



Low back pain and golf: A review of biomechanical risk factors

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ABSTRACT

Golf is an international sport played by a variety of age groups and fitness levels, and although golf has a low to moderate aerobic intensity level, injuries are common among professional and amateur golfers. High amounts of force experienced during the golf swing can lead to injury when golfers lack appropriate strength or technique with the lower back most commonly injured. Research has indicated that trunk muscle activation, hip strength and mobility, and pelvis and trunk rotation are associated with low back pain (LBP). Based on anecdotal evidence, golf practitioners specifically address issues in weight shift, lumbar positioning, and pelvis sequencing for golfers with LBP. This review aims to elucidate the effects of proper and improper golf swing technique on LBP and to help golf practitioners understand how to approach the alleviation of LBP in their clientele.

Introduction

A worldwide game, over 55 million people play golf each year.¹ These golfers are young and old with varying degrees of skill, but regardless of skill level, playing golf provides cardiovascular benefits, social connections, and mental stimulation.^{2,3} Golf's musculoskeletal benefits include: improved balance^{4,5} and increased bone growth when walking while playing.⁶ Because the swing is a multi-joint movement that dynamically loads the body, coordination and muscular strength are also taxed during golf play.^{7,8} Individuals with disabilities gain additional benefits from playing golf.

Nearly one-third of stroke survivors experience depression partially due to a decreased capacity for participating in familiar or novel activities, while active participation is linked with improved emotional well-being.⁹ In post-stroke individuals, motor abilities are often diminished; however, the introduction of a coordinative golf training intervention resulted in increased visual-spatial awareness, balance, and emotional well-being in post-stroke individuals.¹⁰ Similar benefits were also reported for individuals with "severe and enduring mental health problems."¹¹ Internationally, golf initiatives have begun to increase golf's accessibility for physically handicapped people. The possibility of playing golf could provide these individuals with opportunities to receive the aforementioned benefits.⁹ Regardless of modifications, golf is considered a low risk and low impact sport allowing participation without a pressing fear of acute injury. While acute injuries do seldom occur, overuse injuries are most common amongst golfers.¹² Regardless of the type of injury, golf participation decreases, and the previously experienced

benefits of playing are lost.

Though golf injuries frequently result from overuse, injury prevalence is not determined by skill level. The skill differences between professional and amateur golfers often reflects the amount of time devoted to practicing technical golf skills and undergoing physical training. Professional golfers tend to experience overuse injuries because of increased frequency of practice with reduced golf swing variance.^{13–15} For amateur golfers, injuries are often multifactorial; their overuse injuries frequently result from improper technique coupled with musculoskeletal imbalances.^{15,16} The lower back, wrist, and elbow are commonly effected by overuse injuries, and for both male professional and amateur golfers, the lower back is the most frequently injured area.^{3,12,17–19}

Injuries to the lower back, often denoted by low back pain (LBP), have a wide etiology. The golf swing represents one element that contributes to lower back injuries and muscular imbalances because of its asymmetric nature.²⁰ The risk of developing LBP increases when there are technique flaws present in the swing that cause the improper loading of spinal structures.^{21–23} With adequate physical preparation, the body can adapt to and manage the stresses of the golf swing. However, injury risk further increases if a golfer lacks adequate mobility. Because improper golf swing technique compounds musculoskeletal stresses experienced during the swing, technique flaws are a leading contributor to LBP in amateur golfers.²⁴

Qualitatively, golf teaching professionals recognize several specific technique errors that place golfers at higher risk for developing LBP. Golf fitness professionals have additionally identified mobility characteristics that contribute to LBP, while researchers have investigated the

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interaction between muscular activation, kinematic sequencing, and LBP. The present review aims to identify swing technique characteristics, physical inadequacies, and biomechanically analyzed variables that have been implicated in LBP development. Because acute injuries are less common amongst golfers, this review focuses on chronic LBP. This review also provides support for instructional techniques used by golf teaching and fitness professionals aiming to reduce LBP in their clientele by outlining the interaction occurring between the golf swing movement and musculoskeletal structures. The conclusions this review create ideas for researchers to pursue as avenues for investigations that may help reduce the frequency of LBP in golfers. A previous review has outlined the golf swing when divided into phases and the influence LBP can have on swing kinematics and muscle activation,¹⁷ while another review focused on difference between golf swing types (i.e. “classic” vs. “modern”) and the different impacts each swing type has on LBP.¹³ This review builds upon these reviews and emphasizing the specific golf swing and physical characteristics that are associated with LBP along with proposing additional swing flaws that golf teaching professionals believe may be related to LBP development. These risk factors are further explained relative to lumbar and pelvis anatomy and the principles of a proper golf swing. Several databases (PubMed, SPORTDiscus, Academic Search Premier, and Medline) were used in the literature search. The search was conducted in August 2018 with no date restriction. The references of obtained articles were scanned for additional research that would strengthen this review.

Anatomy of the lumbar spine

Spinal vertebrae consist of vertebral bodies and spinous, transverse, and superior articular processes. The vertebral body has cartilaginous plates on both ends which deform under compressive load. Between each vertebra, intervertebral discs also aid with compressive force absorption. During compression, the nucleus pulposus encapsulated within the intervertebral disc presses into the vertebral body causing the cartilaginous end plates to bulge inward, compressing the calcaneus bone of the vertebral body.²⁵ The processes of the vertebrae provide attachment sites for trunk flexor and extensor muscles, and inferior articular facets allow vertebral articulation. Damage to the articulating surfaces between vertebrae can lead to spondylolisthesis which often results in LBP.^{25,26} In addition to the nucleus pulposus, intervertebral discs contain annulus fibroses and end plates. A herniated disc typically refers to an issue with the annulus fibrosus, but may also be the result of damaged end plates and leaking nuclear fluid from the nucleus pulposus.²⁵ Damage to spinal structures results from either repeated stress to the tissues or a one-time stress with a magnitude large enough to cause structural failure. These forces result from muscle activity and from the body's movements.

