

Research Article

Swing Kinematics, Pelvis and Trunk Sequencing, and Lower Extremity Strength in Golfers with and without a History of Low Back Pain

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Abstract

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Poor golf swing technique increases the risk of developing low back pain. This study measured golf swing kinematics; pelvis, lower trunk, and upper trunk sequencing patterns; and lower extremity muscle strength for 12 male golfers with previous low back pain (handicap = 8.0 ± 6.8) and 12 male asymptomatic golfers (handicap = 11.2 ± 7.1). 3D motion capture recorded trunk extension, trunk lateral tilt, pelvic tilt, crunch factor, and trunk segment sequencing during the golf swing. Knee flexion and extension and hip extension and abduction strength were measured with isometric dynamometry. Between groups comparisons found no significant differences for trunk extension, trunk lateral tilt, pelvic tilt, crunch factor, or muscular strength. However, regression analyses revealed that trail hip extension and abduction strength predicted peak pelvic angular velocity during the golf swing, but only for golfers with previous low back pain (p < 0.001). Additionally, the lower trunk supports the transfer of velocity from the pelvis to the upper trunk rather than increasing velocity through a proximal-to-distal sequence. Amateur golfers with a history of low back pain may rely more on hip muscle strength to generate angular velocity in the golf swing.

ABBREVIATIONS

PDS: Proximal-to-Distal Sequencing; LBP: Low Back Pain; NLBP: No Low Back Pain; ADD: Address of the golf swing; IMP: Ball Impact; END: End of the golf swing; LT: Lower Trunk; UT: Upper Trunk

INTRODUCTION

For millions of individuals, golf is an accessible form of physical activity. As moderate intensity aerobic exercise, playing golf can improve an individual's balance [1], coordination [2], muscle strength [2], and visual-spatial awareness [3]. These benefits are largely from the physical demands of the golf swing. A repeatable and efficient golf swing is optimized through a proximal-to-distal sequencing (PDS) pattern [4] and characterized by a sequential increase in linear and angular velocity through linked body segments beginning proximally (i.e., pelvis) and ending distally (i.e., golf clubhead) [5]. PDS involves the transfer of energy through body segments creating a "kinetic chain". Because force development begins with the lower extremities, assessing

lower body strength can provide further insight into golf swing technique and sequencing [6]. In the golf swing, the paraspinal and lower extremity muscles stabilize the pelvis and are vital to increasing clubhead velocity with a PDS [6,7]. PDS is observed in multiple other activities (e.g., baseball throwing), and although currently unexplored for the golf swing, moving outside of a PDS pattern may increase joint torques thereby elevating the risk of injury [8,9]. Adequate hip and knee strength is essential for golf performance but can also support the health and safety of lower extremity joints during the swing, especially if there are increased joint torques due to poor swing technique [10].

For golfers, overuse injuries are more common than acute injuries [11]. Poor swing technique influences the loading of the musculoskeletal system, contributing to the development of low back joint and muscle pain [12-14], and injuries to the lumbar spine account for 26% to 55% of all golf related injuries [11]. Many golf swing instructors work with golfers experiencing low back pain (LBP) and try to lessen the intensity of their pain. Because LBP is a common ailment for golfers, golf-specific

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strength and conditioning professionals have provided clinical recommendations for alleviating some LBP symptoms by improving golf swing technique, and increasing golfers' mobility and strength [15,16], and those recommendations have been successfully used in rehabilitation settings [17]. Golf swing technique characteristics that may incite LBP include excessive trunk extension and lateral bending in the backswing, near impact, and at the end of the golf swing [7]. Both trunk extension and lateral bending increase compressive and shear loads on the lumbar vertebrae and alter spinal force distribution throughout the golf swing [18]. Additionally, the product of pelvic angular velocity and trunk lateral bending at impact, called the 'crunch factor', may be related to LBP development in golfers; although, there is conflicting evidence regarding the influence of the crunch factor on LBP [19,20].

