MOVEMENT, THE MUSCULOSKELETAL SYSTEM AND LOWER BACK PAIN IN YOUNG ELITE MALE GOLFERS

Samantha-lynn Quinn

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

A thesis submitted to the Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, in fulfilment of the requirements for the degree Doctor of Philosophy.

Johannesburg, 2022.

DECLARATION

I, Samantha-lynn Quinn, declare that this thesis is my own, unaided work.

It is being submitted for the Degree of Doctor of Philosophy at the University of the Witwatersrand, Johannesburg.

It has not been submitted before for any degree or examination at any other University.

Quinn

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Signature of candidate

29 day of January 2022 in London, United Kingdom.

DEDICATION

To my amazing husband, for all his support and encouragement over this incredibly challenging time and to my fantastic three children Connor, Judah and Evelyn, who are a constant source of joy and happiness in my life.

JOURNAL ARTICLES EMANATING FROM THE WORK PRESENTED IN THIS THESIS.

Four journal articles have been constructed as fundamental pillars of the current thesis. Due to various time constraints, and the pressures of having to craft the thesis, while being a front-line health worker during the 2020-2021 COVID 19 crisis[1], I have managed to submit only two of the planned papers for publication. Paper 1 is based on chapter 3, paper 2 is based on chapter 4, the third and fourth papers are based on chapters 5 and chapter 6 respectively.

The two papers that have been submitted for publication and these include:

- Title: 'Lower quadrant swing biomechanics identifies golfers with increased risk of lower back pain: A prospective longitudinal cohort study' submitted to the Journal of Sports Rehabilitation. This paper has been invited to respond to the reviewers' comments and resubmit. This paper is based on chapter 3
- Title: 'Increased trunk muscle recruitment during the golf swing is linked to developing lower back pain: a prospective longitudinal cohort study' has been submitted to the Journal of Electromyography and Kinesiology. This paper has been invited to respond to the reviewers' comments and resubmit. This paper is based on chapter 4.

[1] Over the course of the COVID-19 pandemic I worked in one of London's biggest hospitals as a physiotherapist. This hospital was significantly impacted by the COVID-19 pandemic. I was involved in setting up a virtual musculoskeletal COVID-19 rehabilitation service. I also trained to become a vaccinator to assist with the United Kingdom's vaccination drive at one of the mass vaccination centres in London

CONFERENCE PRESENTATIONS

European Congress of Sport Science Conference 2020 (Online conference)

Date: 28-30 October 2021

Title: Injury in elite golfers: poor spinal muscle recruitment is linked to developing back pain.

Type: Oral presentation (virtual)

Presented by: Samantha-lynn Quinn

Authors: Samantha-lynn Quinn, Benita Olivier, Warrick McKinon, and Chloe Dafkin.

ABSTRACT

Young elite male golfers experience golf related lower back pain with a worrying prevalence. Little is known about the musculoskeletal association with lower back pain development in elite golfers.

The major purpose of this thesis was to identify the musculoskeletal factors related to lower back pain in young elite male golfers. This was achieved through four studies on elite male golfers recruited from an elite golf academy in South Africa. The first investigation focused on the lower quadrant swing kinematics associated with lower back pain. The second study explored whether spinal muscle recruitment was associated with developing lower back pain. The third aimed to establish if commonly used functional movement screening tools were able to identify golfers at risk of developing injury. The final study tested whether the measurements made in the above studies as well as conventional monitoring tools would identify parameters associated with improved performance amongst elite golfers.

Golfers who developed lower back pain displayed distinct differences in terms of their kinematics. Golfers at risk of developing lower back pain had: reduced non-dominant leg squat at set up and at the top of the backswing, increased dominant knee flexion during impact and decreased dominant hip abduction during the backswing and during follow through. The incidence of lower back pain was 41%. This considerable injury incidence in a young golfing population is concerning.

Furthermore, elite golfers who developed lower back pain had increased dominant *latissimus dorsi* and dominant *rectus abdominis* activation during the swing. This may have resulted in increased dominant side spinal compression. The increased muscle recruitment of dominant *latissimus dorsi* and dominant *rectus abdominis* appears to be a centralised movement strategy and was not as a result of pain at the time of measurement.

Out of 30 screening tests only three tests were associated with developing lower back pain (all three had weak effect sizes). Functional movement screening was not effective in identifying elite golfers at risk of injury in this study.

Swing characteristics did not affect overall performance, but golf drive distance did. This suggests that swing characteristic modifications, such as those identified in study one and two, can be made in an attempt to reduce lower back pain incidence without impacting negatively on performance, provided the golf drive distance is not negatively affected.

The findings of this PhD address the second step in the TRIPP (Translating Research into the Injury Prevention Practice) model of injury prevention, namely, identifying lower back pain associations. Now that musculoskeletal associations have been identified, future randomised controlled intervention studies should be conducted to determine if modifying these associations reduces the incidence of golf related lower back pain in young elite male golfers.

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LIST OF ABBREVIATIONS

%	percentage
abd:	abduction
add:	adduction
BMI:	body mass index
CI:	confidence interval
EMG:	electromyograpic
ext rot:	external rotation
ext:	extension
flex:	flexion
GRF:	ground reaction force
hr:	hour
int rot	internal rotation
IQR:	Inter quartile range
kg	kilograms
lat flex:	lateral flexion
m	metre
MVC:	Maximum voluntary contraction
n:	Number of participants
PGA:	Professional Golfers' Association
RCT:	Randomised controlled trial.
RE-AIM:	Reach, Efficacy, Adoption, Implementation, Maintenance
rot:	Rotation
SD:	Standard deviation
TRIPP:	Translating Research into the Injury Prevention Practice

OPERATIONAL DEFINITIONS

Angular speed:	Angular speed is ‘the ratio of angular displacement to the time interval’ (Serway et al., 2000).
Amateur golfer:	For the purposes of this study, amateur golfers were defined as any elite player who was not registered as a professional golfer on tour.
Ball speed (km/hour):	This is ‘the speed of the golf ball immediately after impact’ (Trackman, 2021c).
Carry distance:	This is a measure of how far the ball travels through the air. It is defined as ‘The straight line distance between where the ball started and where the trajectory crosses a point that is the same height as where the ball was hit’ (Trackman, 2021a).
Club head speed (km/hr):	This is a measure of the speed of the club immediately prior to impact (Trackman, 2021d).
Cocking of the wrist:	Position in which the golfer’s wrists are in extension. This usually occurs at the top of backswing. The wrists should remain cocked until just before impact (Hume et al., 2005).
Crunch factor	This is a function of 'instantaneous product of lumbar axial angular velocity and lumbar lateral flexion' (Smith et al., 2018).
Distal:	If something is distal it is further away from the structure’s origin (e.g. the hand is distal to the shoulder) (Drake et al., 2009).
Efficacy of an intervention:	This is the ‘performance of an intervention under ideal circumstances’ (Singal et al., 2014).
Effectiveness of an intervention:	This is the performance of an intervention under ‘real world’ circumstances (Singal et al., 2014).
Elite golfers:	This has been defined as ‘professional players competing on tour or amateurs competing in international/ national amateur championships

	(Murray et al., 2020).
Electromyography (EMG):	Electromyography is the study of muscle electrical activity. It gives information regarding muscle recruitment. It cannot be used to measure the strength of muscle contraction (Robertson et al., 2014).
False negative:	A false negative is also known as type II error. This is when a researcher falsely concludes that no effect occurred. In this case the researcher has wrongly failed to reject the null hypothesis (Allen, 2017).
False positive:	A false positive is also known as a type I error. This occurs when the researcher falsely concludes that an effect occurred. In this case the researcher has wrongly rejected the null hypothesis (Allen, 2017).
Functional movement ability:	'Functional movement ability' is a clinical measurement of a person's ability to perform functional movements. This is specific to the needs of the participant. E.g. a sportsman may have a more vigorous set of "functional movements" to perform than a geriatric patient. The tests chosen for this research were selected based on the following criteria: i) clinical reasoning indicated that the test may predict injury, and/or ii) the test is frequently used by clinicians to screen golfers, and/or iii) the test has been shown to predict injury in golf or other sports.
Golfer's hub centre:	This lies near the golfers sternum. This 'hub acts as a centre around which the moment arm rotates on a specific plane' (Hume et al., 2005).
Ground reaction forces (GRF):	A golfer will experience a reaction force from the earth, when he applies force to the earth, during the golf swing. This is called the ground reaction force (GRF). It is equal in magnitude to the force being

applied to the earth, but in the opposite direction (Robertson et al., 2014).

Figure 0.1. provides a visual representation of GRF experienced during the golf drive.

Figure 0.1: Ground reaction forces during the golf swing

Injury incidence:	This is rate at which injury occurs within a population (i.e. elite golfers) during a specified period (British Medical Journal Publishing Group, 2021).
Injury prevalence:	This is the proportion of the population (i.e. elite golfers) that has the injury at a specific time point (British Medical Journal Publishing Group, 2021).
Injury to the lower limb:	Pain or discomfort in the hip, knee, ankle, foot or any region of the lower limb. The injury did not need affect golf participation.
Injury to the neck or thoracic spine:	Pain or discomfort between the occiput and T12. The injury did not need affect golf participation.
Injury to the upper limb:	Pain or discomfort in the shoulder, elbow, wrist, hand or any region of upper limb. The injury did not need affect golf participation.
Kinematics:	Kinematics “is the study of motion without regard to causes” (Robertson et al., 2014).
Lead side:	The side of the golfer closest to the ball at set up (in

	a right handed player this is the left side).
Lower back pain:	This study is concerned with overuse injuries, and as such only golfers who sustain non-contact spinal injuries will be considered as having ‘lower back pain’. ‘Injury’ to the back was defined according to the European guidelines for defining lower back pain (Burton et al., 2006). Lower back pain was defined as ‘pain and discomfort that was localised below the costal margin and above the inferior gluteal folds, with or without leg pain’ (Burton et al., 2006). The lower back pain did not need to result in limited golf participation.
Lower quadrant	This includes the lower limbs and the lower back.
Moment arm	When there is a force acting to rotate an object about an axis, the moment arm is the vector distance perpendicular to the line of action of the force and the axis of rotation (Serway et al., 2000).
Professional golfer:	Strictly speaking a professional golfer can include both teaching professionals and golfers who play golf for a living on one of the Professional Golfers’ Association (PGA) Tours. For the purpose of this thesis a professional golfer is defined as a golfer who plays golf for a living on at least one of the PGA tours.
Proximal:	If something is proximal it is closer to the structure’s origin (e.g. the shoulder is proximal to the hand) (Drake et al., 2009).
Roll distance:	This is a measure of how far the ball rolls after landing (BogeyMedia, 2016).
Sensitivity:	Sensitivity is the ‘ability of a test to correctly identify patients with a disease’ (Swift et al., 2020).
Smash ratio:	This is a measure of the amount of 'energy is transferred from the club head to the ball at impact'

