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Risk Factors Associated With Low Back Pain in Golfers: A Systematic Review and Meta-analysis

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Risk factors associated with low back pain in golfers: a systematic review and meta- analysis

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ABSTRACT

Context

Low back pain is common in golfers. The risk factors for golf-related low back pain are unclear, but may include individual demographic, anthropometric and practice factors as well as movement characteristics of the golf swing.

Objective

The aims of this systematic review were to summarize and synthesize evidence for factors associated with low back pain in recreational and professional golfers.

Data sources

A systematic literature search was conducted in the PubMed, CINAHL and SPORTDiscus electronic databases through September 2017.

Study selection

Studies were included if they quantified demographic, anthropometric, biomechanical, or practice variables in individuals with and without golf-related low back pain.

Study design

Systematic review and meta-analysis

Level of evidence

3

Data extraction

Studies were independently reviewed for inclusion by two authors and the following data were extracted: the characterization of low back pain, participant demographics, anthropometrics, biomechanics, strength/flexibility and practice characteristics. The methodological quality of studies was appraised by three of the authors using a previously published checklist. Where possible, individual and pooled effect sizes of select variables of interest were calculated for differences between golfers with and without pain.

Results

The search retrieved 73 articles. Nineteen of these met the inclusion criteria, including twelve case-control studies, five cross-sectional studies, and two prospective longitudinal studies. Methodological quality scores ranged from 12.5 to 100.0%. Pooled analyses demonstrated a significant association between increased age and body mass and golf-related low back pain in cross-sectional/case-control studies. Prospective data indicated that previous history of back pain predicts future episodes of pain.

Conclusion

This review indicates that individual demographic and anthropometric characteristics may be associated with low back pain but does not support a relationship between swing characteristics and the development of golf-related pain. Additional high-quality prospective studies are needed to clarify risk factors for back pain in golfers.

Keywords

Golf, low back pain, swing, biomechanics, risk factors

INTRODUCTION

Golf is one of the most frequently played sports in the world. More than 6 million people across Europe and 26 million in the United States report playing at least one round per year.[17] Due to the physical activity and social interaction inherent in the sport, playing golf is associated with benefits to cardiovascular, respiratory and metabolic health, particularly in older adults.[42] However, in comparison with other sports and recreational activities, golf is also associated with a moderate risk of musculoskeletal injury.[7,47] Low back pain (LBP) is one of the most common musculoskeletal problems reported by recreational and professional golfers.[21,39,40] The prevalence of low back injuries has been estimated at between 15 to 35% in amateurs and up to 55% in professionals,[10] and is associated with significant time lost from golf play and practice.[16,21] Multiple factors have been identified as potential causes of LBP in golfers. These include movement characteristics of the golf swing, individual demographic and physical characteristics, and volume of play/practice.

Back pain in golfers is often attributed to the mechanical demands of golfing. The golf swing is a repetitive, asymmetrical motion that is associated with high segmental angular velocities and substantial compressive, torsional and shear loading of the spine.[28] In particular, several characteristics of modern swing technique have been identified as potential contributors to low back pain. In comparison with traditional swing mechanics, modern swing technique utilizes increased separation between the upper trunk/shoulders and pelvis at peak backswing and during the downswing.[10,18] The separation angle between the upper trunk and pelvis is called the **"X-factor"(Figure 1a).** Increasing the X-factor may enhance angular velocity of the trunk toward the lead (non-dominant) side and therefore increase the velocity of the clubhead[20] but also requires adequate spinal mobility. Modern swing technique is also associated with increased lateral flexion to the trail (dominant) side. This peaks at impact and during early follow-through. The combination of axial plane angular velocity toward the lead side and lateral flexion toward the trail side is termed the **"crunch factor"(Figure 1b**).[41,50] An additional component of modern swing that has been proposed to contribute to low back pain is the trunk hyperextension, or “reverse – C” position that occurs during follow-through **(Figure 1c)**.[10] Increased trunk hyperextension and crunch factor may result in greater compressive and shearing forces on the lumbar spine. To date however, there is no clear evidence regarding swing mechanics and the development of low back pain in golfers.