Pelvic orientation also affects force generation and energy absorption, which are especially important considering the high angular velocities of the pelvis, trunk, and upper extremities that occur throughout the golf swing. Optimally, the pelvis would have an anterior tilt at address, posterior tilt at impact, and anterior tilt again in the follow-through [21]. Pelvic motion is largely affected by hip range of motion and strength. At impact and during the follow-through, the lead hip is a pivot point around which the body rotates [22]. If a golfer lacks hip mobility, the spinal vertebrae may compensate for the lack of hip rotation. However, because the lumbar vertebrae are limited to approximately 1° of rotation per segment [12], facilitating the rotation of the golf swing through the vertebrae can cause or exacerbate LBP [22-24]. Irrespective of the established relationship between hip range of motion and LBP, the relationship between LBP and hip and knee muscle strength has not been thoroughly explored. Greater lower extremity muscle strength is linked with golf swing performance [25]; however, LBP and erector spinae fatiguability can lead to quadricep activation inhibition and reduced knee extension strength [26]. Investigating the relationship between LBP and hip and knee strength may reveal insights for rehabilitation and strength and conditioning professionals working with golfers with LBP.

There are contraindicated golf swing positions that are presumed to instigate LBP in golfers, but these swing characteristics have yet to be quantified. Therefore, the purpose of this study was to quantify differences in trunk extension, trunk lateral bending, pelvic tilt, crunch factor, and trunk segment sequencing during the golf swing, as well as lower extremity strength between amateur golfers with and without previous LBP. It was hypothesized that golfers with a history of LBP would display more lumbar extension, more lateral bending, greater crunch factor values, different pelvic tilt patterns, and improper pelvis and trunk sequencing during the swing when compared with healthy golfers. Additionally, it was hypothesized that golfers with previous LBP would have less hip and knee strength than healthy golfers.

MATERIALS AND METHODS

Participants

Twelve right-handed male golfers who previously experienced

LBP during golf play and 12 right-handed asymptomatic (NLBP) male golfers participated in this study. A power analysis (beta = 0.95 and alpha = 0.05) was performed using data from previous research to determine 12 participants as an appropriate sample size [7,27]. All participants had a self-reported handicap less than 21, more than 12 months of golf experience, and were free from any neuromuscular injury that would affect their golf swing for at least six months. Participants signed an informed consent document prior to participation, and then completed the Modified Oswestry Low Back Pain Disability Questionnaire [28]. This study was approved by the university institutional review board in accordance with the Declaration of Helsinki.

Procedures

Nine motion capture cameras (5 Vantage and 4 Vero, VICON Inc., Denver, CO, USA) recorded kinematic data at 250Hz. Following a lower body plug-in gait marker set, retroreflective markers were placed on the following anatomical landmarks: anterior super iliac spine, iliac crest, posterior super iliac spine, lateral and medial femoral condyle, lateral and medial malleoli, second metatarsal head, fifth metatarsal head, posterior calcaneus, suprasternal notch, xiphoid process, acromion processes, thoracic vertebrae ten, and right scapula. For marker redundancy, four-marker clusters were placed at the midpoint of the lateral thighs and shanks, and two-marker clusters identified the anterior superior iliac spines following the line of the iliac spine towards the single marker on the iliac crests. Triangular three-marker clusters were placed on to the cervical vertebrae seven (C7) and the lumbar vertebrae four (L4) with the most inferior marker in the cluster placed directly over the respective spinous process; the additional markers were tracking markers used if the primary C7 or L4 marker was missing during any of the trials. Five single markers were also placed on the shaft and head of the golf club.

Prior to golf swing trials, the participants warmed-up by hitting foam golf balls with a pitching wedge, seven-iron, fouriron, and driver for two minutes each. The participants then used their own driver to strike a foam golf ball into a net positioned three meters away. One-minute of rest was provided between each swing trial. After five successful swing trials, maximum muscle strength was tested.

For all muscle strength tests, participants completed three, three-second maximal effort, isometric trials each separated by one-minute of rest. Knee flexion and extension strength were measured with an isokinetic dynamometer (Cybex Dynamometer, Computer Sports Medicine Inc., Stoughton, MA, USA) following manufacturer-outlined procedures. Hip abduction and extension strength were measured with a MicroFET handheld dynamometer (Hogan Industries, Draper, UT, USA), and testing was performed in the side lying and prone positions, respectively. For all strength tests, the participants were familiarized with the protocol before testing. Strength measures were normalized to body weight.