	(Trackman, 2020). The higher the smash ratio the better the energy transfer (Trackman, 2020). The ideal smash ratio is 1.5 (Trackman, 2020).
Specificity:	Specificity 'is the ability of a test to correctly identify people without the disease' (Swift et al., 2020).
Speed (average):	Speed 'is defined as the total distance travelled divided by the total time it takes to travel that distance.' (Serway et al., 2000)
Sway:	A 'sway' swing fault is 'an excessive lower body lateral movement away from the target during the backswing' i.e. The greater trochanter of the trail leg moves past an imaginary vertical line drawn from the lateral malleolus of the trail leg (Titleist Performance Institute, 2021h).
Swing characteristics:	Swing faults are technical 'errors' when the golfer is compared to a 'perfect' biomechanical model. Many elite golfers play with swing faults. These are not necessarily problematic in terms of performance, as many biomechanical adaptions occur due to anatomical variance. I have therefore opted for the term 'swing characteristics' rather than 'swing faults'.
Torque:	Torque is 'the magnitude of force causing rotation of an object' (Blazevich, 2010). Torque = force x moment arm (Serway et al., 2000)
Trail side:	The side of the golfer furthest from the ball at set up (in a right handed player this is the right side).
Total drive distance:	This is the straight line distance travelled by the ball from impact to its resting position (Trackman, 2021b). This is the sum of the carry distance, bounce and roll of the ball (Trackman, 2021b).
Velocity (average):	Velocity is defined as a 'particle's displacement

	divided by the time interval during which that displacement occurred'(Serway et al., 2000).
X-factor:	The difference between the torso and the pelvic rotation at the top of the golf swing is termed the X-factor (Hume et al., 2005)
Young elite golfers	Golfers aged 16 years to 30 years who play at a high level of golf. This includes golfers in this age category who play with a handicap below 6 or golfers who are enrolled at a golf academy with a handicap of 6 or above who are considered elite players by their coaches.

PREFACE AND DESCRIPTION OF THESIS SECTIONS

Treating elite golfers has been a passion of mine for more than 10 years. Over this time, I have become concerned by the number of young elite male golfers complaining of lower back pain. This observation and concern has been echoed in the literature. Young elite golfers have a lower back pain incidence of 57.1% over a one year monitoring period (Smith et al., 2018). This considerable incidence of lower back pain in young elite golfers places them at risk of developing chronic lower back pain, which is one of the leading causes of disability, and is set to increase significantly into the future, especially in low to middle income countries (Hoy et al., 2015)

Preventing golf related lower back pain requires a systematic approach. The Translating Research into the Injury Prevention Practice (TRIPP) model of injury prevention states that once the extent of concern has been established, the second step is to identify the factors associated with injury (lower back pain in this case) (Emery and Pasanen, 2019). According to the systematic review by Smith et al. (2018) this step has not been satisfied and there is a need for good quality prospective studies to clarify the risk factors for developing lower back pain in golfers. This research aims to address this second step by determining the musculoskeletal factors associated with developing lower back pain in young elite male golfers.

This research was presented in this thesis as four studies. The first two longitudinal prospective cohort studies presented aimed to identify the swing kinematics (chapter 3) and muscle recruitment strategies (chapter 4) that were associated with lower back pain in young elite male golfers.

The third longitudinal cohort study (chapter 5) built on these findings and aimed to determine if commonly used, inexpensive, functional movement screening tools were able to identify golfers at risk of injury and lower back pain. If these screening tools proved useful at identifying young elite golfers at risk of injury and lower back pain, then targeted intervention strategies, to address the deficiencies identified by study one (chapter 3) and study two (chapter 4), could be implemented in an attempt to reduce lower back pain incidence and prevalence.

The intervention strategies that are aimed at addressing factors associated with developing lower back pain in golfers are likely to include swing modifications. Swing modification in elite golfers needs to be undertaken with great care as this may affect the elite golfer's primary goal, which is to play excellent golf. With a person-centred approach to injury prevention in mind, it was essential to assess the performance impact of swing characteristics on young elite male golfers. This was achieved through study four (chapter 6), which was a cross-sectional study that aimed to identify the swing, club and ball factors associated with performance in young elite male golfers. The findings from this study will enable golfers, coaches and physiotherapists to make an informed assessment of the risks (to performance) and benefits (in terms of possible lower back pain incidence reduction) of undergoing a swing modification.

This research satisfies step two of the TRIPP model of injury prevention: to identify the risk factors associated with developing lower back pain in elite golfers. By achieving this aim, future researchers are provided with a strong foundation onto which they can build and start to tackle the third step of injury prevention, developing targeted intervention strategies based on the risk factors identified, which should ideally be tested in randomised controlled trials to see if they reduce the incidence and prevalence of lower back pain in young elite male golfers. For a more detailed discussion regarding the motivation and rationale behind this thesis see chapter 1.

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CHAPTER 1: INTRODUCTION

1.1 Introduction

Golf is a popular sport with a moderate risk for sports injury (Cabri et al., 2009, Smith et al., 2018). When looking at professional golfers specifically however, significantly more injuries are sustained when compared to amateur golfers (Cabri et al., 2009). Professional golfers have a lower back pain prevalence between 40-58% (Smith et al., 2018). Young elite golfers have an injury incidence of new or recurrent lower back pain over a one year period of 57.1% (Smith et al., 2018). The young age onset of injury is concerning as lower back pain is one of the leading causes of years lived with disability (Vos et al., 2012). I have been working with young elite golfers for the last 10 years, and my own experience in a South African context confirms these findings (anecdotal evidence). Junior golfers were included in this study and this is supported by a recent systematic review by Rössler et al. (2014) that showed that the six to 18 year old age category is of particular interest for future research as the proportion of life threatening injuries in this age group is considerably higher in children and adolescents (32%) compared to adults (9%). They also showed that injury prevention programmes can result in a 46% reduction in injury in youth sports. Male golfers were selected due to the low numbers of female golfers enrolled at the academy. It should also be noted that the majority of sports injury prevention research has been conducted on girls and that boys are highly under-represented. Rössler et al. (2014) reported that one-eighth of all participants included in injury prevention studies were boys. It was not possible to include girls in the analysis as their swing movement pattern and muscle strength differs with that of males (Zheng et al., 2008).

Most injuries experienced by elite golfers are overuse injuries and are related to times of intense practice (Cabri et al., 2009). As the majority of injuries in elite golfers seem to be related to the repetitive swinging of the golf club, the nature of the golf swing needs to be considered. In order to better understand the lower back pain mechanism, it is essential to take a closer look at the kinematics and muscle activation of elite golfers who develop lower back pain. In a systematic review conducted by Smith et al. (2018) they concluded that there is no clear evidence linking the development of lower back pain in golfers to swing mechanics. They go on to conclude that because of the low heterogeneity and quality of current research, more investigation into how to prevent lower back pain in golfers is required. An essential step in the Translating Research into the Injury Prevention Practice (TRIPP) model of sports injury prevention is identification of the factors associated with injury (Emery and Pasanen, 2019).

This research is focused on identifying the biomechanical and musculoskeletal factors associated with developing lower back pain in young elite male golfers. Epidemiological factors, health status, injury history and differences in training may also contribute to injury in golf and therefore need to be taken into account when identifying the musculoskeletal and biomechanical factors associated with developing lower back pain in elite golf. The considerable lower back pain incidence in elite golfers has prompted widespread use of functional movement type screening in an attempt by therapists and coaches to identify golfers at risk of developing lower back pain (Titleist Performance Institute, 2021b). There is, however, conflicting evidence regarding the usefulness of functional movement screening in identifying athletes at risk of injury (Moran et al., 2017) and this research aims to establish if functional movement screening is able to identify golfers at risk of developing injury. Finally, excellent performance is the ultimate goal of elite golfers. It is therefore essential to determine which factors are associated with performance so that golfers and coaches can make informed decisions about possible performance cost versus the possible injury reduction benefit of adopting an intervention designed to address a particular musculoskeletal or biomechanical factor associated with developing lower back pain.

1.2 Problem Statement

Currently, there are a large number of young elite golfers, with substantial rates of lower back pain. The musculoskeletal and biomechanical associations of lower back pain in elite golfers are unknown. Elite golfers can play with a variety of swing kinematic characteristics. The biomechanical and musculoskeletal factors associated with developing lower back pain in young elite male golfers are unknown. Without this information, physiotherapists and coaches are unable to suggest which swing style may reduce lower back pain incidence and prevalence. Injury prevention interventions should be directed at golfers at increased risk of developing lower back pain. Although movement screening tools are used widely by elite golfers, there is little information as to how effective they are at identifying golfers at risk of developing lower back pain. Finally, the biomechanical factors associated with performance in young elite golfers (including those related to injury risk) are not fully understood. Without this information golfers and coaches are unable to determine the performance cost versus the possible injury reduction benefit of adopting an intervention designed to address a particular musculoskeletal or biomechanical factor.

1.3 Overall aim

The aim of this research was to identify the biomechanical and musculoskeletal factors associated with developing lower back pain in young elite male golfers.