In addition to the mechanics of the golf swing, factors specific to the individual golfer have been proposed to increase the risk of developing LBP. These include limited or asymmetrical hip rotation range of motion,[43] increasing age,[51] and the method used to transport the golf bag.[45] As most low back pain in golfers is attributed to overuse or repetitive strain rather than a single precipitating event,[36] the frequency and duration of playing and practice has also been hypothesized to contribute to symptoms, particularly in professionals.[42] However, the evidence for any of these factors is limited and often conflicting.

Due to the popularity of golf, it is important to establish evidence-informed preventative and rehabilitation strategies for low back pain in golfers. The objective of this review therefore was to systematically appraise, and synthesize where possible, evidence for risk factors that may be associated with low back pain in recreational and professional golfers.

METHODS

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (PRISMA) guidelines were utilized in the development of this review.[33] The protocol was registered in the International Prospective Register of Systematic Reviews (*PROSPERO:* CRD42017067927).

Eligibility Criteria

Peer-reviewed studies were included if they quantified demographic, anthropometric, biomechanical, or practice variables in individuals with and without golf-related LBP. Studies of amateur and professional golfers of all ages and abilities were included. Case-control, cross-sectional and prospective longitudinal study designs were eligible for inclusion. Studies were excluded if they were conference abstracts, case reports, treatment studies, review articles, or if they did not include comparisons of individuals with and without back pain. Studies were also excluded if the full-text was not available in English.

Search strategy

A literature search was conducted in October 2016 in the PubMed, CINAHL and SPORTDiscus electronic databases, without date restriction. The search terms were entered in three groups: 1) low back pain and synonyms (lower back pain, lumbago, sciatica, back ache); 2) golf; and 3) modern swing, swing characteristics, crunch factor, kinematics, kinetics, EMG, biomechanics, handicap, epidemiology, risk factors, risks, predictors and injury prevention. The terms from all three groups were combined with 'AND'. Terms within groups were combined with 'OR'. Reference lists from all accessed articles and previous reviews were also screened to identify any additional relevant studies. The search was repeated using the same search terms in the same databases on 25th September 2017 to identify any research articles published since the original search.

Study selection/data extraction

Two authors independently reviewed the titles and abstracts of all the identified studies to determine eligibility. The following data were extracted from eligible studies:

- Study design
- Study population and sample size (setting, recruitment approach)
- Definition/criteria for low back pain
- Demographics
- Anthropometric variables
- Biomechanical golf swing variables
- Strength and flexibility variables
- Practice/expertise variables

- Other factors (e.g. transport of golf clubs)

Quality assessment

Assessment of study quality and risk of bias was conducted utilizing a previously published 16-item checklist (Table 1).[23,55,56] The total quality score was calculated as the sum of all positively-scored checklist items from numbers 3 – 16 relevant to that study type, divided by the total possible score for that study type (8, 12 and 9 for cross-sectional, case-control and prospective cohort studies respectively) and expressed as a percentage score. Three of the authors (JAS, AH and SPL) first independently scored the studies. The three authors then discussed any study where there was disagreement until a consensus score was reached.

Data synthesis

Where possible, effect sizes for case-control or cross-sectional group comparisons were extracted or calculated. For continuous data, the standardized mean difference was calculated utilizing Cohen's *d*. Confidence intervals (CI) for the Cohen's *d* estimate were also calculated utilizing the z or t-distribution for samples larger or smaller than 30 individuals respectively. Odds ratios (OR) and confidence intervals were extracted or calculated where possible for dichotomous data. For studies where sample frequencies or means and standard deviations/standard errors were not reported, attempts were made to contact the authors to request the data. Meta-analysis, consisting of calculation of a pooled standardized mean difference and 95% confidence interval was then conducted for all variables for which appropriate data were available in at least 2 studies, and where studies were sufficiently similar in population and outcome assessment. A random effects model was utilized to account for remaining study heterogeneity.[5] The I^2 statistic was also calculated, with I^2 greater than 0.75 indicative of substantial heterogeneity across studies.[25] For prospective longitudinal studies, statistical measures of the relationship between independent variables and occurrence of low back pain over the study period were extracted. All statistical analyses were conducted with the open-source R statistical platform (R Foundation for Statistical Computing, Vienna, Austria, version 3.4.1).[13]