Muscular forces either resist or cause movements. A muscle's physiological cross-sectional area determines the amount of force that can be produced, while the attachment and insertion points determine the line of action of the muscle's force.²⁶ Both anterior and posterior trunk muscles affect lumbar spine movement and stability. Anterior trunk muscles cause trunk flexion, rotation, lateral bending, while providing spinal stability. Trunk flexion refers to the torso moving anteriorly and inferiorly toward a stationary lower body, while hip flexion refers to the anterior and superior movement of the lower extremities toward a stationary torso. Trunk flexion results mainly from the activation of the rectus abdominis and the three muscles of the abdominal wall [external oblique (EO), internal oblique (IO), and transverse abdominis (TA)]. The abdominal wall muscles each have an increased flexor moment capacity because of their attachment onto the linea semilunaris (positioned on the lateral border of the rectus abdominis) which affects the line of action and moment arm of each muscle. The EO, IO, and TA also cause trunk rotation and lateral bending.²⁵ The psoas muscle group attaches to the lumbar vertebrae and inserts on the lesser trochanter of the femur; however, EMG testing suggests that the psoas activates only during hip flexion. The psoas muscles also provide lumbar stability when there is a

hip flexor torque.²⁵ The EO, IO, and TA provide spinal stability, specifically under axial compressive forces, by forming a hoop structure around the abdomen which increases stiffness under load.

Posteriorly, the erector spinae (ES), multifidus, and latissimus dorsi each contribute to trunk extension. The iliocostalis lumborum and longissimus thoracis, two muscles in the ES muscle group, cause extensor moments for the trunk and stabilize against anterior shear forces in the lumbar spine. The multifidus also produces an extensor moment, but its short length limits it to providing small corrections and support at specific spinal levels stressed during motion. The multifidus also creates small rotational moments in the transverse plane.²⁵ When the trunk is loaded while in flexion, the posterior shear torque produced by the ES muscles stabilizes the vertebrae. Additional stabilization comes from the quadratus lumborum during flexion, extension, and lateral bending tasks.

Many lower extremity muscles originate from the pelvis or the lumbar spine (e.g. psoas major and minor) and affect spinal stability and movement. The gluteus maximus (GMx) and gluteus medius (GMe) originate along the ilium and attach onto the femur. The GMx extends the hip, while the GMe abducts, externally rotates, and provides stability at the hip. The biceps femoris, semitendinosus, and semimembranosus originate from the ischial tuberosity and insert onto the tibia and fibula, and these muscles are responsible for knee flexion and hip extension. The rectus femoris, vastus lateralis, vastus intermedius, and vastus medialis make up the quadriceps muscle group and extend the knee. The rectus femoris, which originates from the anterior inferior iliac spine, also flexes the hip. Lower extremity muscles that contribute to hip movement can exert force onto the pelvis because of their insertion points. The orientation of the lumbar spine, pelvis, and lower extremities affects the efficiency of stabilizing muscles. A muscle's ability to produce force changes as a factor of its length with the ends of a joint's range of motion (ROM) having lower force potentials. When movement patterns sub-optimally position muscles for force production, loads about the spine can increase, and lower back disorders develop.

Lower back disorders

Lower back disorders commonly occur in the general population. Analysis of data from a study with nearly 15,000 participants found that 19.5% of women and 13.9% of men reported developing chronic LBP during a 9-year span. Furthermore, increased work physical activity intensity was correlated with higher rates of LBP for both men and women.²⁷ The presence of LBP often alters individuals' movement patterns, which may cause further musculoskeletal pain or injury to eventually develop. In fact, multiple studies have reported that LBP affects hip and lumbar spinal motion.^{28–32} LBP can additionally influence lumbar movement by reducing segmental velocity and acceleration during movement and by impairing trunk repositioning accuracy.²⁸ The interactions between muscles, ligaments, and vertebrae change based on the position of the body, the velocity of the movement, and the force required for the movement. These interactions determine the success of the movement and the stresses experienced by the body. Because of movement complexity, low back disorders have varying etiologies correlating with pain in different musculoskeletal areas.

In some individuals with chronic LBP, motor training to increase the activation of gluteal muscles during hip extension reduced the amount of pain experienced.²⁵ Lumbopelvic movement requirements depend on an individual's habitual activities and the activity's muscular requirements. Repeated movement patterns result in muscular adaptations that accommodate the movement demands. For example, external hip rotation ROM of the dominant limb was significantly increased in individuals who participated in rotational activities at least twice a week.³³ Limitations in either lumbar or pelvic regions caused the other region to compensate through abnormal motion,³¹ predisposing individuals to injury.

Low back disorders are also common in athletes. An extensive meta-analysis of back pain prevalence in Olympic-discipline sports postulated

that back pain may be more common in athletes compared to general populations. According to this meta-analysis, the development of back pain in athletes depends on sport-specific movement demands, spinal loading characteristics, sex, and age.³⁴ Some spinal stabilization can be provided through muscle activation during movement, but muscular imbalances may lead to injury through increased shear forces.³⁵ Specific to golf, the rotation in the golf swing loads the spine with torsional, compressive, and shear forces, jeopardizing spinal stability. LBP is common among athletes of various sports and especially common in golf.