1.4 Objectives:

The objectives of this research were to:

- Describe the lower quadrant 3D kinematics of the golf swing in young elite male golfers (chapter 3).
- Identify the demographics and injury history of young elite male golfers (chapter 3-5).
- Establish how intensely and frequently young elite male golfers train (chapter 3-5).
- Establish the incidence of injury in young elite male golfers over a period of six months (chapter 3-5).
- Identify the musculoskeletal and biomechanical factors associated with developing lower back pain in young elite male golfers (chapter 3-5).
- Determine the golf swing performance (performance of the club and ball flight) of young elite male golfers (chapter 3-6).
- Determine the competition performance (order of merit) of the young elite male golfers enrolled in the study (chapter 3-6).
- Describe the muscle activation of young elite male golfers during the swing (chapter 4).
- Establish the functional movement ability of young elite male golfers (chapter 5).
- Determine if commonly used screening tools were able to identify young elite male golfers at risk of developing injury (chapter 5).
- Identify the swing characteristics of young elite male golfers (chapter 6).
- Identify the musculoskeletal and biomechanical factors associated with performance in young elite male golfers (chapter 6).

All of the above mentioned objectives were met.

1.5 Significance of the study

By establishing the factors associated with developing lower back pain in young elite male golfers physiotherapists and coaches will be able to identify golfers at risk of developing lower back pain. Once these golfers have been identified, they may be given appropriate exercise programmes to address musculoskeletal problems and functional movement problems; and/or their golf swing biomechanics could be altered to a preferable swing pattern (in terms of injury). These interventions should then be monitored using randomised controlled trials to determine if they reduce injury incidence. Once the factors associated with performance are established physiotherapists, coaches and golfers will be able to assess the performance cost versus the injury reduction benefit of adopting an intervention designed to address a particular musculoskeletal or biomechanical factor.

1.6 Study setting

The studies were conducted at the Golf School of Excellence, Huddle Park Johannesburg. This is an elite golf academy that offers: PGA (Professional Golf Association) Diploma, Junior Academy (full-time golf programme for 14-18 year olds), Greenkeeping course, Academy Programmes (full time or part-time holistic elite training programme that prepares golfers for a playing career) and private lessons. Although the studies were conducted concurrently and many of the golfers partook in all four studies, the cohorts were not identical. E.g. Some of the golfers were excluded from the EMG studies because their sensors did not trace. Some of the golfers from the performance study were excluded because they did not take part in the yearly order of merit competition.

1.7 Type of study

This study comes from a post positivism worldview (Cresswell and Clark, 2009). The research asks the question, ‘Which biomechanical and musculoskeletal factors are associated with lower back pain in young elite male golfers?’. In order to answer this question empirical data will be analysed, and as such this study requires a quantitative approach. Based on this reasoning the researcher decided to conduct a quantitative empirical research study on the biomechanical factors associated with developing lower back pain in young elite male golfers. The findings of this research will be presented as three prospective longitudinal cohort studies and one cross-sectional study.

1.8 Conceptual framework

This research ascribes to the TRIPP model of injury prevention (Emery and Pasanen, 2019). The studies presented in this research aim to satisfy step two of the TRIPP model of injury prevention, namely, to identify the risk factors associated with developing lower back pain in elite golfers. A more detailed discussion of this framework is provided in section 2.4.

1.9 Outline of this thesis

Chapter 1: Provides an introduction to the thesis and details the significance of the studies conducted in this thesis.
Chapter 2: Is a literature review in which the relevant current research on young elite male golfers is summarised and applied to the studies conducted in this thesis.
Chapter 3: Presents the findings of the first study, which aimed at answer the question: Which lower quadrant kinematic factors are associated with developing lower back pain in young elite male golfers?
Chapter 4: Presents the findings of the second study, which aimed to answer the question: Which muscle activation factors are associated with developing lower back pain in young elite male golfers?
Chapter 5: Presents the findings of the third study, which aimed to answer the question: Are commonly used functional movement screening tools were able to identify golfers at risk of injury?
Chapter 6: Presents the findings of the fourth study, which aimed answer the question: Which swing characteristic factors associated with performance in young elite male golfer?
Chapter 7: Provides a conclusion to the thesis by summarising the major findings and focusing on the overall clinical significance and impact of this thesis. It also provides recommendations for future studies.
An overview diagram of this thesis has been provided in Figure1.1.

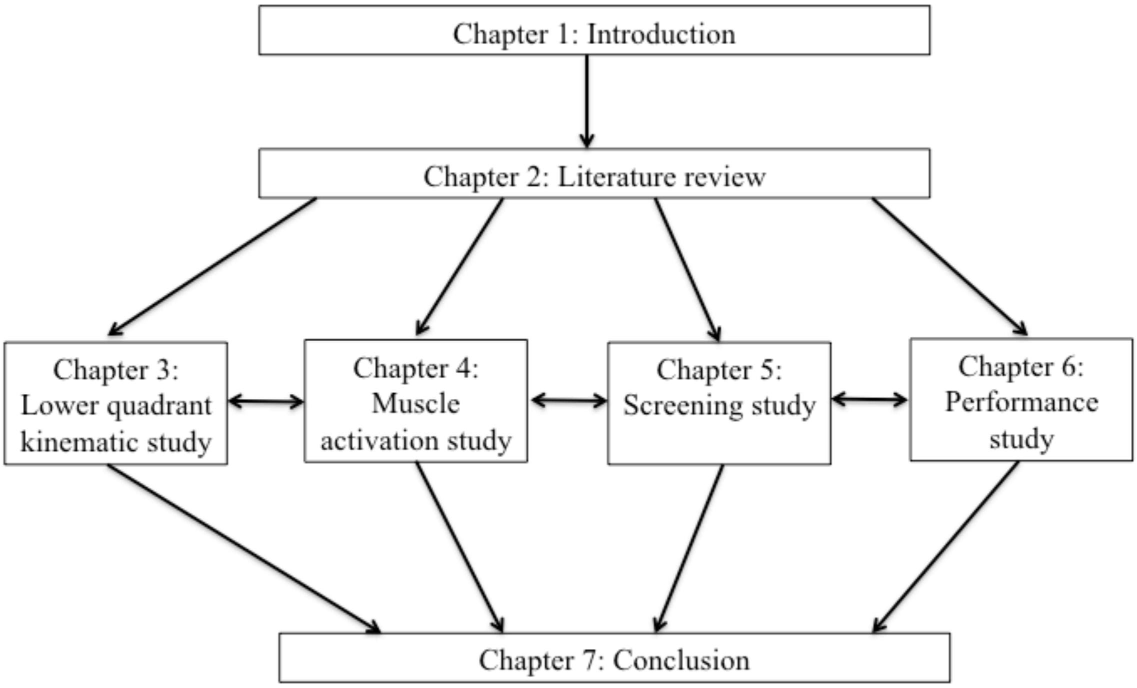

Figure 1.1: Thesis overview

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CHAPTER 2: LITERATURE REVIEW

2.1 Introduction

This narrative literature review aims to provide an overview of lower back pain in young elite male golfers and the factors that may influence lower back injury risk. This will be addressed by expounding the literature regarding: lower back pain, injury prevention in golf, screening for injury risk, the elite golfer drive kinematics, elite golfer muscle activity during the swing, golf injury risk, the role of performance on injury prevention, confounding variables that may influence injury risk.

Golf is a very popular sport with approximately 55 million people playing golf across the world (Farrally et al., 2003). The positive health impact of so many people engaging in physical activity is diminished by the moderate injury incidence seen in golfers (Smith et al., 2018). This is particularly concerning in elite golfers as they sustain significantly more injuries than recreational golfers, the majority of which occur in the lower back (Smith et al., 2018). Young elite male golfers have a new or recurrent lower back pain incidence of 57.1% over a one year period (Smith et al., 2018). This substantial injury incidence in the lower back is concerning. Firstly, as it is occurring in a young golfing population and secondly, because chronic lower back pain is one of the leading causes of years lived with disability (Vos et al., 2012). It is therefore imperative to conduct research aimed at reducing lower back pain incidence and prevalence in young elite golfers.

According to the TRIPP model of sport injury prevention it is essential to establish the aetiology and mechanisms so that interventions can be developed to address the risk factors identified (Emery and Pasanen, 2019). The injury mechanism of lower back pain in young elite male golfers is currently unknown. This problem was highlighted in a recent systematic review conducted by Smith et al. (2018) which concluded that as a result of low quality and heterogeneity of the current evidence, more research is needed to enable evidence based prevention of lower back pain in golfers.

The movement characteristics of the golf swing, individual physical and demographic characteristics as well as volume of play have all been proposed as possible mechanisms of injury in golfers (Smith et al., 2018). This research is focused on identifying the biomechanical (golf swing movement and muscle activity) and musculoskeletal factors associated with injury development in young elite male golfers. This addresses the second step of the TRIPP model of sport injury prevention so that future intervention studies can be designed to address the risk factors identified. South Africa has a large number of young elite golfers, which makes this research relevant in a South African context (Golf RSA, 2021).

2.2 Injury in golf

Golf is a moderate intensity physical activity and playing golf has notable health benefits, which include improved overall physical health, improved mental health and increased life expectancy (Murray et al., 2017). The positive health benefit of playing golf is, however, compromised in elite players by their high rate of injury. Professional golfers sustain significantly more injuries than amateur golfers (Smith et al., 2018) with an injury prevalence of around 88% (Cabri et al., 2009). Professional golfers sustain on average two injuries per year, the majority of which occur in the lower back, followed by the wrist and shoulder (Cabri et al., 2009). Most injuries experienced by elite golfers are overuse-injuries and are related to times of intense practice (Cabri et al., 2009).

Despite significant health related benefits to playing sport, sport has been identified as the leading cause of injury in youth (Emery et al., 2015). Relevant to the current research, the substantial injury incidence in young golfers is particularly concerning as there is an increased risk of developing post-traumatic arthritis as the golfer ages (Emery and Pasanen, 2019). There is evidence to suggest that three to ten years post sports knee trauma, youth will present with more clinical symptoms, reduced knee related quality of life, poorer dynamic balance, weaker knee muscles, higher adiposity and more structural changes on imaging (tenfold increase in MRI-defined osteoarthritis) (Emery and Pasanen, 2019). This concern regarding post injury osteoarthritis development is supported by radiographic studies on golfers that have shown significantly more right-sided osteophyte formation at L3/L4 and facet joint degeneration at L3/L4 and L4/L5 relative to a control group (Sugaya et al., 1999).