RESULTS

Search results

Nineteen studies were retained for the review. Of these, twelve were case-control studies, five were cross-sectional studies and two were prospective longitudinal studies. (Figure 2)

Study characteristics

Ten of the studies investigated recreational golfers. Of these, three specified a minimum duration of golf experience or frequency of play for inclusion[11,15,44] , and two required a handicap below 20.[30,52] Three studies included both professional and elite recreational golfers,[21,27,35] and four investigated professional golfers exclusively.[16,22,34,53] (Table 2)

Methodological quality

Agreement among the three reviewers on each checklist item ranged from 80 to 100%. The least agreement occurred on items 4 (participation rate) and 14 (control of individual confounding factors). (Table 2)

Prevalence and incidence of low back pain

In the cross-sectional studies, prevalence of golf-related LBP in recreational golfers varied from 12.4%[21] to 26.9%.[3] In cross-sectional studies of professional golfers, prevalence ranged from 40.0 to 58.1%.[21,22] In these studies, it was unclear whether the reported prevalence of low back pain was specific to the time of the study, over the course of a year, or lifetime prevalence. In the longitudinal studies, the incidence of new or recurrent back pain episodes was 31.6% (novice recreational golfers) and 57.1% (young elite golfers) across the course of a year or a playing season respectively.[6,16]

Demographic factors

The pooled results from nine case-control and cross-sectional studies indicated that greater age was significantly associated with LBP (SMD 0.57, CI 0.07 – 1.07, I^2 79.9%, Figure 3a). The studies included in this meta-analysis included cohorts of both professional and recreational golfers with disparate age distributions. Therefore, separate sub-analyses for the relationship between age and LBP in recreational and professional golfers were also conducted. These demonstrated the same trends (recreational golfers SMD 0.50, CI -0.14 – 1.14, I^2 80.0%; professional golfers SMD 0.83 CI -0.95 – 2.61 I^2 89.1%, Figure 3b). One of the four studies reporting the association between sex and LBP found that male golfers are more likely to experience pain (OR 3.4, CI 1.3 - 13.4),[44] but this finding was not replicated in other cohorts.[3,40,43] One study reported a higher percentage of low back injuries in professionals compared with recreational golfers (OR 4.7, CI 2.7 - 8.3).[21] In the prospective study data, the only demographic factor that was a significant predictor of occurrence of back pain over twelve months (in novice recreational golfers) was a previous history of back pain (relative risk 9.8, CI 4.5 - 21.4).[6]

Anthropometric characteristics

Pooled results from case-control and cross-sectional studies indicated that mass was significantly associated with LBP (SMD 0.36, CI 0.09 - 0.63, I^2 0.0%, Figure 4a). Golfers with LBP were heavier than healthy controls. Separate sub-analyses for recreational and professional golfers were again conducted to account for the different data distributions in each group. Sub-analyses demonstrated that a relationship between mass and low back pain existed only in recreational golfers (recreational golfers SMD 0.64, CI 0.21 – 1.06, I^2 0.00%; professional golfers SMD 0.08 CI -0.45 – 0.60 I^2 0.00%, Figure 4b). One longitudinal study showed that, in trainee professional golfers, **Body Mass Index (BMI)** was significantly negatively correlated with frequency (% time) of LBP symptoms over a 10-month period (r = -0.7).[16] There was no evidence that hand dominance is associated with LBP.[43]

Golf swing movement characteristics

Kinematic and muscle activation characteristics of the golf swing in individuals with and without LBP were investigated in seven case-control and cross-sectional studies. All but two studies[34,52] divided the swing into address, backswing, downswing, impact and follow-through events and phases.