Lower back pain in golfers

Regardless of skill level, golfers more frequently experience overuse injuries than acute injuries. Most golf-induced overuse injuries affect the back, shoulder, knee, or elbow, with the lower back being the most common.^{15,36–39} Because of differences in swing intensity and training frequency, injury mechanisms differ between professional and amateur golfers.

For professional golfers, overuse injuries are commonly a result of the volume of practice and play³ and because of decreased variability in the swing which increases the cyclic nature of musculoskeletal loading. Typically, an amateur golfer's swing contains more flaws than a professional's swing,⁴⁰ and these errors can contribute to injury development.^{13,19,21,23,41} Improper loading from poor swing technique can damage soft tissues causing degeneration of skeletal joints over time.^{42,43} According to the cumulative load theory, repeated loading with high force increases the overall stress experience by the system, which can damage musculoskeletal tissues over time. Injury due to repeated loading is why LBP is frequently reported as an overuse injury rather than an acute injury in golfers.¹⁶ It has been further suggested that the asymmetrical pattern of the golf swing may lead to deterioration of the right lumbar spine around L4-L5 in right-handed golfers.⁴³ Repeated lateral bending with simultaneous pelvic rotation may be a mechanism contributing to this deterioration.^{43,44}

The modern golf swing emphasizes force production through the increasing degrees of pelvic and shoulder separation. Modern golfers prioritize rotational velocity which results in larger compressive and anteroposterior loads after impact when compared to traditional golf

swing. The presence of large spinal loads requires muscle activity to support spinal structures; however, golfers with LBP may have a decreased capacity to anticipate musculoskeletal perturbations occurring during the swing resulting in stabilizing muscles activating after the stresses have subsided.¹⁷ Although, the level of force experienced during the swing can be altered by changing the backswing length.⁴⁵

Biomechanical analysis of the Golf swing

The golf swing is traditionally segmented into different phases, and four commonly agreed upon phases are address, backswing, downswing, and follow-through.^{17,46,47} Further separation of these phases helps identify additional important events during the golf swing. The following events will be considered along with the four phases mentioned above: top of the backswing, acceleration phase, impact, early follow-through, and late follow-through (Fig. 1).

Two variations of the golf swing have been previously described. The 'classic' golf swing is common amongst older golfers and was performed by most touring professional golfers before the 1980s. In the 'classic' golf swing, the pelvis freely rotates away from the target during the backswing often causing the left heel to raise off the ground.¹⁷ The golfer also finishes the swing in an erect "I" position in the follow-through.^{13,47} The lengthening of golf courses and the development of modern equipment has caused golfers to adapt their golf swings to hit the ball further, creating the 'modern' golf swing.^{17,47} The modern golf swing is characterized by restricted pelvic motion during the backswing, lateral bending at impact, and lumbar extension during the follow-through.

Proximal-to-distal sequencing

Regardless of the swing type, the most effective golf swings use proximal-to-distal sequencing (PDS). The goal of PDS is to increase the velocity of the most distal segment to a greater velocity than what would be possible using isolated muscle actions.⁴⁸ The concept of PDS stems from the summation of speed principle, stating that when a proximal segment reaches its maximum velocity and its lowest acceleration, the linked and more distal segment will begin its motion, eventually reaching a higher maximum velocity.⁴⁹