2.3 Lower back pain

2.3.1 Lower back pain in the general population

Lower back pain is an extremely prevalent condition that affects people across all age groups (Hartvigsen et al., 2018). The global point prevalence of lower back pain was 7.3%, which implies that 540 million people were affected by lower back pain globally at one time point (Hartvigsen et al., 2018). The biggest increases in disability caused by lower back pain over the last couple of decades, were seen in low and middle income countries (Hoy et al., 2015), the prevalence of which, is now estimated at: 18% in the general population, 31% in the elderly population, and 44% in workers (Jackson et al., 2015). This prevalence in low and middle income countries is likely to increase into the future due to a predicted: i) increase in the ratio of older to younger people; ii) increase in the number of people living in low and middle income countries (current estimates predict that by 2050 3.5 billion people over the age of 40 years will be living in low and middle income countries compared to 645 million in high income countries) iii) increased rate of obesity, which is a strong predictor of lower back pain (Hoy et al., 2015).

The vast majority of patients (90 to 95%) with lower back pain have 'non-specific lower back pain and this category of back pain is the primary focus of this thesis (Hartvigsen et al., 2018, Hall et al., 2021). Although several lumbar structures (such as the intervertebral discs, trunk muscles and facet joints) have been proposed as possible sources of non-specific lower back pain, clinical tests do not reliably attribute pain to any of these structures (Maher et al., 2017). In this category of lower back pain (non-specific) a specific nociceptive source cannot be identified and imaging is not recommended (Hartvigsen et al., 2018, Hall et al., 2021). For this reason participants in this research did not require imaging or further investigations to confirm the diagnosis of lower back pain.

It is important to view lower back pain within the bio-psychosocial model of health (Hartvigsen et al., 2018). Lower back pain can be influenced by biophysical, psychological, social and genetic factors and as well as co-morbidities (Hartvigsen et al., 2018). Biophysical attributes linked to developing lower back pain include alterations in muscle composition, muscle size, and co-ordination (Hartvigsen et al., 2018). Psychological factors such as depression, catastrophising, reduced self-efficacy, anxiety and fear avoidance may negatively impact upon patients with lower back pain (Hartvigsen et al., 2018). Reduced socio-economic status has also been shown to impact upon lower back pain (Hartvigsen et al., 2018). The focus of this research is upon the biophysical attributes of lower back pain in elite golfers. It was outside the scope of this thesis to control for psychological, social and genetic factors in this thesis, but future studies on young elite golfers may seek to address these factors.

2.3.2 Lower back pain in the sporting population

In a systematic review by Trompeter et al. (2017) on lower back pain in sport, combined results of 12 studies showed a lower back pain lifetime prevalence range from one to 94%; and a one year prevalence of range from 24 to 66%. These wide ranges are due to methodological differences between studies on how lower back pain in athletes is defined (Trompeter et al., 2017). Future research should involve a consensus statement regarding the definition of lower back pain in sports participants as well as reliable and validated instruments to measure lower back pain prevalence in sport (Trompeter et al., 2017). In the absence of such a statement or consensus, 'injury' to the lower back in this thesis was defined according to the European guidelines for defining lower back pain (Burton et al., 2006). Lower back pain was defined as 'pain and discomfort that was localised below the costal margin and above the inferior gluteal folds, with or without leg pain' (Burton et al., 2006). The lower back pain did not need to result in limited golf participation.

The relationship between lower back pain and activity has been well researched (Trompeter et al., 2017). A sedentary lifestyle has been associated with increased risk of lower back pain, while on the opposing side of the spectrum, high physical work loads has also been associated with developing lower back pain (Trompeter et al., 2017). Playing sport offers has the potential to both treat lower back pain and to cause lower back injury (Trompeter et al., 2017). It has been proposed that the relationship between lower back pain and activity may follow a U-shaped curve (Trompeter et al., 2017) (Figure 2.1). A cross-sectional study conducted by Heneweer et al. (2009) on 8000 people over the age of 24 years showed a moderate increased risk in lower back pain for both those engaged in a sedentary lifestyle and those involved in strenuous physical activity. In this study engaging in sport for one to two and a half hours was associated with a reduced risk of chronic lower back pain (Heneweer et al., 2009).

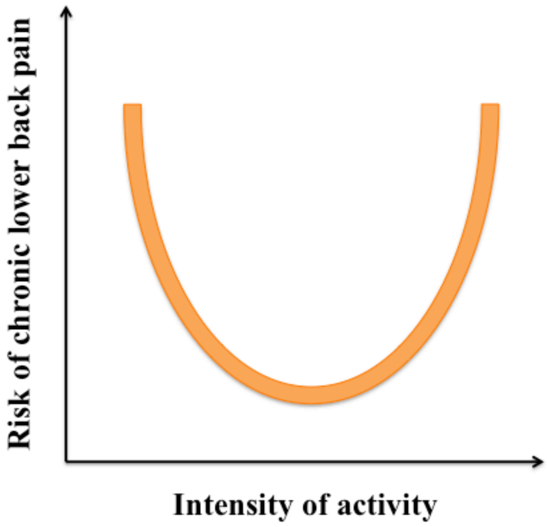

Figure 2.1: U shape curve comparing risk of chronic lower back pain to intensity of activity.

2.3.3 Lower back pain in elite golfers

Lower back pain prevalence in professional golfers ranges from 40 to 58% and is associated with substantial time loss in play and practice (Smith et al., 2018). Fifty-five per cent of golfers report having developed lower back pain in their lifetime (Cabri et al., 2009). These lower back pain injuries commonly include: internal disc disruption, muscle strain and facet joint capsule trauma (Gluck et al., 2008).

Lower back pain in golf has been attributed to the repetitive asymmetrical nature of the golf swing (repetitive strain type injury). This is exacerbated in professional golfers as they practice for longer periods of time and have less swing variability. In chronic repetitive strain type injuries the load does not need to exceed the tissue plasticity threshold. The repeated movement pattern without sufficient recovery time results in permanent tissue deformation which results in chronic repetitive strain type injury. Over time the tissue fatigue can further increase the risk of injury. The actions of the trunk muscles combined with the ground reaction forces produced by the golf swing exert compressive forces on the lumbar spine. This compressive loading of the lumbar spine has been proposed as a possible mechanism of back pain in elite golfers (Edwards et al., 2020).

This is supported by studies that have shown that golfers with lower back pain have larger compressive and lateral shear forces than golfers without lower back pain when performing tasks such as bending or lifting (Edwards et al., 2020). Muscular weakness, low resistance to fatigue and lack of endurance in the abdominal and trunk musculature of golfers has frequently been proposed as possible risk factor for developing golf related lower back pain (Marta et al., 2012).

2.3.4 The effect of lower back pain on muscle recruitment patterns

Changes in lumbar spine muscle recruitment patterns which include; greater co-contraction, redistribution of muscle activity and delayed postural control of deep trunk muscle, has been observed in patients with lower back pain (Devecchi et al., 2021). Van Dieën et al. (2019) proposes that there are two divergent motor control patterns that individuals can adopt after having developed lower back pain: 'tight control' and 'loose control'. The 'tight control' motor adaptation involves increased co-contraction of the trunk musculature which results in reduced lumbar movement, but this improved control comes at the expense of increased spinal loading. The 'loose control' motor adaptation involves reduced muscle activity in order to reduce spinal load, but this comes at the expense of spinal movement control (Van Dieën et al., 2019). Not only does muscle recruitment change after developing lower back pain, muscle recruitment has also been shown to influence the development of lower back pain (Niederer et al., 2018). Exercises which encourage postural control, increased strength, muscle endurance and motor control have been shown to prevent lower back pain (Niederer et al., 2018).

2.4 Injury prevention in golf

In order to reduce injury incidence, Van Mechelen et al. (1992) described a systematic four step process. This framework is the foundation for developing and evaluating sports injury prevention programmes (Emery and Pasanen, 2019).

According to this model step one should involve establishing the extent of the concern by monitoring incidence and severity (Van Mechelen et al., 1992). The high injury incidence in young golfers is particularly concerning and makes a strong argument regarding the incidence and severity of the problem (step 1).

The second step in the Van Mechelen et al. (1992) involves identifying possible risk factors involved. This involves determining if there is a statistical association between injury and the proposed variable. In order to determine if the statistical association should be proposed as a possible cause, it is essential to determine if the statistical association is valid and free from bias (where possible confounding variables should be controlled for) (Meeuwisse, 1994).

Associated factors can be both traumatic (struck by a golf ball or a fall from a golf cart) and non-traumatic (e.g. forearm extensor tendinopathy due to repeated loading). Non-traumatic injuries are the focus of this thesis. Non-traumatic injuries can include both overuse (e.g. repetitive strain injury to the lower back) and acute injuries (e.g. twisting an ankle while walking on the golf course). As the majority of golf injuries are due to the repetitive nature of the golf swing, the focus of this thesis is on overuse injuries (Edwards et al., 2020).

Associated factors can be both intrinsic and extrinsic (Meeuwisse, 1994). Extrinsic factors are those that act upon the golfer such as the weather, golf course conditions, rules and equipment (club and ball factors). Intrinsic factors are the focus of this thesis and these include factors such as the golfer’s movement patterns (biomechanics) and conditioning (Meeuwisse, 1994). Despite the intentional focus on the intrinsic factors in this research, it is important not to see these factors in isolation. There is significant interplay both within intrinsic factors and between intrinsic factors and extrinsic factors (Meeuwisse, 1994) Figure 2.2 presents the multifactorial model of athletic injury proposed by Meeuwisse (1994). E.g. a golfer’s poor conditioning may make him susceptible to injury and this may be compounded if he plays on a very wet golf course. Where possible attributing or confounding variables were accounted for in this research. The following variables were accounted for when presenting findings in this thesis: differences in gender, age, anthropometrics, health status, injury history, training, club performance, ball performance, and competition performance.