Data Analysis

Kinematic data were processed in Visual 3D (Version 6,

C-Motion, Germantown, MD, USA) and filtered with a fourthorder Butterworth low-pass filter at 8 Hz. When analyzing trunk motion, lower trunk (LT) and upper trunk (UT) segments were created to differentiate between lumbar and thoracic vertebrae movement. LT and UT segment coordinate systems were defined using the Cardan rotation sequence conventions described in a previously validated multi-segment trunk model of the LT and UT [29]. In contrast to the previous model, the present study placed marker clusters at L4 rather than at L1 in order to define a larger portion of the lumbar spine in the analysis of LT motion. Pilot testing for this study verified the accuracy of these model adaptations. For the analysis of trunk and pelvis motion in this study, the golf swing was simply defined by the address position (ADD), ball impact (IMP), and the end of the swing (END). ADD was the frame before the clubhead began to move posteriorly. IMP was the frame when the golf clubhead was the closest to its initial frontal plane location at ADD as determined by reflective markers placed on the clubhead. Finally, END was considered the instance when the golf club reversed direction in the sagittal plane. All golfers in this study were right-handed with their left leg as the lead leg (i.e., leg closest to the target) and their right leg as the trail leg (i.e., furthest from the target).

Trunk extension was calculated as the deviation of the LT and UT segments in the sagittal plane relative to the position at ADD. Pelvic tilt was analyzed as the change in the angle of the pelvis segment in the sagittal plane between ADD and IMP or between IMP and END. Trunk lateral bending was defined as the deviation of the LT relative to the pelvis in the frontal plane. The pelvis crunch factor was calculated as the product of pelvis angular velocity and trunk lateral bending at IMP, while the LT crunch factor used LT angular velocity in the same calculation [20]. All reported segment positions are the difference between the instantaneous position and the position of the segment at ADD. The time between peak segment angular velocity and IMP was calculated for the pelvis, LT, and UT, and used to identify the trunk sequencing patterns.

Statistical Analysis

Statistical analyses were completed using SPSS (Version 27, SPSS Inc., Chicago, IL, USA). Two-tailed independent *t*-tests with the Bonferroni correction (α -level = 0.00833) applied were used for demographic comparisons and evaluation of pelvis, LT, and UT peak angular velocities. To control for Type-I error, three multivariate analyses of variance (MANOVA) were conducted to investigate group differences for swing kinematics (i.e., LT extension at END, pelvic tilt angle at IMP and END, LT lateral bending at IMP and END, and pelvis and LT crunch factor at IMP), pelvis, LT, and UT sequencing patterns, and lower extremity strength. Homogeneity of variance for the data was assessed with Levene's test and was satisfactory (p > 0.05).

Linear regressions with backwards model selection (predictor exclusion criteria of $p \ge 0.100$) were conducted to identify relationships between lower extremity strength and peak pelvis, LT, and UT angular segment velocity for both groups. For all analyses, the α -level was set to 0.05. Calculations of effect

size used Cohen's *d* for bivariate analyses (small = 0.2, moderate = 0.6, large = 0.8) and partial η^2 for multivariate analyses (small = 0.01, moderate = 0.06, large = 0.14). Results are reported as means ± standard deviations.

RESULTS

Findings from independent t-tests did not reveal significant group differences for age (LBP = 35.1 ± 13.8 yrs, NLBP = 34.4 ± 15.7 yrs; p = 0.913), handicap (LBP = 8.0 ± 6.8 , NLBP = 11.2 ± 7.1 ; p = 0.267), BMI (LBP = 28.5 ± 5.2 kg/m², NLBP = 27.4 ± 4.2 kg/m²; p = 0.574), golf experience (LBP = 16.8 ± 12.5 yrs, NLBP = 19.1 ± 11.3 yrs; p = 0.636), or peak clubhead velocity (LBP = 38.9 ± 2.42 m/s, NLBP = 38.6 ± 2.67 m/s; p = 0.785). The LBP group scored $10.5\% \pm 8.9\%$ on the modified Oswestry LBP Disability Questionnaire indicating a minimal level of disability.