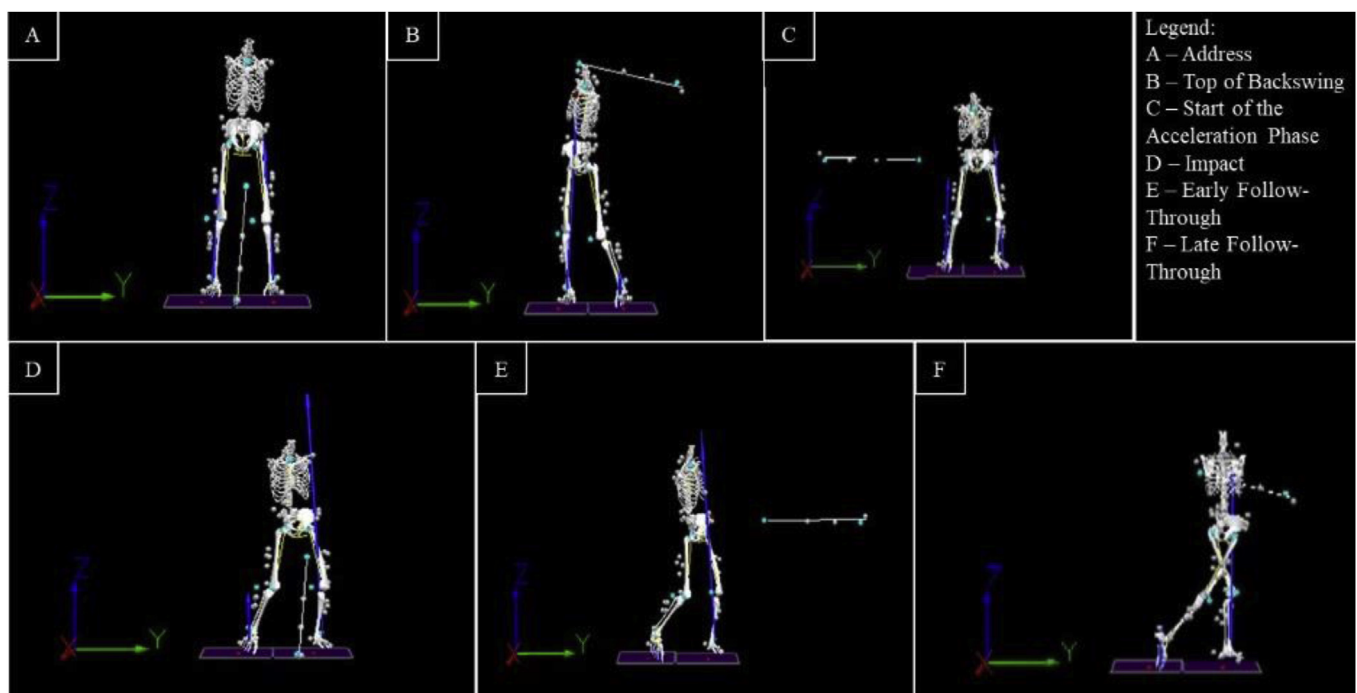


Fig 1. Phases of the golf swing. Unlabeled phases include the backswing which begins at A and ends at B and the downswing which begins at B and ends at D.

PDS in the modern golf swing follows a cascade of events beginning at the top of the backswing. The downswing motion is initiated with pelvic rotation toward the target. In response to pelvic rotation, the upper torso also begins to rotate causing the arms to move toward the golf ball. Once the pelvis reaches maximum velocity, the torso continues to accelerate. Maximal torso angular velocity and acceleration correspond with decreased pelvic angular velocity and acceleration when peak values are compared.⁵⁰ Similar kinematic and kinetic relationships occur between the torso and arms, and the arms and golf club: as proximal segments reach maximal velocity, energy is transferred into the distal segments and the motion is continued and velocity increased. Increases in segment angular velocity have been kinematically quantified to progress from 488 deg/s at the pelvis to 945 deg/s at the left hand. Using the timing of each peak angular velocity relative to impact, golfers used a PDS pattern with the exception of peak linear hand velocity.⁵¹ The early occurrence of peak linear hand velocity may result from the supination and wrist medial deviation that occurs as the golf club approaches impact so that the quality of ball contact is maximized.

Golf swing kinetics and muscle activity

Generating club head velocity requires efficient use of the body's musculoskeletal structure. The magnitude and timing of a golfer's ground reaction forces (GRF) impacts the amount of velocity generated. Low handicap golfers have a peak vertical GRF approximately 6% earlier in the swing than high handicap golfers.⁵² GRF has been combined with lower extremity joint kinematics to calculate the amount of work being done during the golf swing in relation to clubhead velocity. Total leg work during the golf swing explained 40% of the variance in clubhead speed that was not explained by golfers' age, meaning that total leg work is a significant predictor of club head velocity.⁵³ This finding insinuates that increasing force production will increase clubhead speed.

Compressive forces on the L4-L5 vertebrae are the result of trunk muscle activity and GRF during the golf swing.⁵⁴ These forces have been reported between 6.5 and 8+ times body weight immediately after impact.^{45,54,55} This is in part because of the coactivation of paraspinal muscles assisting the trunk to maintain its neutral position throughout the swing.⁵⁴ Paraspinal muscle activation may also increase anterior-posterior shear forces during the follow-through phases, affecting spinal stability.

The gluteal muscle activity resists pelvic rotation during the backswing allowing a greater stretch of the trunk musculature.⁵⁶ During the backswing, the GMe acts eccentrically, while the GMx stabilizes the pelvis.⁵⁶ Architecturally, the GMe is designed to produce large forces over a short distance, which is beneficial for hip stability. However, hip internal rotation may limit GMe activity.⁵⁷ The GMx and biceps femoris interact with the ES through the thoracolumbar fascia and ligamentum sacrotuberale, respectively. These muscles support the motion of the lumbar spine and pelvis and facilitate the transfer of energy into the torso and upper extremities,²⁹ which is essential for PDS in the golf swing.

Mechanisms of injury: low back pain

Acute musculoskeletal injuries occur when a force that exceeds a tissue's plasticity threshold is applied at one instance during movement. In chronic injuries, force levels may not surpass the plasticity threshold, but the repeated nature of the tissue loading can increase stress levels and result in injury. Ligament, tendon, muscle, and bone plasticity allow tissues to adapt to repeated loads; however, if either internal or external stresses (or a combination of both) surpass tissue limits, acute damage occurs resulting in permanent tissue deformation.⁵⁸ When tissues undergo moderate loads or strains, they adapt to the stress and recover stronger. Abnormal tissue stresses can result in structural aberrations after recovery that lead to chronic injury. Over time, tissue fatigue can also increase the risk of injury development.²⁵

In golfers, LBP is a common indicator of chronic injury. With

improper loading patterns, repetitive stress on the tendons and ligaments connecting the gluteal and ES muscles to the pelvis and vertebrae can lead to the development of LBP. Some golfers may be predisposed to developing LBP irrespective of golf swing technique. Interestingly, body mass index (BMI) has been negatively correlated with LBP development in young golfers,¹⁸ while body mass was positively related to LBP.^{59,60} Golfers with BMI > 25 experienced more instances of LBP from mechanisms outside of golf.³⁷ Body composition may impact the distribution of force through the body, and taller golfers or golfers with longer swing arcs need to have adequate musculature to support the lumbar spine.¹⁸ Increasing age has also been linked to LBP risk.⁶¹ LBP is a multifaceted injury that requires a holistic approach to treatment. For golfers, BMI and age should be considered with golf swing technique and physical characteristics when addressing LBP. The following injury mechanisms are a combination of factors recognized by the research community and by golf teaching and fitness professionals as contributors to LBP in golfers.