Pooled analyses of kinematic data (Table 3) were limited by heterogeneity in methodology, particularly in the approach taken to modelling trunk motion, and results were inconsistent. Two studies investigated crunch factor, defined as the instantaneous product of trunk or lumbar axial angular velocity and trunk or lumbar lateral flexion angle. There was no significant difference between peak crunch factor in individuals with and without LBP in either study. Peak X-factor was reported in two studies, with conflicting results (Table 3).

Two studies investigated the timing of trunk muscle activity during the golf swing.[12,27] Pooled analysis of both studies indicated no relationship between timing of lead side external oblique onset relative to the beginning of backswing in golfers and LBP (SMD -1.33 CI -4.83 – 2.18, I^2 95.82). Cole et al., reported that onsets of bilateral upper and lower lumbar erector spinae were earlier relative to the beginning of backswing in the LBP group (*d* range = 0.7 – 1.0*).*[12] In one study, differences in amplitude of erector spinae and external oblique activity between individuals with and without LBP showed different trends in high-handicap and low-handicap golfers,[9] while another reported no difference in abdominal muscle activity between groups in professionals.[27] Silva et al., [48] reported that activity of the lead biceps femoris during backswing was the most important factor to distinguish between golfers with and without LBP using a non-linear machine learning approach.

Strength/flexibility characteristics

Several cross sectional/case control studies demonstrated a relationship between trunk and hip muscle performance and LBP. (Table 4) Peak trunk extensor strength, endurance of the trunk extensors and flexors, and endurance in the side bridge position did not predict development of LBP over 10 months in young professionals.[16] However, side-to-side asymmetry of side-bridge endurance was significantly associated with development of LBP (r = 0.6), explaining 36% of the variability.

Pooled analyses of trunk extension range of motion data (SMD 3.2, CI -2.6 - 9.0, I^2 98.0%) and two out of three individual studies investigating active trunk motion in all other planes did not indicate an association between trunk range of motion and LBP.[30,52,53] Four studies investigated hip ranges of motion. Pooled analyses of lead and trail hip internal rotation did not demonstrate an association between range of motion and LBP (lead limb SMD 1.25, CI -1.3 - 3.8, I^2 96.8; trail limb SMD 0.13, CI -0.3 - 0.5, I^2 0.0%). Similarly, lead and trail hip external rotation were not associated with LBP (lead limb SMD 0.1, CI 0.7 - 0.9, I^2 61.3%; trail limb SMD 0.1, CI --0.9 - 1.1, I^2 72.8%). Two studies reported that side-to-side asymmetry in hip internal rotation was significantly greater in individuals with LBP, with the LBP groups having reduced range of motion in the lead hip[43,53] but appropriate data were not available to pool these results or calculate effect sizes.

Practice characteristics

The pooled analysis of case-control and cross-sectional studies demonstrated no relationship between handicap and low back pain (SMD 0.0, CI -0.3 - 0.4, I^2 0.0%). Although multiple studies investigated frequency and duration of play/practice, the heterogeneity in how practice characteristics were measured precluded pooled analyses. One study reported that there was a lower risk of LBP in individuals who performed less than 1 hour of full shot practice per week (OR 0.5, CI 0.3 - 0.8)[40] and another described increasing rates of spinal pain with increasing rounds and shots played per week.[21] However, multiple other studies found no significant difference in playing frequency or chipping/full shot practice in individuals with and without LBP.[3,34,43,44] There was no evidence of any influence of warm-up, stretching or strengthening behaviors on LBP status in either the case-control/cross-sectional[21,22] or prospective studies.[6] Gosheger et al.,[21] reported that in their sample, individuals who reported regularly carrying their golf bag were significantly more likely to have experienced LBP.

DISCUSSION

This study confirms that LBP is a widespread problem in golfers. Pooled analyses indicated that LBP is associated with individual demographic and anthropometric characteristics, but current evidence does not conclusively link kinematic or electromyographic features of swing technique to golf-related LBP.