Group differences assessed with combined dependent variables through the MANOVA analyses were not significant for swing kinematics (p = 0.693, partial $\eta^2 = 0.23$), sequencing patterns (p = 0.643, partial $\eta^2 = 0.20$), or lower extremity strength (p = 0.828, partial $\eta^2 = 0.23$). Mean swing kinematic and lower extremity muscle strength values are provided for reference in Table 1, and pelvis, LT, and UT sequencing patterns are presented in Table 2.

Conversely, independent *t*-tests revealed that peak angular velocity magnitude was significantly different between pelvis, LT, and UT segments (Table 2). Regardless of group, peak angular velocity of the pelvis (95.2ms \pm 21.4ms before IMP) and the LT (90.2ms \pm 19.1ms before IMP) occurred significantly earlier than the peak angular velocity of the UT segment (80.3ms \pm 12.7ms before IMP) (p < 0.001, d = 0.889, and p = 0.003, d = 0.669, respectively), but no significant differences were noted between the timing of the peak angular velocity for the pelvis and LT segments (p = 0.182; d = 0.281).

 Table 1: Descriptive values of swing kinematics and lower extremity muscle strength (Mean ± SD).

		LBP	NLBP			
Crunch Factor at IMP	Pelvis	2495.2 ± 2673.4	1571.6 ± 1658.7			
(deg ² /s)	LT	1798.5 ± 1716.0	1239.9 ± 1322.4			
LT lateral bending (deg)	IMP	10.3 ± 8.4	6.1 ± 4.4			
	END	5.8 ± 8.0	2.4 ± 6.7			
LT extension (deg)	END	10.9 ± 4.8	10.3 ± 4.7			
Pelvic Tilt Angle (deg)*	IMP	-14.7 ± 7.9	-11.7 ± 4.9			
	END	-16.2 ± 14.6	-18.2 ± 8.9			
Knee Extension Strength (% body weight)	Trail	285 ± 50	268 ± 95			
	Lead	275 ± 76	261 ± 87			
Knee Flexion Strength	Trail	181 ± 32	188 ± 56			
(% body weight)	Lead	164 ± 34	172 ± 56			
Hip Extension Strength (% body weight)	Trail	78.8 ± 24.2	72.7 ± 27.3			
	Lead	78.8 ± 30.3	69.7 ± 24.2			
Hip Abduction Strength (% body weight)	Trail	85.6 ± 18.5	81.0 ± 25.5			
	Lead	83.3 ± 23.1	81.0 ± 23.1			
*Pelvic tilt angle represents the difference in sagittal plane pelvic tilt between ADE and IMP or IMP and END. Negative values represent posterior tilt.						

Abbreviations: LBP: Low Back Pain; NLBP: No Low Back Pain; IMP: Ball Impact; END: End of the golf swing; LT: Lower Trunk.

Table 2: Peak angular velocity for the pelvis and trunk segments.

	Segment Ar	Segment Angular Velocity (deg*s ⁻¹)					
	Pelvis	LT	UT	<i>p</i> -Value ["]		Size*	
LBP 422.8 ± 100.8		342.1 ± 94.0	479.0 ± 83.7	Pelvis vs LT	< 0.001	0.829	
	422.8 ± 100.8			Pelvis vs UT	< 0.001	0.606	
			LT vs UT	< 0.001	1.539		
NLBP 425.1 ± 80.2	337.5 ± 65.3	465.8 ± 72.2	Pelvis vs LT	< 0.001	1.198		
			Pelvis vs UT	0.003	0.533		
			LT vs UT	< 0.001	1.864		
There were no significant group differences as determined by MANOVA analysis							
(p = 0.643).							
" α -level with Bonferroni correction = 0.00833							
* Cohen's d							

Abbreviations: LBP: Low Back Pain; NLBP: No Low Back Pain; LT: Lower Trunk; UT: Upper Trunk.