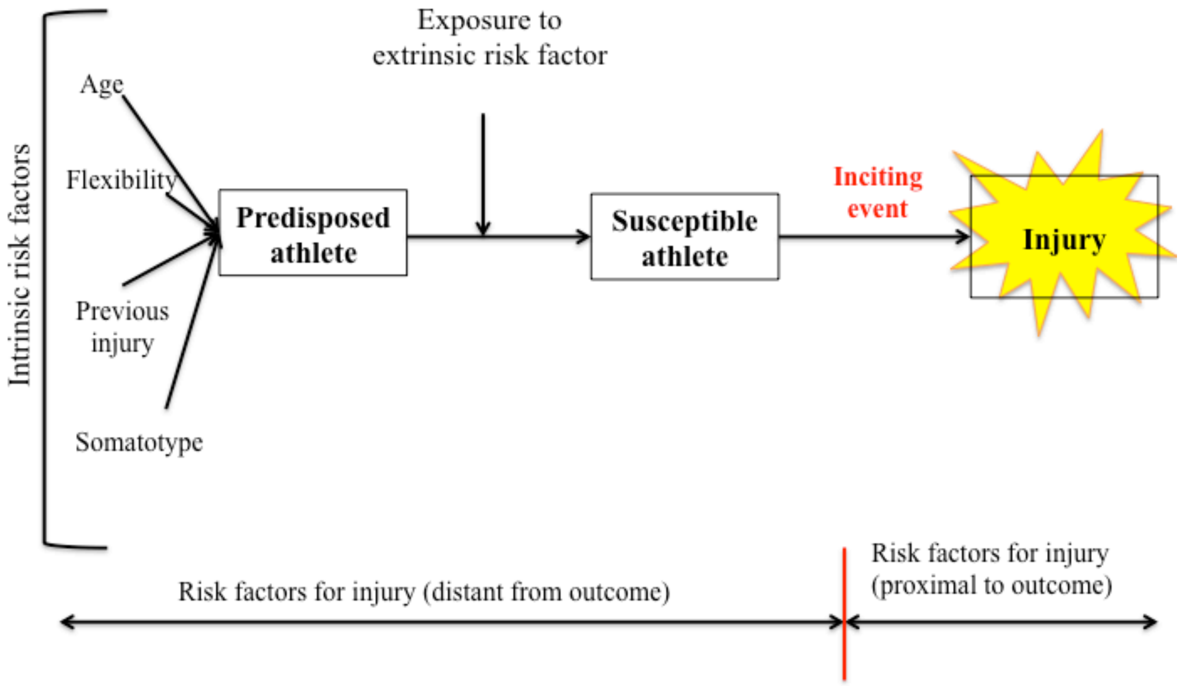

Figure 2.2: Multifactorial model of athletic injury described by Meeuwisse (1994)

The third step in the Van Mechelen et al. (1992) involves introducing interventions aimed at addressing the variable that has been identified as a possible cause. These interventions should ideally be randomised controlled trials that are aimed at addressing the risk factor identified (Emery and Pasanen, 2019). This is not, however, always possible due to ethical challenges or feasibility and as such, less rigorous study methods are often employed including quasi-experimental, cohort and case controlled trials (Emery and Pasanen, 2019).

The fourth step involves monitoring the incidence and severity of the concern identified (Van Mechelen et al., 1992). Only once a factor has been tested can it be confirmed as an injury mechanism or 'cause'. A 'cause' is defined as the 'something that brings about an effect or a result'(Merriam-Webster, 2021). This is different to an 'association' which describes how 'related or connected' two factors are (Merriam-Webster, 2021). An association describes if two factors occur together either more or less often than expected by chance'. Figure 2.3 provides an overview of where this research fits into this model.

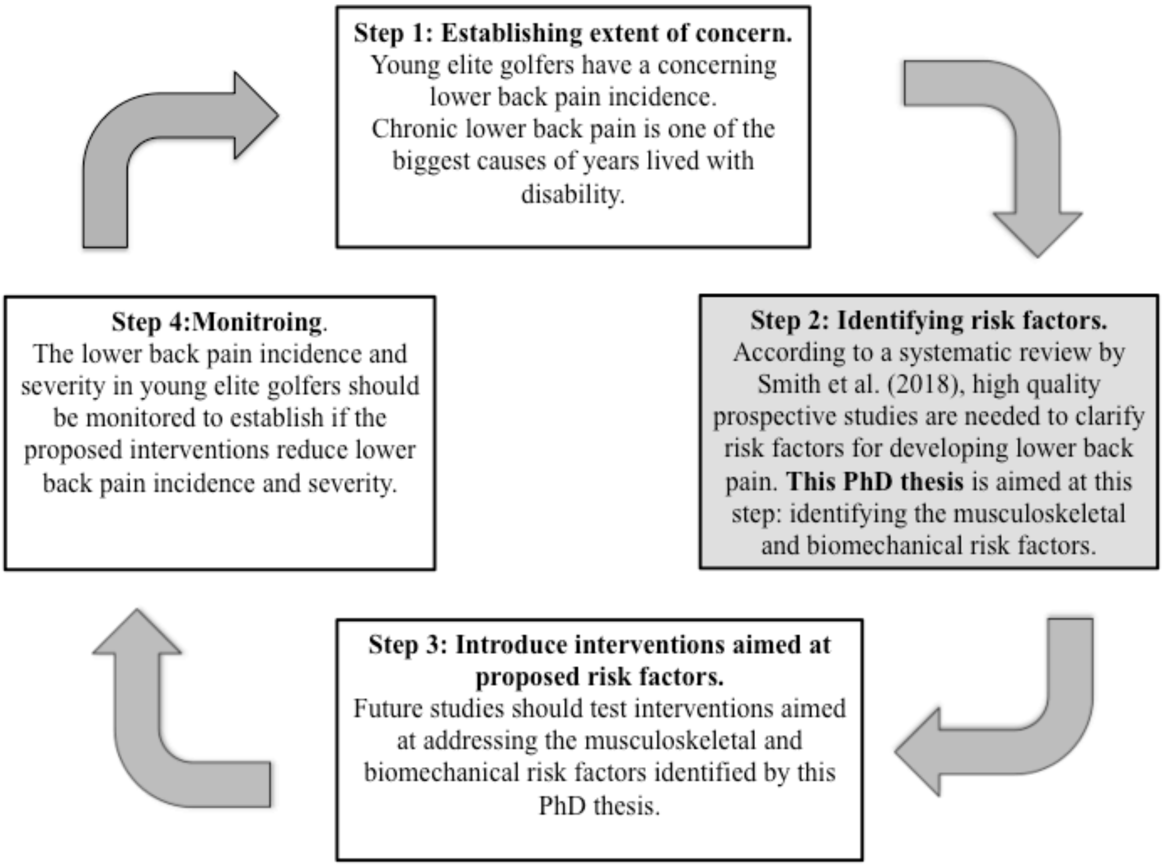

Figure 2.3: The focus of the present thesis (shaded step two) and how it is associated other parts of the model of injury prevention proposed by Van Mechelen et al. (1992)

Once an injury prevention strategy has been established, it is important that the intervention will be adopted and sustained by athletes and their coaches (Emery and Pasanen, 2019). In order to achieve this the TRIPP framework has been proposed (Emery and Pasanen, 2019). This TRIPP framework is an extension of the van Mechelen Model of injury prevention and describes two additional steps (Emery and Pasanen, 2019). Step five is understanding the 'real-world' context into which the intervention is being developed (Emery and Pasanen, 2019). In the target population being addressed in this research, namely young elite male golfers, a significant concern is the impact that an injury intervention would have on the golfers' competition performance. In order to address this concern, one of the studies conducted in this research was focused on the factors that influence performance. If a factor is associated with injury, but essential for performance the 'real-life' uptake of the intervention addressing the factor will be low. If a factor is associated with injury, but the performance element of the factor is unknown, once again the ‘real-life’ uptake of the intervention addressing the factor will be low especially if it involves swing modification. Golfers are reluctant to alter swing biomechanics as this may affect swing consistency and competition performance. If a factor is associated with injury, but has no influence on performance, the ‘real-life’ uptake of the intervention addressing the factor may be improved.

Step six involves evaluating the intervention in the ‘real-world’ context (Emery and Pasanen, 2019). The RE-AIM (Reach, Efficacy, Adoption, Implementation, Maintenance) framework can be used to monitor a ‘real-life’ intervention (Emery and Pasanen, 2019). This includes monitoring: i) if the target population is being reached; ii) the efficacy and effectiveness of the intervention programme; iii) if the intervention is being adopted by the target staff, settings and institutions; iv) if the programme is being implemented consistently and cost effectively; v) if the intervention effects are being maintained over time (Emery and Pasanen, 2019). Figure 2.4 presents an overview of the TRIPP model of injury prevention and where this research falls within this model.

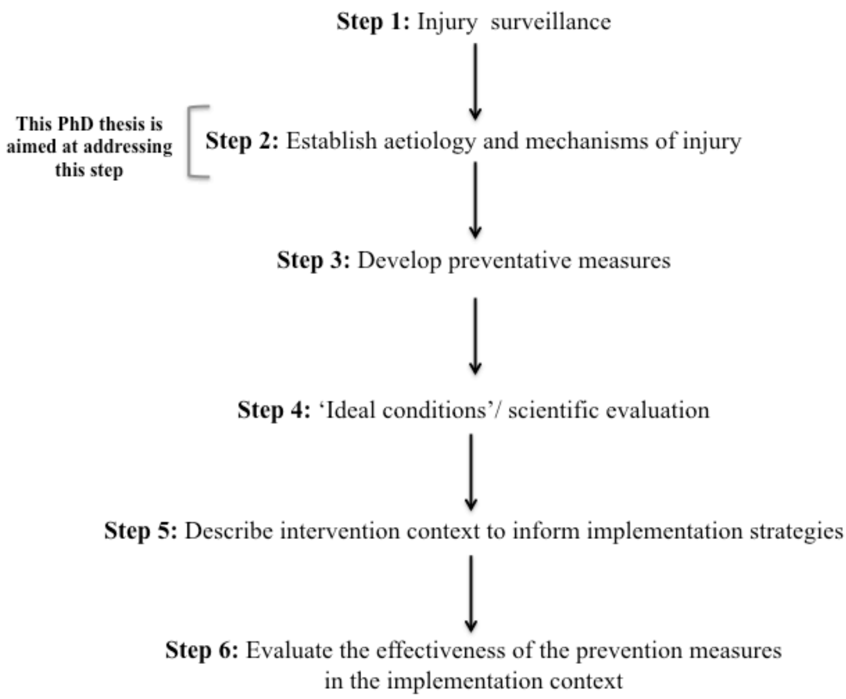

Figure 2.4: Overview of the TRIPP model of injury prevention and where this research is focused

Prevention strategies can be focused on training strategies, policy modification, sport rule changes or equipment recommendations (Emery and Pasanen, 2019). This research is focused on identifying musculoskeletal and biomechanical factors associated with injury so that future researchers can implement training modifications addressing the musculoskeletal and biomechanical injury associations identified.