In this review, age and previous history of symptoms emerged as potential contributors to LBP. The average age of recreational golfers in the pooled data was 51.5 years, consistent with reported average ages of recreational golfers in the US, Europe and Australia.[2,14,49] In the general population, the prevalence of LBP also increases with age until the sixth decade.[29] This has been attributed to a transition from short, acute episodes of pain in young adulthood to more persistent symptoms over time.[54] One high quality longitudinal study indicated that the strongest predictor of future episodes of golf-related LBP is a previous history of low back pain.[6] This finding also supports results from studies of the general population and in other athletic groups.[8,54] Other predictors of future episodes of LBP following an initial episode include the severity of pain during the initial episode,[19] alterations in central nervous system structure and function[38] and depression and psychological distress.[46] These factors were not investigated in any of the studies reviewed and should be included in future studies of golf-related LBP.

This review found that in recreational golfers, as in non-golfers, greater mass is associated with more LBP. This is potentially due to increased spinal loading. However, increased mass may also be a consequence of reduced physical activity due to the presence of pain.[32] In young professional golfers in contrast, development of LBP over time was associated with a lower BMI. The mechanism by which lower BMI may increase risk for low back pain does not appear to be mediated by muscle mass, as in the longitudinal study by Evans et al.,[16] there was no relationship between BMI and strength. They speculated that taller individuals with lower body mass may be at heightened risk of injury due to increased trunk range of motion or increased lever arm for forces at the spine, but these hypotheses have not been further examined.

This study does not indicate a consistent link between features of modern swing and golf-related low back pain. Increased X-factor, crunch factor and trunk hyperextension may all result in greater loading of the spine and may be associated with asymmetrical patterns of spinal degenerative changes.[50] However, the absence of significant group differences in these swing mechanics in current studies likely reflects a multifactorial relationship between cumulative mechanical loading and an individual's risk of developing low back pain. Although two small studies demonstrated altered timing and activation of the trunk musculature during the swing in individuals with back pain, the characteristics that were affected were inconsistent and varied in golfers with high and low handicap.[9] As substantial evidence in non-golfers indicates that motor control adaptations with low back pain are highly individual,[26] further research with larger samples will be needed to elucidate changes in motor control of the trunk musculature in specific sub-groups of golfers. Additional epidemiological work will also be needed to clarify if the prevalence of LBP is increasing as result of changes to swing mechanics.

The results in this review do not support a relationship between lead/trail hip range of motion and LBP. Biomechanical analysis in healthy professional golfers indicates that golfers with limited lead hip internal rotation utilize greater lumbopelvic motion throughout the golf swing and suggests that this increased spinal motion may lead to back pain over time.[31] However, this relationship is not consistently evident in current research, and this may be due to disparities between available single-planar joint range of motion measured in an unweighted position and the dynamic, multi-planar motion utilized during the swing.[24]

Individual cross-sectional and case-control studies reported impairments in multiple aspects of trunk muscle performance in golfers with LBP. As these studies examined different variables, data could not be pooled.[15 35].[52] Decrements in trunk muscle strength and endurance have also been reported in non-golfers with low back pain. These have primarily been attributed to deconditioning, exertional pain, and fear avoidance.[1,4,37] In the longitudinal study that reported that trunk endurance asymmetry was predictive of back pain in young elite golfers, multiple participants had a history of LBP at baseline and therefore it is unclear to what extent this strength asymmetry was a result of previous episodes of pain rather than a cause of ongoing symptoms.

Pooling of data in this review was limited by study heterogeneity and is reflected by high I^2 statistics for some variables. There was substantial variability in how LBP was operationalized in terms of severity or duration across studies. Additionally, studies that investigated the biomechanics of the golf swing utilized disparate approaches to estimating global or regional trunk motion. The methodological quality of studies in this review varied widely. However, quality scores in the present study were similar to those in previous systematic reviews of risk factors for musculoskeletal disorders utilizing the same methodological checklist .[23,56] Only three studies in the review controlled for potential confounding factors in the analysis [6,9,44] and five reported measures of association and confidence intervals.[6,16,43,44,48] Very few reported the participation rate relative to the available population or utilized blinded assessment.

CONCLUSION

Age and body mass are associated with golf-related low back pain. BMI and previous history of back pain may predict golfers who will experience symptoms. However, due to generally low quality and heterogeneity of current evidence, additional research is needed to facilitate evidence-based prevention and rehabilitation of low back pain in golfers.