Although there were no group differences in normalized muscle strength, the lower extremity muscles that significantly predicted peak angular velocity of the pelvis, LT, and UT were different between the LBP and NLBP groups (Table 3). For the LBP group, trail hip extension strength was a positive predictor of peak angular velocity for the pelvis (β (SE)=15.93(2.70); p = 0.01) and LT (β (SE)=13.97(3.06); p = 0.004), while trail hip abduction strength negatively predicted pelvis peak angular velocity (β (SE)=-11.73(1.81); p = 0.007). For the NLBP group, peak angular velocity was not significant associated with either measure of hip strength.

DISCUSSION

The present study compared swing kinematics, pelvis and trunk segment sequencing, and lower extremity strength between male golfers with a history of LBP and asymptomatic controls using a multi-segment trunk model. Primarily, this research demonstrated that golfers with a history of LBP have similar swing kinematics, pelvis and trunk sequencing patterns, and lower extremity strength to healthy golfers; thus, the hypotheses related to golf swing kinematics and muscle strength were not supported. However, additional analyses identified that pelvis, LT, and UT peak angular velocity is predicted by the strength of different lower extremity muscle groups depending on the presence or absence of LBP.

Proximal-to-Distal Sequencing

An effective golf swing follows PDS where successive body segments increase velocity until IMP with the golf ball [4]. If trunk segment motion followed a PDS pattern, peak angular velocity would occur first in the pelvis, followed by the LT, and finally the UT. However, for all golfers in this study the pelvis and LT effectively reached peak angular velocity at the same time followed by the UT. This pattern was observed even when segment timing data was divided by group. The golfers in the LBP group seemed to move their pelvis and LT in unison as peak angular velocity for the segments occurred 93.3 \pm 20.0ms and 93.8 \pm 21.2ms before IMP, respectively, and the UT reaching peak angular velocity 80.0 \pm 10.3ms before IMP. Conversely, the NLBP golfers seemed to use a more typical PDS pattern with the peak angular velocity of the pelvis, LT, and UT occurring 97.0 \pm 23.3ms,

 86.7 ± 17.0 ms, and 80.6 ± 15.2 ms before IMP, respectively (Figure 1). The differences in these observed trends were not supported statistically, which may be a result of the variability of the golf swing from the participants' wide range of skill levels or from the different degrees of LBP recovery for the golfers with a history of LBP. Similarly, compensating adaptations to trunk movement patterns have been noted in individuals with back pain [30]. For golfers with LBP, the erector spinae activates significantly earlier during the swing than for healthy golfers [27, 31], and the erector spinae activity magnitude may depend on joint stability and loading [32]. Future research should continue to investigate multi-segment trunk sequencing patterns to identify if similar

Table 3. Regression coefficients for peak angular segment velocity (β (Std. Error) after backwards model selection for comparisons of lower extremity strength with pelvis, LT, and UT segment velocities.

	LBP			NLBP			
Variable	Pelvis	LT	UT	Pelvis	LT	UT	
Adjusted R-Square	0.982	0.915	0.717	0.669	0.411	0.606	
Intercept	5.82 (0.51)***	1.45 (0.61)	4.93 (0.63)***	4.85 (0.89)***	3.70 (0.97)**	5.15 (0.88)***	
Trail Knee Extension	-1.42 (0.43)*			-5.71 (1.29)**	-3.60 (1.40)*	-4.12 (1.27)*	
Lead Knee Extension	1.51 (0.188)**	0.640 (0.27)	1.13 (0.22)***	9.22 (2.26)**	6.14 (2.46)*	7.12 (2.23)**	
Trail Knee Flexion	4.85 (0.81)**	2.89 (1.29)		4.87 (1.24)**	3.97 (1.35)*	4.64 (1.23)*	
Lead Knee Flexion	-5.71 (0.80)**	-3.97 (1.49)*		-8.92 (2.56)**	-6.77 (2.78)*	-7.73 (2.52)*	
Trail Hip Extension	15.93 (2.70)**	13.97 (3.06)**					
Lead Hip Extension	6.22 (2.25)						
Trail Hip Abduction	-11.73 (1.81)**						
Lead Hip Abduction							

* Correlation is significant at p < 0.05

** Correlation is significant at p < 0.01

*** Correlation is significant at p < 0.001

Abbreviations: LBP: Low Back Pain; NLBP: No Low Back Pain; LT: Lower Trunk; UT: Upper Trunk.