Some may argue that training modifications may not alter injury prevalence as many injuries can be attributed to the nature of the sport (e.g. one sided repetitive nature of the golf swing). This view point is not, however, supported by the literature. A combined analysis of seven randomised controlled trials on young athletes in various different sports, showed that neuromuscular training strategies, which typically involve exercise aimed at improving balance, strength, agility; are able to reduce lower extremity risk by 35% (Emery and Pasanen, 2019). A systematic review by Lauersen et al. (2014) across various ages and sports on the effectiveness of exercise interventions in preventing sports related injuries found strength training was able to reduce sports injury by one third and that overuse injuries could be halved.

2.5 Screening for injury risk

Musculoskeletal screening has been proposed as possible means of reducing lower back pain in elite golfers and forms part of the pre-participation assessment that has been mandated by the International Olympic Committee (Ljungqvist et al., 2009). According to the Wilson-Junger criteria screening programmes need to be assessed using the following criteria: i) the condition being screened should be an important health problem; ii) the problem can be detected at an early stage; iii) there is a greater benefit to early treatment than treatment at a later stage; iv) there is a suitable test to detect the condition at an early stage (Wilson and Jungner, 1968).

It is easy to satisfy criteria one; lower back pain in elite golf is a significant health problem (Smith et al., 2018). Criteria two and four are more difficult to satisfy. Typically, when screening for disease there is a fairly clear distinction between those that have the underlying disease versus those that do not have the disease (detection of a gene or a virus) (Bahr, 2016).

A screening tests should ideally have both high sensitivity (able to identify all those at risk) and specificity (able to exclude those who are not at risk of developing injury). In sports injury screening there is a large overlap between healthy and 'at risk' athletes in terms of risk factors (e.g. trunk muscle endurance), which makes it more difficult to determine a cut-off point in the test to discriminate between athletes at risk and those not at risk. This large overlap has an impact on both the specificity and sensitivity of musculoskeletal screening tests. If the cut-off point is set too high (e.g. golfers are identified at risk if they are unable to hold a plank for more than 10 min) then screening sensitivity may be compromised so that not all golfers at risk are identified. If the screening point is set too low (e.g. golfers are identified at risk if they are unable to hold a plank for more than 1 min) then specificity may be impacted which would result in golfers not at risk of injury taking part in intervention programmes that are not suitable for them. This is not cost-effective and it may take up valuable training time, which may impact on performance, which is the golfers primary goal (Bahr, 2016).

According to a systematic review by Bahr (2016) there are three essential steps required to develop and validate a screening tool. The first step is to identify the strength of risk factor association with injury development. The second step in developing an injury screening tool involves screening golfers with the same test, but in this second scenario, a predetermined cut-off score is used to separate golfers at risk of injury from those not at risk. The golfers should then be monitored for injury over a set period of time. Step three involves conducting a randomised controlled trial in which athletes at risk are identified using the screening tool and then randomly put into a control or a treatment group. The treatment group is given an intervention aimed at addressing the risk factor identified by the tool. The golfer should then be monitored and if injury incidence reduces in the treatment group, the screening tool can be validated for that patient group. Figure 2.5 provides an overview of how this research fits into the screening model. For a more detailed discussion of the screening tools used in this research see chapter 5. There is on going debate as to the usefulness of screening in identifying athletes at risk of injury (Bahr, 2016).

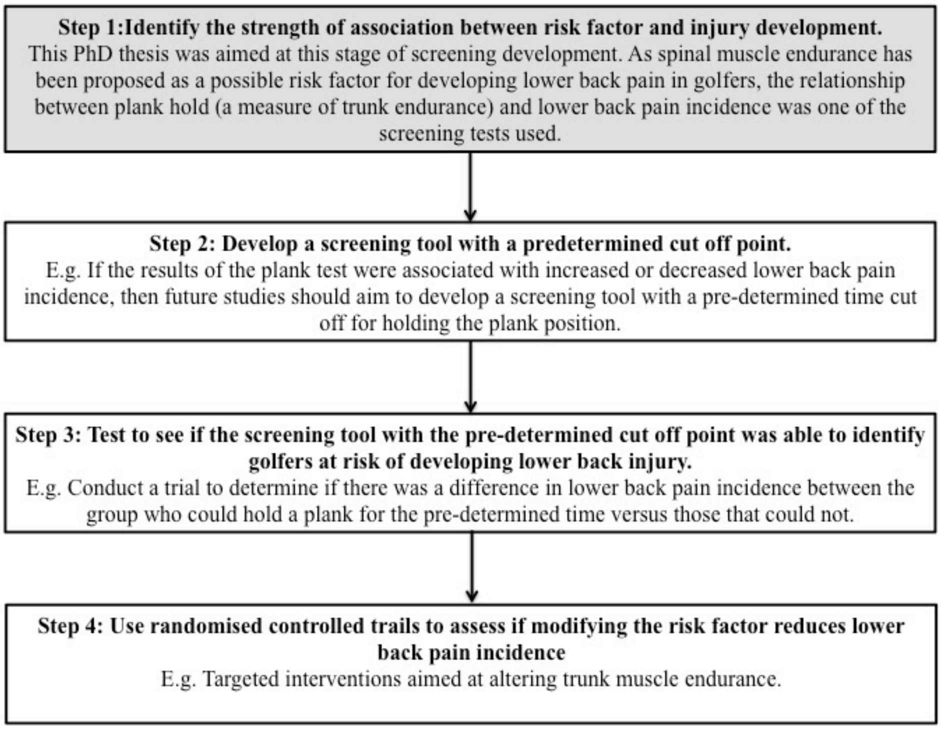

Figure 2.5: How this research fits into the screening model described by Bahr (2016)

2.6 Injury definition

In order to screen for injury, a clear definition of injury is needed. Best practice involves using the definition of injury provided by the sports specific injury consensus statement as this enables comparisons between studies and helps to inform golfers, medical professionals, coaches and policy makers as to the typical injury pattern and injury risk across the sport (Murray et al., 2020). When the studies presented in this thesis were conducted, the 'International consensus statement: methods for recording and reporting of epidemiological data on injury and illnesses in golf,' had not been published (Murray et al., 2020).

In the absence of such a statement, injury to the lower back was defined in this research according to the European guidelines as 'pain and discomfort that was localised below the costal margin and above the inferior gluteal folds, with or without leg pain' (Burton et al., 2006). The lower back pain was self-reported and did not need to limit golf participation or score above a certain pain level to be considered as an injury. No imaging was necessary to confirm the lower back injury. This is in line with the evidence that shows that acute lower back pain is typically self-limiting and does not require imagining unless the patient presents with: radicular symptoms that does not respond to conservative treatment, myelopathy, neurogenic claudication or signs of serious pathology (‘red flags’) (Rao et al., 2018, Andersen, 2011). This guidance is echoed in the Nice Guidelines for patients 16 years or older which states that medical professionals should not ‘routinely offer imagining in non-specialist setting for people with lower back pain with or without sciatica’(National Institute for Health and Care Excellence (NICE), 2016). Golfers in this research were, however, encouraged to attend a free physiotherapy assessment. This did not, however, prove to be a practical solution as there was a high rate of non-attendance to these assessments. Reasons given for non-attendance were: travelling to tournaments and intensive training schedule. Golf specific exposure was recorded in the training log and the golfers were classified according to their performance level.

In 2020 an international consensus statement was developed for the recording and reporting of epidemiological data on injuries and illness in golf (Murray et al., 2020). A table of their recommendations has been provided below. It is unfortunate that these recommendations were not available before the research in this thesis was conducted, as I would have followed their recommendations.

Table 2.1: Table of International consensus statement: methods for recording and reporting of epidemiological data on injury and illnesses in golf recommendations and those contained in this thesis

Recommendation	Categories	Included in the research presented in this thesis
Definition of injury	1. Area 2. Handedness 3. Side of injury	The area of injury was defined. The handedness of the golfers was recorded. The side of the injury was not recorded. It could, however, be argued that the lower back does not have a side as it is one mechanical structure.
Mode of onset	4. Acute 5. Repetitive: • Sudden • Gradual	Not included in this research.
Mechanism of injury	1. No identifiable single event 2. Non-contact trauma. 3. Direct contact with an object 4. Direct contact with the ground 5. Indirect contact with an object. 6. Indirect contact with the ground.	The mechanism of injury was included in the prior lower back pain baseline demographics questionnaire. Mechanism of injury was not, however, captured during the six-month monitoring period.
New or subsequent injuries	1. New 2. Subsequent 3. Recurrent 4. Exacerbation	Prior lower back pain in the six months prior to inclusion in the study was included in the analysis.
Severity of injury or illness	1. Number of days the player was unable to play or train. 2. Number of days from the onset of injury until full recovery.	The number of days the golfer was unable to play or train was included in the baseline injury assessment. The number of weeks the player had lower back pain was included in the injury log.
Recording of golf-specific exposure	1. Golf competition 2. Golf practice: • On course practice • Driving range • Putting/short game 3. Training: • Fitness training. • Weight training • Cardiovascular training. • Stretching or mobility training. • Proprioception exercises. • Warm up or cool downs	This information was included in the golfers training log.
Performance level	1. Elite: Professional players competing on tour or amateurs competing in international/national amateur championships. 2. Sub-elite: PGA teaching professionals, amateurs competing in regional/ county/state tournaments or with handicap ≤5 3. Recreational: Handicap above 5	In the absence of the consensus statement, the golfers in this research were categorised as 'young elite golfers'. The participants needed to be recognised as an elite player by the academy's coaches, male and between the ages of 16 and 30 years old. Junior golfers (under 18 years old) were included in the study, therefore, a handicap cut off was not applied. Many of the golfers played in amateur competitions and had handicaps below 5.