Established risk factors

A higher frequency of play has been correlated with an increase in overuse injury risk,⁶² and this risk is further inflated when a golfer exhibits poor technique.^{13,19} Forces capable of causing injury are generated during the golf swing, and if not properly sequenced, these forces dissipate into joints and soft tissue increasing the risk of injury.⁴² The following variables are recognized as detrimental to the golfer and are often tied to LBP either through research or through professional observation. These swing technique flaws place the musculoskeletal system in suboptimal positions that interfere with golf swing performance.

Spinal forces and muscle activation

A combination of GRF and paraspinal muscle activity affect the lumbar spine during the golf swing.^{19,54} Compressive and lateral shear forces were 26.3% and 75.5% larger in patients with LBP than in healthy individuals when performing identical bending and lifting tasks, which is possibly the result of increased coactivation of anterior and posterior trunk muscles in LBP patients.³⁰ Individuals with LBP may increase muscle activation in order to stabilize the spinal region by increasing vertebral compression and limiting sagittal plane movement. They may also alter trunk flexion velocity and positioning as coping mechanisms; however, previous research indicated that these changes had no effect on spinal loading during a lifting task,³⁰ though it is known that injured individuals change their movement patterns to avoid pain.⁶³ It has been suggested that golfers with LBP have less abdominal muscle activity during the golf swing, which possibly results in less trunk flexion during the downswing.³⁸ Interestingly, in healthy individuals increasing trunk flexion angle from 0° to 45° resulted in an increase of trunk rotation ROM of 19%, while pelvis rotation ROM decreased by 45% between the two trunk flexion positions.⁶³ In golfers with LBP, a combination of less abdominal muscle activity with less trunk flexion could lead to less total trunk ROM and more pelvic ROM and require more rotation to be facilitated by the lumbar vertebrae. Increased pelvic ROM could also predispose golfers to using technique with the contraindication positions addressed in the subsequent sections of this review.

When compared to asymptomatic golfers, golfers with LBP exhibit less ES muscle activity during the swing,⁵⁹ which may be a mechanism that reduces spinal loading. However, because the ES helps to stabilize the spine through its attachments on the pelvis, vertebrae, and ribs, a decrease in ES activity could be detrimental to golfers with LBP.⁶⁴ In fact, LBP golfers were reported to activate their ES before beginning the backswing, which was significantly earlier than non-LBP golfers.⁶⁵ The early activation of the ES indicates its role in stabilizing the lumbar spine and may be indicative of reduced multifidus activity in golfers with LBP.⁶⁵ In golfers with chronic LBP, ES endurance was related to knee extensor muscle inhibition, as individuals with a high ES fatiguing rate could not fully activate their knee extensors.⁶⁴ The causes of muscular

differences between LBP and healthy golfers is unknown, but addressing muscular strength and endurance deficits through training may help with LBP management.¹⁶

Because the pelvis provides origin and insertion points for the ES, GMx, GMe, rectus femoris, biceps femoris, semimembranosus, and semitendinosus, investigation of hip and knee muscle activation during the swing may provide insights into LBP etiology. For example, GMx endurance was less in patients with LBP compared to healthy controls during a back extension task.⁶⁶ Similar gluteal muscle patterns were reported during trunk flexion for patients with LBP. Lacking the stability provided by the GMx, individuals with LBP increased ES activation during flexion.²⁹ Because of ES activation causes trunk extension, increased activation would limit trunk flexion. Healthy professional golfers displayed more trunk flexion with a higher trunk flexion velocity during the downswing than those with LBP. The discrepancy between groups was hypothesized to be the result of greater abdominal muscle activation in healthy golfers during the downswing³⁸; however, investigations into this hypothesis have reached conflicting conclusions.

Horton, Lindsay, & Macintosh⁶⁷ reported no differences in rectus abdominis, EO, or IO muscle activity during the golf swing between healthy or LBP golfers. However, a further investigation reported a delay in lead EO activation during the backswing of LBP golfers compared to healthy golfers, although this delay did not reach statistical significance.⁶⁵ The differences in EO muscle activity may indicate that LBP golfers rotate their trunks with greater velocity, which could be a factor influencing the development of LBP. The EO may act as trunk stabilizer in golfers with LBP, although more research is needed to determine the role of the EO in the golf swing specifically for golfers with LBP.

Hip strength and mobility

The golf swing has asymmetrical muscular demands. During the backswing of right-handed golfers, the external rotators (e.g. GMx, GMe, psoas major and minor, and piriformis) of the lead hip are stretched eccentrically. Simultaneously, the opposite motions occur in the rear hip as clockwise pelvis rotation is restricted by the internal rotators (e.g. adductor longus, brevis, and magnus; and pectineus). Counter-clockwise pelvic rotation begins the downswing and is the result of gluteal and hip rotator muscle actions. Because these muscle actions are not equal in direction, force, or velocity, ROM deficiencies and strength imbalances can develop overtime. Limitations in hip ROM may be a product of capsular tightening.³⁹

At impact and during the follow-through, the lead hip acts a pivot point for the body to rotate around.³⁹ The motion of the hips, pelvis, and spine are highly interrelated through shared musculature and innervation.³¹ If a golfer lacks lead hip rotation ROM, pelvis and torso rotation during the backswing and follow-through may be facilitated by the spinal vertebrae, exacerbating LBP. However, there is a lack of consensus among researchers regarding the correlation between hip ROM and LBP.