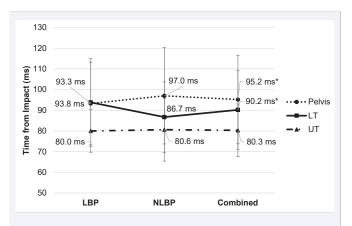


Figure 1 Sequencing patterns of the pelvis, LT, and UT for the LBP and NLBP groups and the entire sample combined. Standard deviation bars are shown for group means.

*Indicates a significant difference from the UT.

trunk movement patterns occur for golfers who are currently experiencing LBP and if these golfers are subconsciously or protectively altering their swings.

Regardless of sequencing pattern, peak angular segment velocity did increase from the pelvis to UT but was lowest in the LT for both groups. Past researchers have noted similar angular velocity behavior when comparing the pelvis and UT [33] as well as the LT and UT [34,35]. However, investigating the pelvis, LT, and UT segments together highlights the unique nature of the LT in the PDS, and the role of the lumbar spine in the transfer of velocity to more distal segments in the golf swing [36]. The LT segment may provide stability rather than mobility in the golf swing [35]. To maintain competitive clubhead speeds, golfers with LBP history may use methods other than PDS to develop velocity during the swing, while golfers without pain use proper swing technique to generate angular velocity.

As a possible explanation for this difference, peak segment angular velocity was best predicted by the strength of different muscle groups for the LBP and NLBP groups (Table 3). For golfers with past LBP, peak angular velocity of the pelvis was predicted by trail hip extension and abduction strength and bilateral knee extension and flexion strength. None of the measured hip strength values were related to angular velocity in the NLBP group. Gluteal muscle activation affects pelvic stability during the golf swing, which may also impact the development of LBP [18,37]. Bilateral hip extension strength measures were positively related to pelvic angular acceleration, although this association was only statistically significant for the trail hip. Hip extension strength may be related to the stability of the pelvis as the golfer prepares to perform a powerful downswing. During the backswing, the muscles supporting the articulation of the trail hip with the pelvis are isometrically activated as the gluteal muscles resist pelvic rotation allowing a greater stretch of the trunk musculature [37]. Interestingly, trail hip abduction strength was a negative predictor of pelvic angular velocity. Hip abduction strength may be a passive contributor to the golf swing, as the gluteus medius, a major hip abductor, acts eccentrically during close-chained rotational movements and provides hip stability by producing large forces over short distances [37]. During the force and velocity generating portion of the golf swing prior to IMP, the trail hip is internally rotated for right-handed golfers. Internal rotation of the hip has been shown to reduce gluteus medius activation during a squat [38], so golfers may not rely on the hip abductors to generate force because of the suboptimal, elongated muscle position. For golfers with previous LBP, the ability to predict pelvic angular velocity from hip strength may indicate that pelvic stability is more central to velocity development when LBP is present.

Consistent for both groups, the bilateral knee extension and flexion predictive values had opposite relationships with peak angular velocity, which could be representative of the force couple created by the lower extremity to rotate the body during the golf swing. Rotation in the backswing is partially facilitated by lead knee extension and trail knee flexion, which predominantly occur isometrically, to create a clockwise rotation (for a righthanded golfer). Conversely, to begin counterclockwise rotation in the downswing, the lead knee actively moves into flexion and trail knee extends. Previous research has identified that the total work performed by both legs can predict clubhead velocity, though lead leg work is a stronger predictor of clubhead velocity [39]. For both groups, lead knee extension strength was a significant predictor of pelvis and UT angular velocity; however, this relationship was stronger among the NLBP group. It is interesting to note the difference in the magnitudes of the β -coefficients for knee flexion and extension strength for each group. Pelvis, LT, and UT angular velocities were much more strongly predicted by hip muscle strength for golfers with previous LBP and by knee muscle strength for healthy golfers. The trend that identified a lack of separation between the pelvis and LT for golfers with past LBP (Figure 1) may be a result of the greater need for hip muscle strength to generate angular motion. However, this was simply an observed trend that was not supported statistically. Future research should pursue a more thorough investigation of the contribution of hip musculature to the golf swing specifically for golfers with LBP.