2.7 The elite golfer drive kinematics

The movement characteristics of the golf swing have been proposed as one of the possible mechanisms of injury in golfers (Smith et al., 2018). In order to understand the possible movement injury associations, a closer look at the swing of elite golfers is required, particularly the golf drive, during which large muscle force and torque is produced in order to generate maximum club head speed at the point of impact so as to maximise golf drive distance (Hume et al., 2005, Hellström, 2009).

Golf drive distance is affected by controlling club head speed and clubface angle at impact (Hellström, 2009). Golf drive distance is directly proportional to 'linear club head velocity at the point of impact' (where the golf club strikes the golf ball) (Hume et al., 2005). Linear club head speed is a function of the angular club head velocity and the length of the lever created by the club and the arm at the point of impact (Hume et al., 2005). Therefore in order to increase golf drive distance, the golfer must aim to increase both angular club head speed and the length of the club and arm lever at impact. The arm and club lever can be maximised by holding the club at the upper most part of the handle and by extending the elbow at impact (Hume et al., 2005). Angular velocity can be influenced by ground reaction forces (GRF) (Edwards et al., 2020), body weight transfer, the principle of sequential summation of forces (Tinmark et al., 2010) and the use of the stretch shortening cycle (Hume et al., 2005).

The golf swing is typically divided into four phases: set-up (Figure 2.6), backswing (Figure 2.7), downswing (Figure 2.8) and follow through (Figure 2.9) (Edwards et al., 2020). For the sake of clarity the following explanation of the golf swing phases will apply to a right handed golfer (these explanations can be reversed in the case of a left handed male player).

In the set up position 50 to 60% of the golfers weight should be on the right foot, the feet should be shoulder width apart, the knees should be flexed to 20 to 25 degrees, the trunk should be flexed to 45 degrees at the hips and there should be a lateral shoulder tilt of 16 degrees towards the right (Figure 2.6) (Hume et al., 2005, Cole and Grimshaw, 2016). The optimal set up position should enable the golfer to align properly with the target, have good static balance, dynamic balance, a good golf posture and the correct grip of his club (Hume et al., 2005, Cole and Grimshaw, 2016). Golfers that have excessive curvature in the lower back at set up in which the golfer sticks their tail bone out too much are described as having an 'S-posture' (Titleist Performance Institute, 2021h) (see chapter 6 for detailed explanations of swing characteristics). Golfers whose shoulders are slumped forward and their thoracic spine appears rounded at set-up are described as having a 'C-posture' (Titleist Performance Institute, 2021h).

Figure 2.6: Set up position of golf swing

During the backswing what is referred to as a 'one-piece takeaway' should occur, during which the triangle formed by the chest and arms should be kept for the first 40 to 60 cm while the club travels away from the ball (Hume et al., 2005). The shoulders should then continue to rotate whilst the arms move upwards (Hume et al., 2005). During this time, the hips will move into a more rotated position (Hume et al., 2005). As the hands reach the level of the hips, the right shoulder should abduct and externally rotate while the right elbow flexes (Hume et al., 2005, Cole and Grimshaw, 2016). During this time the left shoulder should adduct and internally rotate (Hume et al., 2005, Cole and Grimshaw, 2016).

At the top of the backswing the right shoulder should be in 75-90 degrees abduction and approximately 90 degrees external rotation. The left shoulder is internally rotated and horizontally adducted across the chest, the left elbow is extended and the wrist and hands are held in a cocked position. There is a lot of controversy as to the amount of weight shift that occurs during the optimal backswing. Typically at the top of the backswing 40% of body weight is on the left leg (Hume et al., 2005) (Figure 2.7).

Elite player's backswing is usually less than one second (approx. 00.82 sec) (Hume et al., 2005, Loock et al., 2013). The ideal backswing positions the golfer so that the hub centre and club head are aligned in manner enables a powerful and accurate downswing (Hume et al., 2005, Loock et al., 2013). A good backswing provides a foundation for the downswing's kinetic chain and stretches the structures involved in the stretch shortening cycle (Hume et al., 2005). The structures involved in the stretch shortening cycle are responsible for power generation during the downswing (Hume et al., 2005).

The primary difference between the classic and the modern golf swing is the considerable emphasis placed on restricting pelvic rotation during the back swing (40 to 50 degrees) while at the same time encouraging the thorax to rotate to almost double that range (90 to 100 degrees) (Cole and Grimshaw, 2016). The difference between the torso and the pelvic rotation at the top of the golf swing is termed the X-factor (Hume et al., 2005). A study by Meister et al. (2011) on 10 professional and five amateur golfers, showed peak X-factor was highly predictive of club head speed at impact (more so than peak upper-torso rotation or peak pelvic rotation on their own). A review article by Cole and Grimshaw (2016), however, found conflicting evidence regarding the claim that increasing the X-factor and subsequent stretch shortening cycle results in increased golf drive distance.

Golfers who play with excessive lower body lateral shift away from the target during the backswing are described as having a 'sway' during their swing (Titleist Performance Institute, 2021h). Golfers with excessive trunk lateral flexion towards the target during the backswing are described as having a 'reverse spine angle' during the swing (Titleist Performance Institute, 2021h).

Figure 2.7: Top of the backswing

The downswing is the most important determinant of the swing performance (Loock et al., 2013). The downswing can be further divided into early downswing (top of the backswing (Figure 2.7) to the point where the club is parallel to the ground) and the acceleration phase (from the point where the club is parallel to the ground to impact (Figure 2.8)) (Marta et al., 2016). The downswing was initially described as a planar double pendulum that transmits maximum force onto the ball at impact (Hume et al., 2005, Cole and Grimshaw, 2016). The initial modelling had two segments which included arms as the upper segment and the club as the lower segment. This model was challenged to include a third segment which included the trunk (Cole and Grimshaw, 2016). An average downswing in an elite golfer lasts 0.23 seconds (Hume et al., 2005). Elite golfers initiate the downswing with reversal of the of pelvic rotation followed by upper torso rotation (Meister et al., 2011).

During the downswing the hub should be maintained within the base of support. In order to maximise energy transfer and precision the downswing plane should be slightly less shallow than the backswing plane. During the downswing the left shoulder externally rotates while moving towards the midline. During this time the right shoulder adducts and internally rotates while the right elbow extends. The wrists should remain in the cocked position until just before impact so as to maximise the angular velocity of the club head (Hume et al., 2005).

In order to increase transfer of energy from one body segment to the next (summation of speed principle) the kinetic chain should be initiated from proximal to distal: lower limb, followed by trunk and shoulders and lastly wrists and hands (Hume et al., 2005, Cole and Grimshaw, 2016, Edwards et al., 2020). The purpose of proximal to distal sequencing is to increase the velocity of the distal segment to a greater velocity than would have been possible using individual muscle contractions (Edwards et al., 2020). Professional golfers have average angular velocities of 498 degrees per second for the hip, 723 degrees per second for the shoulder, 1165 degrees per second for the arm and 2090 degrees per second for the club head (Hume et al., 2005). At the point of impact (Figure 2.8): spine flexion is 34 degrees (compared to 45 degrees at set up); spine lateral flexion away from the target is 28 degrees (compared to 16 degrees at set up) (Hume et al., 2005). During the downswing the left leg experiences a compression force of 80% bodyweight (Hume et al., 2005). Golfers with excessive lower body lateral shift towards the target during the downswing are described as having a ‘slide’ during the swing (Titleist Performance Institute, 2021h). Golfers who play with forward thrust of the hips and legs towards the ball during the downswing are described as have ‘early extension’ during the swing (Titleist Performance Institute, 2021h).

Figure 2.8: Impact position

During the follow through the hands and wrists continue to follow the swing plane while the left shoulder externally rotates and abducts and the right shoulder internally rotates and adducts. When the hands reach shoulder height the elbows flex so as decelerate the speed of the trunk while maintaining postural control. During this deceleration phase the trunk and the hips rotate towards the left (left hip internal rotation) and the left ankle supinates. At the end of the follow through (Figure 2.9) the golfer should be trunk should be facing the target in a well balanced position. He should be in trunk extension and lateral flexion, his hands should be behind his left ear and his head should be rotated towards the left. In order to maximise ground reaction force during the swing elite golfers should use their body mass to ensure that their legs are pushed down into the ground. In order to maximise transference of the ground reaction force, the golfer should aim to keep his body as rigid as possible. During the downswing phase weight is transferred from the back leg (right leg) to the front leg (left leg). Elite golfers transfer more of their weight at a faster rate during the downswing than recreational golfers (Hume et al., 2005).

Golfers who play with reduced hip rotation during the swing are described as having ‘restricted hip rotation’ (Titleist Performance Institute, 2021h).

Figure 2.9: End of follow through

2.8 The elite golfer drive kinematics and lower back pain risk

The repetitive and asymmetrical nature of the golf swing; high spinal segmental angular velocities; large compressive, torsional and shearing spinal loads during the swing, have been proposed as possible mechanisms of lower back pain in elite golfers (Smith et al., 2018, Edwards et al., 2020).

Increased X-factor and X-factor stretch seen in elite golfers may increase golf drive distance, but in order to perform these movements, elite golfers require excellent spinal mobility and spinal endurance to repeated stretch (Smith et al., 2018). Repeated over stretching of the spinal muscles has been proposed as a possible mechanism of lower back pain in elite golfers. Anecdotal evidence postulates that up to 41% of lower back injury in golf occurs during the impact (Figure 2.8) and early follow through (Cole and Grimshaw, 2016). This is due to the high peaks in lateral flexion, anteropostero shear forces and compressive loads experienced by the spine during this time (Cole and Grimshaw, 2016).

The crunch factor and trunk hyperextension during the follow through have also been proposed as possible mechanisms of lower back pain in elite golfers. This is due to the proposed increase in compressive and shearing forces that the spine is likely to experience during these movements (Smith et al., 2018).