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Table 1. Checklist for assessment methodological quality for cross sectional (CS), case-control (CC) and prospective cohort (PC) study designs.[23,56]

Domain	Item #	Description	CS	CC	PC
Study objective					
	1	Positive, if the study had a clearly defined objective	+	+	+
Study population					
	2	Positive, if the main features of the study population are described (sampling frame and distribution of the population according to age and sex)	+	+	+
	3	Positive, if cases and controls are drawn from the same population and a clear definition of cases and controls is given and if subjects with the disease/symptom in the past 3 months are excluded from the control group		+	
	4	Positive, if the participation rate is at least 80% or if the participation rate is 60–80% and the non-response is not selective (data shown)	+	+	+
	5	Positive, if the participation rate at main moment of follow-up is at least 80% or if the non-response is not selective (data shown)			+
Measurements					
	6	Positive, if data on history of the disease/symptom is collected and included in the statistical analysis	+	+	+
	7	Positive, if the outcome is measured in an identical manner among cases and controls		+	
	8	Positive, if the outcome assessment is blinded with respect to disease status	+	+	
	9	Positive, if the outcome is assessed at a time before the occurrence of the disease/symptom		+	
Assessment of the outcome					
	10	Positive, if the time-period on which the assessment of disease/symptom was based was at least 1 year			+
	11	Method for assessing injury status: physical examination blinded to exposure status (+); self-reported: specific questions relating to symptoms/disease/use of manikin (+), single question (−)	+	+	+
	12	Positive, if incident cases* were included (prospective enrolment)		+	
Analysis and data presentation					
	13	Positive, if the measures of association estimated were presented (OR/RR), including CI and numbers in the analysis	+	+	+
	14	Positive, if the analysis is controlled for confounding or effect modification: individual factors	+	+	+
	15	Positive, if the analysis is controlled for confounding or effect modification: other factors	+	+	+
	16	Positive, if the number of cases in the final multivariate model was at least 10 times the number of independent variables in the analysis	+	+	+
Total possible score (sum of items 3 – 16)			**8**	**12**	**9**

Table 2. Overview of cross sectional (CS), case-control (CC) and prospective cohort (PC) studies included in review. Bold font indicates that the study found a significant difference between golfers with and without low back pain (LBP) for that variable.

Study	**Design**	**Quality score (%)**	**Population characteristics**	**Low back pain criteria**	**N (M:F)**	**Potential risk factors (group comparisons available)**				
						Demographics	Anthropometrics	Swing characteristics	Strength/ flexibility	Practice characteristics
Batt 1992[3]	CS	12.5	Members of a British golf club	Site of injury (back); differentiated between injuries received playing golf and injuries affecting golf	193 (164:29)	Age; sex				Handicap; years of experience; rounds per month
Burdorf et al., 1996[6]	PC	100.0	Male novice recreational golfers at Dutch ranges and clubs	Lifetime history of low back pain (**frequency, duration and severity of episodes**); 1-year incidence of back pain	196 (196:0)	Age; education; occupation; physical activity at work;	Height; weight			Involvement in other sports; playing frequency; handicap at 1 year; number of lessons; regular warm-up
Cole & Grimshaw 2008[12]	CC	25.0	Not reported	≥20mm pain severity on VAS	27 (27:0)	Age	**Height**; mass; BMI	Onset and cessation of external oblique and **erector spinae** activity		Handicap
Cole & Grimshaw 2008[9]	CC	33.3	Not reported	≥20mm pain severity on VAS	30 (30:0)	Age	Height; **mass**	Amplitude of **external oblique** and **erector spinae** activity		Sub-grouped into high-handicap and low-handicap cohorts
Cole & Grimshaw 2014[11]	CC	25.0	Golfers at local private and public courses in Australia; over 18 years; playing for >12 months; current handicap	History of LBP when playing or practicing golf	27 (27:0)	Age	**Height;** mass; BMI	Trunk lateral flexion; trunk and hip axial rotation and separation angle; trunk axial angular velocity; crunch factor		Handicap
Evans & Oldreive 2000[15]	CC	16.7	Recreational golfers from single UK club; playing twice weekly; age 20-45 years; playing > 2 years	History of LBP that prevents playing golf in last 2 years; no pain in previous 3 months	20 (20:0)				**Endurance of transversus abdominis muscle**	