In professional and amateur golfers with chronic LBP, passive and active lead hip internal rotation is limited when compared to non-lead leg internal rotation⁶⁰ and when compared to golfers without LBP.³⁹ Professional golfers with limited hip rotation often have asymmetrical hip rotation strength ratios, and these hip mobility characteristics place the lumbar vertebrae at higher risk for injury over time.⁶⁸ Possible hyper-tonicity of the lead hip external rotators developed through repeated eccentric loading may restrict internal rotation ROM and affect internal and external hip rotator strength.³⁹

Contradicting the above findings, Tsai et al.⁶⁹ reported no hip ROM differences between golfers with LBP and healthy controls. A similar lack of differences has been reported for hip rotation ROM in non-golfing populations with LBP. However, individuals with LBP displayed significantly decreased bilateral hip extension ROM and bilateral rotation asymmetries.³² Decreased hip extension ROM may result in an anterior pelvic tilt and a lordotic lumbar curve.³² While these hip ROM results conflict with previous investigations, the discrepancy may result from measurement error, specifically differences in measurement techniques

regarding pelvic stabilization during testing.³²

As most injury prevention research is correlational, case studies provide opportunities to formulate hypotheses about causal variables. Previous case studies have reported the alleviation of LBP through strengthening trunk and hip musculature, improving ROM, and altering swing technique.^{19,42,70} If golfers rotate past functional limitations during the swing, the spinal vertebrae and the tendons and ligaments connecting the musculature around the vertebrae are stressed leading to a higher risk of injury.³³ Further research is needed to elucidate the relationship between LBP and hip mobility.

Lateral bending and pelvic rotation

The combination of torso lateral bending and pelvic rotation, commonly referred to as ‘crunch factor,’ has previously been identified as possible contributors to LBP development in golfers.⁴⁴ Repeated performance of the golf swing is correlated with degradation of the L4-L5 vertebrae in golfers with LBP because of the ‘crunch factor.’⁴³ Lumbar vertebrae segments uniquely contribute to axial rotation and lateral bending motions. During trunk rotation, the L2-L4 vertebrae bend away from the direction of rotation, while L4-S1 bend toward the direction of rotation resulting in the desired trunk motion.⁷¹ The highest values for the combination of axial rotation and lateral bending are most frequently observed during the downswing, at impact, and in the early follow-through of the golf swing (Fig. 2).

Research findings conflict regarding the relationship between the ‘crunch factor’ and injury risk. Although the asymmetric and rotational nature of the golf swing places high loads on the spinal structures, researchers have reported a lack of repeatability for the ‘crunch factor’ as a risk factor for LBP. For instance, the ‘crunch factor’ did not differ between LBP and asymptomatic golfers when calculated using a lumbar motion monitor³⁸ or 3-D motion analysis.⁷² Originally, the ‘crunch factor’ was calculated with pelvic angular velocity and torso lateral bending analyzed as equal contributors; however, it has been suggested that replacing positional lateral bending data with instantaneous torso lateral bending velocity may better correspond with the risk for injury development.⁷³ Further research investigating the ‘crunch factor’ is needed to determine its relevance to LBP risk in golfers.



Fig. 2. Crunch factor. The combination of lateral bending with pelvic rotation, shown here at impact, stresses the right side of the lumbar vertebrae (for right-handed golfers).

Trunk over-rotation

Golfers with LBP move through dynamic ROMs in the golf swing, specifically trunk rotation ROM, that exceed passive ROMs limits.^{38,69} Excessive trunk rotation may indicate an effort to increase pelvis and torso separation for power generation; however, dynamically moving outside of active ROM could be detrimental to spinal health. Individuals with LBP, both golfers and non-golfers, tend to move with less trunk flexion.³⁸ The degree of trunk flexion is reported to affect trunk rotational ROM, with less trunk flexion correlating to less trunk and pelvis rotation,⁶³ which may increase forces on lumbar vertebrae. A shortened backswing that reduces trunk rotation was reported to decrease spinal loads compared to a full golf swing.⁴⁵ A separate test of the shortened golf swing reported no significant changes in club head speed or shot accuracy.⁴¹ The shortened golf swing may be a method for reducing over-rotation without severely sacrificing performance.

Potential risk factors

Golf teaching and fitness professionals consider several positions during the golf swing to be detrimental to the health of the lower back. It is understood that poor swing technique often leads to injury.² Although anecdotally accepted by golf instructors, there is limited biomechanical research associating the following swing flaws with LBP.

Lateral flexion without weight shift

In the backswing, golfers transfer around 40% of their weight onto their rear leg,⁴⁷ which is often accompanied by trunk lateral bending toward the same leg (Fig. 3). However, when a golfer's trunk laterally bends toward the lead side, a 'reverse spine angle' is adopted.⁷⁴ This position in the backswing places spinal vertebrae in a suboptimal position for starting the downswing.⁷⁵ A lack of weight transfer to the rear leg during the backswing may be caused by forward pelvic tilt, straining the golfer's spine and hips and reducing the golfer's capability of performing a properly sequenced downswing.⁷⁶ Professional golfers with LBP have displayed significantly more lead side lateral bending in the backswing.³⁸

Lumbar hyperextension

In an optimal address position, golfers have 45° of trunk flexion and a neutral spine profile.⁴⁷ In cases where lumbar curvature is abnormal, spinal vertebrae and musculature are placed in disadvantageous positions.⁶³ Compared to a neutral spinal position, 22.5° of lumbar flexion reduced trunk rotation ROM by 5% and pelvis ROM by 17%, while lumbar hyperextension (Fig. 4) reduced trunk rotation ROM by 4.2% but increased pelvis ROM by 4%.⁶³ Paraspinal muscle activity that compresses vertebrae to promote stability in suboptimal lumbar curves may contribute to the reduced ROM.^{19,63} A golf swing requires rotation of the trunk and pelvis and having a neutral lumbar spine at address increases a golfer's rotational capacity.

