8 (12) | 16 (11) | −20 to 4 | .18 |
| Non-Lead External | 25 (9) | 24 (9) | −10 to 9 | .88 |
| Non-Lead Internal | 3 (8) | 6 (15) | −16 to 10 | .67 |

Note. LBP = low back pain; NP = no pain; CI = confidence interval; Lead = lead hip; Non-Lead = Non-Lead hip.

**Table 3 Clinical Measures of Hip Range of Movement between the LBP and NP Groups**

| Clinical Measure | LBP Mean (SD)° | NP Mean (SD)° | 95% CI for the Difference | p Value |
|---|---|---|---|---|
| **Standing Measures** | | | | |
| Active Non-Lead Internal | 30 (7) | 41 (12) | −22 to −1 | .04 |
| Active Non-Lead External | 59 (9) | 55 (6) | −4 to 12 | .32 |
| **Active Lead Internal | 26 (9) | 46 (7) | −29 to −11 | <.01 |
| Active Lead External | 63 (11) | 55 (9) | −3 to 18 | .16 |
| **Prone Measures** | | | | |
| Active Non-Lead Internal | 28 (9) | 35 (11) | −18 to 3 | .14 |
| Active Non-Lead External | 49 (10) | 41 (15) | −6 to 22 | .23 |
| Active Lead Internal | 25 (11) | 38 (10) | −24 to −2 | .03 |
| Active Lead External | 54 (11) | 40 (12) | 1 to 25 | .03 |
| Passive Non-Lead Internal | 30 (8) | 39 (12) | −19 to 2 | .10 |
| Passive Non-Lead External | 51 (11) | 42 (15) | −6 to 22 | .23 |
| **Passive Lead Internal | 26 (10) | 42 (8) | −27 to −7 | <.01 |
| Passive Lead External | 56 (9) | 42 (10) | 3 to 24 | .02 |
| **Sitting Measures** | | | | |
| Active Non-Lead Internal | 29 (10) | 32 (11) | −15 to 8 | .49 |
| Active Non-Lead External | 32 (8) | 25 (8) | −2 to 15 | .11 |
| Active Lead Internal | 26 (8) | 33 (9) | −17 to 2 | .10 |
| Active Lead External | 38 (6) | 28 (8) | 2 to 17 | .02 |
| Passive Non-Lead Internal | 36 (6) | 44 (14) | −20 to 4 | .17 |
| Passive Non-Lead External | 38 (7) | 36 (12) | −9 to 12 | .76 |
| Passive Lead Internal | 30 (9) | 41 (13) | −22 to 1 | .08 |
| Passive Lead External | 49 (5) | 41 (12) | −2 to 17 | .13 |

** All variables were statistically significant ($p < .01$). Note. CI = confidence interval; LBP = low back pain; NP = no low back pain; Lead = Lead hip/left hip; Non-lead = Non-lead hip/right hip.

indicate that golfers with LBP had an average of 8° less peak LHIR during the golf swing compared to the NP group, and were on average 6° closer to their end of LHIR range. These differences in LHIR during the swing, whilst not statistically significant, may be of sufficient magnitude to suggest a clinically meaningful difference and warrant further investigation in a larger group.

The results of this study also identified that golfers with LBP have significantly less LHIR measured in prone and standing than golfers without LBP with moderate-strong correlation observed between the LHIR during golf swing and the passive prone measure. While there is some conjecture in the literature regarding the relationship between LHIR and LBP in golfers (Murray et al., 2009; Tsai et al., 2010; Vad et al., 2004), the results of this study support that the sitting clinical