Evans et al., 2005[16]	PC	33.3	Trainee professionals in the Queensland PGA	Moderate or severe symptoms; symptom impact on golf; presence/absence of leg pain	14 (14:0)		**BMI**		Endurance of abdominals and erector spinae; **endurance asymmetry;** peak hip and trunk extensor strength; hamstring and **hip flexor** flexibility; lumbar spine range of motion	
Gosheger et al., 2003[21]	CS	12.5	Golfers at 24 German courses; professional and recreational golfers	Site of symptoms (lumbar, thoracic, cervical spine, categories collapsed into spine for most analyses); trauma versus overuse; duration of absence from golf; symptoms related to or unrelated to golf	703 (456:187)	Age; sex	**BMI**			Stretching and warm-up behaviors; rounds per week; driving range shots per week; **golf bag carrying; professional status**
Gulgin & Armstrong 2008[22]	CS	12.5	Professional golfers on LPGA Tour	Site of symptoms (right, left, bilateral, upper back, mid back, low back)	31 (0: 31)	Age	Height; weight		Passive hip internal and external rotation range of motion; side-to-side asymmetry	Stretching routine; strengthening program
Horton et al., 2001[27]	CC	25.0	Professional and elite recreational golfers; members of Alberta PGA or Alberta GA; under 55 years	Report of always or often experiencing LBP after golf; symptoms for > 6 months	18 (18: 0)	Age	Height; weight; BMI	Amplitude of rectus abdominis, external oblique and internal oblique activity before and after practice session; **onset of external oblique** and internal oblique before and after practice session	Abdominal muscle fatigue before and after practice session	

Kalra et al., 2012[30]	CC	25.0	Handicap ≤ 20; right-handed; 25-65 years	History of LBP for > 2 weeks; affecting golf within past year; ODI score ≥ 24%; symptoms central or on right side; symptoms resulted from or aggravated by golf;	30 (not reported)				**Trunk strength; trunk range of motion; hamstring flexibility**	
Lindsay & Horton 2002[34]	CC	25.0	Members of Alberta PGA	Report of always experiencing LBP after golf	54 (54:0)	Age	Height; mass	Trunk flexion, extension, **lateral flexion**, axial rotation; peak trunk angular **flexion**, extension, **lateral flexion** and axial velocity		Rounds per month; practice sessions per month; balls per practice session; putting sessions per month; time per putting session
Lindsay & Horton 2006[35]	CC	25.0	Members of Alberta PGA; elite amateurs; patients of local physical therapy clinics; under 50 years	Report of always or often experiencing LBP after golf; symptoms for > 6 months	39 (39:0)	Age	Height; mass		Trunk axial rotation strength; trunk axial **rotation endurance**	
Murray et al., 2009[43]	CC	41.7	Members of two British golf clubs	Current LBP or history of LBP within previous year; symptoms for > 2 weeks; over-use rather than trauma	64 (43:21)	Age; sex	Height; **weight;** handedness		Hip active and passive **internal** and external rotation; side-to-side hip asymmetry	Handicap; rounds per week; years of experience
McHardy et al., 2007[40]	CS	12.5	Members of golf clubs randomly selected from across Australia	Current golf-related LBP or history of golf-related LBP in past 12 months	1725 (1316:318)	Age; sex				Handicap; duration of chipping/putting practice per week; **full shot practice per week**; games per week
Nicholas et al., 1998[44]	CS	75.0	Members of NY State GA; over 21 years; playing ≥ 1 year	Back condition from golf	368 (294:74)	**Age; sex;** history of smoking; history of alcohol intake	**Self-report of over-weight**			Handicap; holes per week; weeks of play per year; years of play
Silva et al., 2015[48]	CC	25.0	Right-handed golfers	Report of experiencing back pain after playing 18 holes > 65% of the time	21 (not reported)	Age	Height, mass	Discriminant capacity of non-linear muscle activation patterns of **rectus femoris, biceps femoris, semi-tendinosis,**		Handicap; years of play