The equal clubhead velocities between groups suggests that golfers with LBP may rely on different muscle activation patterns and magnitudes to perform successful golf swings. In general, there is ample evidence suggesting that muscular strength has beneficial properties for avoiding musculoskeletal injury and improving golf performance [25], and golfers should seek to increase their strength and flexibility to improve performance and bolster their longevity in the game. A preliminary recommendation for golfers with LBP is to strengthen the gluteal muscle structures as this may improve their ability to stabilize the pelvis and LT during the golf swing.

Swing Kinematics

Radiological investigations have suggested that combined lateral bending and axial rotation leads to deteriorated L4-L5 vertebrae on golfers' trail side [40]. Golfers with LBP may have earlier lateral bending in the golf swing which can expose the lumbar vertebrae to shear stresses and more microtrauma accumulation over time [41]. The crunch factor has been used to analyze and predict golf performance variables [35,42]; however, its relevance is disagreed upon in assessments of golfers with LBP [13,19]. In the present study, there were no differences between the crunch factor values of golfers with past LBP and asymptomatic golfers. Furthermore, the results indicated that golfers with LBP history have similar trunk positioning and pelvis motion through the golf swing when compared with asymptomatic golfers.

Similarly, trunk extension angles were not different between groups. The trunk extension angles in this study were similar to previously reported measures collected with a lumbar motion monitor [7], suggesting that LT extension angles at END may not significantly contribute to LBP in golfers. Pelvic tilt angles were also not significantly different between groups. In a previous study, individuals with LBP had reduced pelvis rotation range of motion when performing pain inducing tasks [43], but people who regularly participate in rotational activities had greater

ability to move the pelvis [41]. Because the golfers in this study reported playing golf for an average of 16 years, they may have a greater capacity for pelvic motion regardless of pain.

As with all research, this study has limitations. This study included golfers with variable skill levels (sample handicap (mean \pm SD) = 9.59 \pm 7.02) which increased the overall representation of the golfing community, but also reduced the precision of the data. Additionally, the golfers in the LBP group had previously experienced LBP while golfing, but were free from pain during testing. The length of time since the last occurrence of LBP for the golfers was not controlled in this study and may have also increased the variability in the study. As such, these data should be interpreted conservatively. The golfers with LBP in this study were classified as having a minimal level of disability, which may have limited the impact of LBP on their swing kinematics. The link between lower extremity strength and golf swing kinematics and sequencing was indirect without a measure of joint kinetics. Future research should use force plates to solidify the connection between lower extremity strength and golf swing joint kinetics. Additionally, this research was performed within a laboratory space which could have influenced the ecological validity of the golf swing data.

CONCLUSION

Swing kinematics, trunk and pelvis sequencing patterns, and lower extremity strength were compared between LBP and NLBP golfers. There were no statistically significant differences in swing kinematics between LBP and NLBP groups; however, different relationships for peak pelvis and trunk angular velocity and muscle strength emerged between the LBP and NLBP groups. Additional trends for different PDS patterns of the pelvis and trunk segments were identified between groups. Pelvis, LT, and UT segment angular velocity magnitudes did not follow a PDS pattern for either group because the LT had the lowest angular velocity, suggesting that stability, rather than mobility, may be a primary function of the LT during the golf swing. Future research should investigate the role of the LT in the transfer of energy during the swing. Coupled with kinetic investigations, golfers and golf coaches would benefit from understanding the role of hip musculature in force development during the golf swing, especially for golfers with LBP or limited hip mobility.

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