**Table 4 Reliability of the Clinical Measures of Hip Range of Movement**

| Clinical Measure | Day 1 (°) | Day 2 (°) | ICC | 95% Confidence Interval |
|---|---|---|---|---|
| **Standing Measures** | | | | |
| Active Non-Lead Internal | 36.0 (8.9) | 35.6 (9.1) | 0.67 | 0.3 to 0.86 |
| Active Non-Lead External | 59.7 (14.8) | 61.7 (13.1) | 0.87 | 0.70 to 0.94 |
| Active Lead Internal | 35.9 (12.0) | 38.5 (11.3) | 0.91 | 0.80 to 0.96 |
| Active Lead External | 55.2 (7.7) | 58.1 (12.3) | 0.83 | 0.63 to 0.92 |
| **Prone Measures** | | | | |
| Active Non-Lead Internal | 36.5 (12.0) | 36.4 (9.0) | 0.89 | 0.75 to 0.95 |
| Active Non-Lead External | 44.3 (12.9) | 43.7 (9.3) | 0.85 | 0.67 to 0.94 |
| Active Lead Internal | 24.0 (11.6) | 36.0 (12.5) | 0.93 | 0.84 to 0.97 |
| Active Lead External | 48.9 (14.7) | 44.2 (13.9) | 0.92 | 0.81 to 0.97 |
| Passive Non-Lead Internal | 39.0 (11.5) | 39.5 (9.0) | 0.90 | 0.80 to 0.90 |
| Passive Non-Lead External | 46.5 (11.4) | 46.4 (8.6) | 0.89 | 0.76 to 0.94 |
| Passive Lead Internal | 37.0 (12.3) | 37.2 (11.4) | 0.98 | 0.96 to 0.99 |
| Passive Lead External | 50.8 (14.5) | 45.7 (13.9) | 0.96 | 0.91 to 0.98 |
| **Sitting Measures** | | | | |
| Active Non-Lead Internal | 32.5 (7.7) | 33.2 (6.9) | 0.96 | 0.90 to 0.98 |
| Active Non-Lead External | 30.2 (8.9) | 29.4 (7.9) | 0.91 | 0.79 to 0.96 |
| Active Lead Internal | 31.3 (11.5) | 29.1 (10.1) | 0.95 | 0.89 to 0.98 |
| Active Lead External | 30.2 (9.4) | 30.2 (8.7) | 0.80 | 0.57 to 0.91 |
| Passive Non-Lead Internal | 41.3 (13.3) | 43.8 (11.6) | 0.94 | 0.94 to 0.97 |
| Passive Non-Lead External | 43.3 (13.1) | 40.6 (15.0) | 0.97 | 0.94 to 0.98 |
| Passive Lead Internal | 42.0 (13.5) | 40.9 (14.8) | 0.96 | 0.90 to 0.98 |
| Passive Lead External | 41.7 (11.6) | 42.3 (12.8) | 0.94 | 0.86 to 0.98 |

Note. Data are mean (standard deviation); CI = confidence interval; ICC = Intraclass correlation coefficient. All variables were statistically significant ($p < .01$).