Lumbar hyperextension often occurring in the follow-through and is commonly referred to as the 'reverse C' position (Fig. 4). Anterior-posterior shear forces are increased when golfer hyperextend the lower back in the follow-through.^{54,77} Abdominal muscle activity during the early follow-through may indicate an effort to reduce lumbar hyperextension, while additionally increasing compressive forces.⁵⁴ However, no differences in abdominal muscle activity have been reported between healthy golfers and those with LBP.⁶⁷

Improper pelvic sequencing

Pelvic orientation impacts the musculature around the lumbar spine and the position of the vertebrae. Ideally, golfers address the ball with a neutral lumbar spine and slight anterior pelvic tilt. Variation in spinal positions affects the distribution of forces on the spine throughout the swing.⁷⁷ Proper pelvic motion in the golf swing has been previously described as anterior pelvic tilt at address, posterior pelvic tilt at impact, and anterior pelvic tilt in the follow through.⁷⁴ This sequence of pelvic motion is the result of proper muscle activation patterns for PDS in the swing (Fig. 5).

Anterior trunk muscle activation during the downswing may flex the lumbar vertebrae leading to posterior pelvic tilt.³⁸ However, it was hypothesized that golfers with LBP may have reduced anterior trunk muscle capacity,³⁸ with a lack of abdominal strength leading to improper pelvic

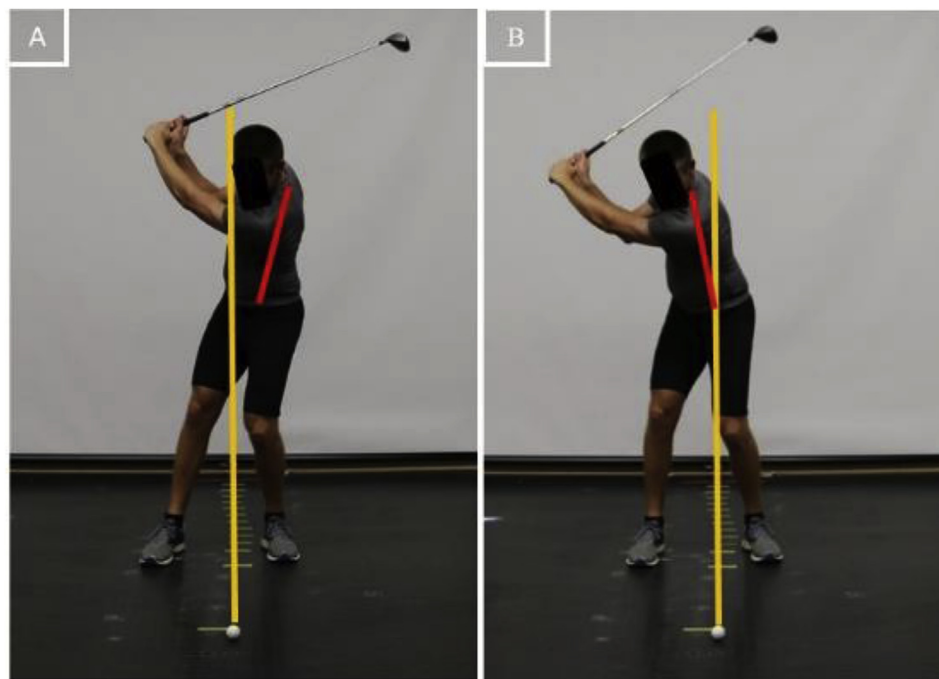


Fig. 3. Reverse spine angle represented by A. Leaning toward the lead side (A) decreases golfer's ability to rotate the pelvis and trunk during the downswing. Image B illustrates proper lateral weight shift during the backswing.



Fig. 4. Lumbar hyperextension at address (B) and the follow-through (C). Image A illustrates a neutral spine position at address. Lumbar lordosis, or “S-posture,” at address decreases pelvic mobility and increases spinal loads (B). The “reverse C” position at the end of the follow-through compresses lumbar vertebrae (C).