								external oblique, erector spinae and gluteus maximus		
Tsai et al., 2010[52]	CC	25.0	Male, right-handed golfers with handicap < 20	Report of mechanical LBP aggravated by golf within previous 2 years; asymptomatic	32 (32:0)	Age	Height; mass	Axial trunk/pelvis separation; **peak axial trunk rotation**; peak L5-S1 moments	**Peak trunk and hip strength**; trunk and hip active range of motion; hamstring flexibility; FABER test; active spinal repositioning error; center of pressure velocity in single-limb stance	Handicap; estimated driving distance
Vad et al., 2004[53]	CC	33.3	Professional golfers on PGA Tour	Report of LBP limiting golf performance for > 2 weeks in previous year	42 (42:0)	Age			**Hip internal rotation range of motion**; FABER test; **side-to-side hip asymmetry;** trunk flexion and **lumbar extension** range of motion	

Table 3. Summary of individual study findings for swing kinematics, with calculated effect sizes (Cohen's d) and confidence intervals (CI) for group comparisons

Study	**Variable**	**Finding in LBP group**	**Swing phase**	**Effect size (CI)**
Lindsay & Horton 2002[34]	Peak trunk lateral flexion to lead side	Increased	Entire swing	2.0 (0.4 – 3.5)
Lindsay & Horton 2002[34]	Peak trunk lateral flexion angular velocity	Increased	Entire swing	1.3 (-0.1 – 2.7)
Lindsay & Horton 2002[34]	Trunk flexion angular velocity	Decreased	Entire swing	2.1 (0.5 – 3.7)
Tsai et al., 2010[52]	Peak trunk axial rotation to trail side	Decreased	Entire swing	1.6 (0.7 – 2.4)
Cole & Grimshaw 2014[11]	Peak crunch factor	No difference	Follow-through	0.1 (-0.7- 0.9)
Lindsay & Horton 2002[34]	Peak crunch factor	No difference	Entire swing	0.2 (-1.1 – 1.5)
Tsai et al., 2010[52]	Peak X-factor	No difference	Entire swing	0.3 (-0.4 - 1.1)
Cole & Grimshaw 2014[11]	Peak X-factor	Tend toward decreased	Peak backswing	0.7 (-0.1 – 1.6)

Table 4. Summary of individual study findings for trunk and hip muscle strength and performance, with calculated effect sizes (Cohen's d) and confidence intervals (CI) for group comparisons where appropriate data were available

Study	**Variable**	**Finding in LBP group**	**Effect size (CI)**
Evans & Oldreive 2000[15]	Transversus abdominis endurance	Decreased	1.3 (0.3 - 2.3)
Kalra et al., 2012[30]	Trunk strength in all planes	Decreased	
Lindsay & Horton 2006[35]	Trunk axial rotation endurance toward lead side	Decreased	1.4 (0.5 - 2.3)
Tsai et al., 2010[52]	Peak isokinetic trunk extension	Decreased	1.04 (0.3 - 1.8)
Tsai et al., 2010[52]	Peak isometric lead hip adduction	Decreased	1.0 (0.2 - 1.7)

Figure 1. Characteristics of modern swing technique. a) X-factor. Axial separation between upper trunk and pelvis at backswing and during downswing. b) Crunch factor. Combination of trunk lateral flexion and axial angular velocity at impact and early follow-through. c) Reverse-c. Trunk hyperextension during follow-through.

Figure 2. PRISMA flow chart of study search and inclusion procedures

Figure 3. Pooled standardized mean difference in age between golfers with and without low back pain. a) All available data. b) Sub-analyses of studies explicitly reporting samples of recreational (top) and professional (bottom) golfers.

Figure 4. Pooled standardized mean difference in body mass between golfers with and without low back pain. a) All available data. b) Sub-analyses of studies explicitly reporting samples of recreational (top) and professional (bottom) golfers.

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