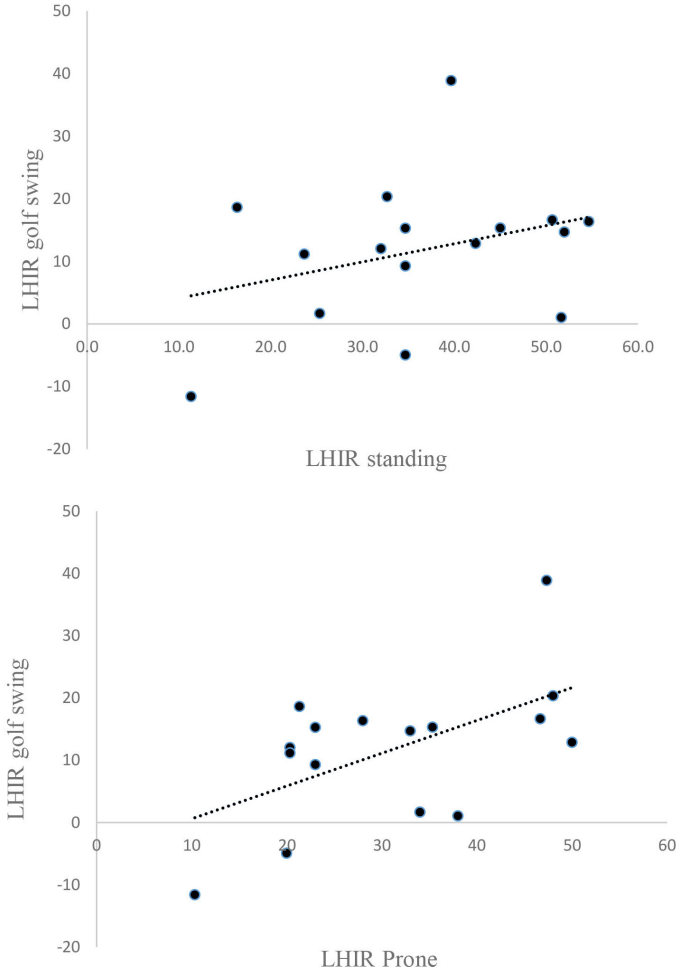


**Figure 1** — a) Scatterplot showing the correlation between LHIR measured in standing with LHIR measured during the golf swing and b) Scatterplot showing the correlation between LHIR measured in prone with LHIR measured during the golf swing.

measure should not be utilized in the golfing population as it didn't identify this discrepancy in mobility. This is logical given that hip rotation is measured in 90° of hip flexion with this measure and therefore maximal range may be limited by different soft or hard tissue structures than when the measure is taken with the hip starting in neutral (Gulgin et al., 2010). Both the prone and standing measure clearly demonstrate a significant difference in LHIR in the group of golfers with LBP (Table 3).

Interestingly, the LBP group demonstrated less hip internal rotation and greater hip external rotation in all positions. Having less hip internal rotation and greater hip external rotation in the LBP group compared to a more symmetrical hip IR/ER ratio in the NP group, points to differences in the femoral neck position. The anatomical make-up of the femur in relation to the hip may be more retroverted in golfers who experience LBP. This may have large implications for golf instructors and health care providers who may need to suggest a greater lead foot turn out at address in golfers with reduced LHIR. This could have implications on the risk of LBP from golf swings.

The cross sectional design of this study precludes any conclusion regarding the direction of the relationship between reduced LHIR and LBP. While speculative, it has been suggested that if a golfer uses less LHIR during the swing, the rotational load on the lumbar spine may be increased (Grimshaw & Burden, 2000; Vad et al., 2004), in turn enhancing the strain exerted onto the lumbar spine and therefore the risk of LBP (Adams & Hutton, 1981). These mechanisms have also been proposed in other athletic groups (Morton, Barton, Rice, & Morrissey, 2014; Sadeghisani et al., 2015; Vad, Gebeh, Dines, Altchek, & Norris, 2003). On the other hand, reduced mobility in LHIR may reflect a neuromuscular response to LBP (Sadeghisani et al., 2015). Further prospective research is required to investigate the relationship between LHIR, lumbar mechanics, loading and LBP, to determine whether it impacts on spine loading and is a risk factor for the development of LBP or a response to LBP itself. While the results do support the use of screening LHIR in a prone position in golfers, the implications of these findings remains largely unknown.

## Limitations

This study was cross sectional therefore, it is unknown if relationships were causal or associative. This study was limited to a relatively small population of male golfers. Therefore, future studies should include larger populations and examine the relationship between LHIR and lumbar spine kinematics and kinetics during the golf swing. Finally, a prospective study could be utilized to determine whether LHIR is modifiable in a clinical setting or during the golf swing.

## Conclusions

Golfers with a history of LBP have reduced LHIR when measured in prone and standing, as well as a trend for reduced LHIR during the golf swing. The prone passive measure seems to be the most valid and reliable for the assessment of LBP risk in golfers. These findings may have implications for the assessment and management of golfers with LBP.

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