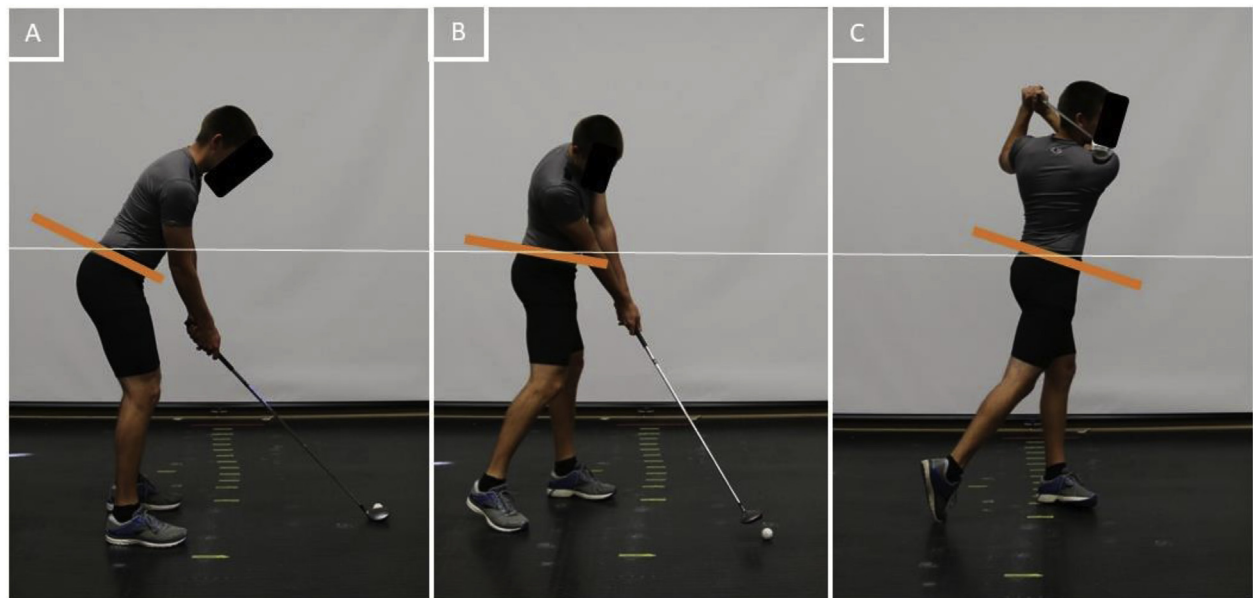


Fig. 5. Pelvic positioning at address (A), impact (B), and the follow-through (C). Proper pelvic sequencing is demonstrated in the figure. Pelvic positioning during the swing affects trunk and hip mobility, and improper sequencing can indicate muscular weaknesses.

motion. The interaction between the pelvis and lumbar spine relies on proper muscle activation patterns coupled with adequate ROM among the muscles manipulating the pelvis (e.g. EO, IO, ES, GMx, GMe).

In a health population, trunk and pelvis rotation were measured at different degrees of trunk flexion and with different spinal postures. Trunk rotation was optimized at 45° of trunk flexion with a neutral spinal posture, while pelvis rotation had an inverse relationship with trunk flexion angle (maximum pelvis rotation at 0° of trunk flexion).⁶³ Interestingly, pelvis rotation was largest when the spine was in a moderately kyphotic position and smallest when hyperextended. With suboptimal trunk or pelvis orientations, golfers may increase their risk for developing LBP because of limitations in rotational ROM. The nature of pelvic motion in golfers with LBP has been investigated in the transverse plane via hip rotation ROM, but more research is needed to analyze sagittal and frontal plane motion of the pelvis.

Conclusion

Individual golfers have unique swing techniques determined by physical characteristics and previously learned motor patterns. However, when the golf swing is technically flawed, there is a greater risk of experiencing injury,^{13,19,21,23,41} and with injury, golfers receive fewer health benefits because of reduced playing time.^{3,40} The most common golf injury occurs at the lower back.²³ A complex and asymmetrical motion, the golf swing causes imbalanced loading of the body's musculature which leads to the development of abnormal musculoskeletal characteristics, especially if a golfer is in compromising positions. The forces developed in the swing, hip strength and ROM, lateral bending coupled with pelvic rotation, and trunk over-rotation have all been previously associated with LBP. However, anecdotal evidence from golf teaching professionals has highlighted several positions during the golf

swing that may correlated with the development or etiology of LBP. Trunk motion during the backswing and follow-through, lumbar hyper-extension, and improper pelvic sequencing have all been identified as possible confounding factors contributing to LBP in golfers.

Practical application

This review should be used by golf teaching professionals and golf fitness professionals as an aid in assessing golfers experiencing LBP. Measurement of golfers' ROM and observation of their swing technique may allow instructors to isolate potential contributors to LBP. Because golfers have unique physical and swing characteristics, individualized evaluations should be performed primarily. Following evaluation, specialized physical training and swing technique training may benefit golfers with LBP.

In physical training, general strength and conditioning practices should be followed, especially for individuals with current LBP. Specific and progressive loading of the ES, GMx, GMe, and abdominal muscles can improve trunk stability throughout the swing, which may reduce LBP symptoms.²¹ Examples of exercise that can be used to strength trunk and hip muscles include: hip bridge, plank, bird dog, superman, and sit-ups.^{21,78} More recent evidence supports the use of resistance training to improve golfer performance and in rehabilitation. Increasing lower body strength through squatting and deadlifting can increase clubhead speed and strengthen anterior and posterior trunk muscles.⁷⁹ These exercises should be performed with correct form as directed by a fitness professional.

Prior to playing, golfers should complete a golf-specific warm-up beyond simply swinging a golf club. Researchers have developed rotational-based warm-ups that elicited significant increases in clubhead speed.^{80,81} Considering the LBP is most commonly a chronic injury, both physical training and pre-round warm-ups need to be performed consistently to maximize risk reduction. Additional investigations into golfers' daily activities and lifestyles may highlight factors outside of golf that contribute to lower back pain. This review is not an exhaustive list of risk factors for LBP, and should be used as a supplementary resource in the process of treating LBP. Future research should investigate the relevancy of the potential risk factors to the development of LBP.

Conflict of interest

There are no conflicts of interest associated with this publication.

Submission statement

This manuscript has not been published and is not under consideration for publication elsewhere.

Authors' contributions

NE designed and developed the project, performed the review, drafted and revised the paper. CD performed the review and revised the paper. HW designed and developed the project, performed the review, revised the paper.

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