Previous studies have shown that golfers with lower back pain have: increased trunk flexion at set up; use more lateral flexion during the backswing; reduced spinal rotation during the swing and reduced trunk flexion velocity compared to golfers without lower back pain (Lindsay and Horton, 2002). There is, however, an overall lack of consensus and evidence regarding swing mechanics and lower back pain risk (Smith et al., 2018). For a detailed discussion on the kinematics associated with developing lower back pain see chapter 3.

2.9 The elite golfer drive muscle activity

The muscle activation patterns of the golf swing have been proposed a possible mechanism of lower back pain in elite golfers (Smith et al., 2018). In order to understand this mechanism, a closer look at the muscle activation patterns during the golf swing is required.

At set up there are low levels of activity bilaterally in *erector spinae*, as well as *biceps femoris, upper trapezius* and *anterior deltoid* on the left side. As this position is primarily a static posture, the other muscles of the trunk and lower extremity are largely inactive (Cole and Grimshaw, 2016).

There is debate as to which region initiates the backswing. Some authors propose that the pelvic muscles initiate the back swing followed by the torso. Others propose that the backswing involves simultaneous activation of the trunk and arm muscles to enable rotation (Loock et al., 2013).

During initiation of the backswing the left *external oblique* and the right *internal oblique* muscles facilitate the rotation of the trunk away from the target. During this phase the left *rectus abdominis* and the paraspinal muscles contract thereby producing left sided lateral trunk flexion seen in the mid to late stages of the backswing. The paraspinal muscles also work to counter act the flexor moment created by the abdominal muscles. During early backswing the right *erector spinae* muscle activation increases and fires simultaneously with the left *erector spinae*. As the trunk rotates away from the target, there is an increase in activity in the right *trapezius*, *rhomboid* and *levator scapulae* muscles in order to facilitate right scapula retraction and elevation. At the same time there is an increased muscle activation of the left *anterior deltoid* and left *pectoralis major* muscles which results in left shoulder internal rotation and left scapula protraction. Throughout the backswing there are mid levels of *biceps femoris* and *semimembranosus* muscle activation as they resist knee extension (Cole and Grimshaw, 2016).

The upper torso muscles which includes the *pectoralis, deltoid, trapezius,* and the *rhomboids* are highly active during the backswing (Loock et al., 2013). The *erector spinae, abdominal oblique, latissimus dorsi* and *gluteals* have been shown to be active throughout the backswing (Loock et al., 2013). *Biceps femoris, gluteus maximus, semitendinosus, rectus femoris, vastus medialis and lateralis, peroneus longus, tibialis anterior, gastrocnemius medialis and lateralis* have been shown to have low to medium activation (3-24 % MVC) during the backswing (Marta et al., 2016).

The downswing is initiated by a strong contraction of the right *gluteus maximus,* right *gluteus medius*, right *semimembranosus* and right *biceps femoris* which results in extension of the right hip and initiation of the anti-clockwise rotation of the pelvis. At the same time this is augmented by a simultaneous contraction of the left *adductor magnus* which assists pelvic rotation. During the very early stage of the backswing *erector spinae* activity is increased on the right side and this is activation is said to counteract the gravitational and rotational forces acting on the body. Soon after the downswing is started the left *latissimus dorsi* activates in order to bring the left arm down while the right *external oblique* and the *left internal oblique* act to rotate the trunk. At this time there is a maximal contraction of the *abdominal oblique* muscles and bilateral contraction of the *erector spinae* muscles which act to protect the lower back by preventing excessive lumbar flexion. During the early phases of the downswing the left *rhomboids,* left *levator scapulae*, and left *trapezius* muscles act to elevate and retract the left scapula. At the same time the right *pectoralis major* and right *serratus anterior* act to protract and internally rotate the right shoulder. The wrist and forearm movements during the downswing is enabled by bilateral activation of the *flexor carpi ulnaris* and the *flexor carpi radialis*, which act to overcome the inertia of the club and accelerate the club downwards and forwards towards the ball. As the club approaches impact both *pectoralis major* muscles reach their maximum activation which may act to stiffen the body to prepare for maximum ground reaction force transmission at impact. This maximum contraction of the left *pectoralis major* muscle just before impact may also help control retraction and external rotation of the left shoulder (Cole and Grimshaw, 2016).

The *erector spinae, gluteus maximus* and *abdominals* have been shown to be active throughout the downswing (Loock et al., 2013). During the follow through the left and right *gastrocnemius*, right *biceps femoris*, right *semitendinosis*, and right *gluteus maximus* have been shown to reach a peak with medium to high levels of activity (23–94% MVC) (Marta et al., 2016). During the acceleration phase of the downswing the left *gluteus maximus,* left *semitendinosus,* right *vastus medialis* and right *vastus medialis* have been shown to peak with medium to high muscle activity levels (21-44% MVC) (Marta et al., 2016). The thoracic and lumbar *erector spinae* muscles have also been shown to peak during the forward swing and acceleration phase of the downswing (Sorbie et al., 2018). The *latissimus dorsi, pectoralis major* and *subscapularis* muscles have been shown to be the most active muscles acting on the shoulder during the swing (Marta et al., 2012). Their peak muscle activation occurs during the acceleration phase of the downswing (Marta et al., 2012).

From impact to late follow through the left *gluteus medius* exhibits a low and steady level of muscle activity which assists with lateral movement of the hips during this phase. The *gluteus medius* on the right side has high activity levels as it acts to abduct and extend the right hip and enable pelvic rotation during the early phase of follow through. As the golfer heads towards late follow through the right *gluteus medius* muscle activation decreases. Throughout the follow through phase the left *biceps femoris*, left *vastus lateralis* muscles activate as they act to provide a good foundation on which to complete the anti-clockwise rotation of the body. Following impact the right *external oblique* and left *internal oblique* demonstrate a mild reduction in activity as they continue to act to rotate the trunk. The *erector spinae* muscle activity deceases soon after impact, with the left side displaying higher levels of muscle activation than the right. Similar to the *abdominal oblique* muscles the left *rhomboids*, left *levator scapulae*, left *trapezius*, right *serratus anterior*, right *pectoralis major* only have slightly reduced activity levels during early follow through. These muscles (the left *levator scapulae*, left *trapezius*, left *rhomboids*, right *serratus anterior,* right *pectoralis major*,) do, however, go on to decrease during late follow through. Similarly the bilateral *erector spinae* and left *external oblique* muscle activity continues to lower during late follow through, but the left *external oblique* remains active during late follow through (at similar levels to those seen at early follow through). The left *external oblique* has high muscle activity during late follow through and this may aid trunk deceleration (Cole and Grimshaw, 2016).

Throughout the follow through *semitendinosus, biceps femoris, rectus femoris, gluteus* *maximus, vastus medialis and lateralis, peroneus longus, tibialis anterior, gastrocnemius medialis and lateralis* have been shown to have decreasing activity (5-60% MCV). The *subscapularis* muscles have been shown to have high levels of activation on both sides during the follow through. The *anterior deltoid, posterior deltoid, middle deltoid, infraspinatus* and *supraspinatus* and have been shown to have weak to moderate activation throughout the various phases of the golf swing (Marta et al., 2012).

2.10 The relationship between muscle activity and developing lower back pain in elite golfers

2.10.1 The relationship between the elite golf drive muscle activity and developing lower back pain

Lumbar musculature is responsible for much of the force generation for the swing action and for protecting the lumbar structure against injury (Gluck et al., 2008). Muscle activation characteristics have been examined in golfers with lower back pain versus golfers without lower back pain (Smith et al., 2018).

Horton et al. (2001) showed no difference in the abdominal muscle activity between golfers who currently had lower back pain and those who did not have lower back pain. They did, however, observe a delay in lead *external oblique* muscle onset at the start of the backswing in golfers with lower back pain (Horton et al., 2001). In a similar study Cole and Grimshaw (2008b) showed that golfers with lower back pain had reduced *erector spinae* activity at impact and increased *external oblique* activity at the top of the backswing. In another study published by Cole and Grimshaw (2008a) it was shown that that *erector spinae* muscle activation prior to initiation of the backswing occurred earlier in participants with back pain. They proposed that this earlier activation strategy may compensate for dysfunctional deep spinal stabilisers.

Pooled analysis of the studies conducted by Horton et al. (2001) and Cole and Grimshaw (2008a) showed no relationship between lower back pain and the timing of lead side external oblique onset at the beginning of the backswing (Smith et al., 2018). These studies provide valuable insight into the differences between golfers with lower back pain and those without lower back pain. They do not, however, inform if these changes in muscle recruitment occurred as a result of lower back pain or if they were associated factors in the development of lower back pain. For a more detailed discussion on the role of muscle activation during the golf swing on developing lower back pain see chapter 4.

2.10.2 The relationship between general muscle function and developing lower back pain in elite golfers

Studies have also been performed on general muscle function (while not engaged in swinging the golf club), which compared golfers with lower back pain to those without lower back pain.

These studies conducted on general muscle function showed that trunk endurance may play a role in developing lower back pain in golfers. One such study conducted by Evans and Oldreive (2000) on twenty male golfers aged between 20-44 years compared static *transverse abdominis* activation endurance of golfers with a history of lower back pain to golfers without a history of lower back pain. They demonstrated that golfers with a history of lower back pain had reduced *transverse abdominis* static muscle contraction endurance compared to golfers without a history of lower back pain. (Evans and Oldreive, 2000). Lindsay and Horton (2006) similarly conducted a study comparing 40 healthy controls to 32 elite healthy golfers and seven golfers with lower back pain (mean age of 33years). They found that the golfers with chronic lower back pain had less trunk endurance in the non-dominant direction compared to either healthy control group (Lindsay and Horton, 2006). In addition, Suter and Lindsay (2001) conducted a study comparing 25 golfers with lower back pain (elite and professional golfers with a mean age of 37 years) to 16 healthy controls who were involved in recreational activities, but not elite or professional golf. They found that in the group of golfers with chronic lower back pain, there was association between reduced lower back endurance and knee extensor inhibition (Suter and Lindsay, 2001). This was not, however, the case with the healthy non-golfer control group (Suter and Lindsay, 2001).

The relationship between poor trunk endurance and developing lower back pain has also been observed in the non-golfing population. A study conducted by Ebrahimi et al. (2005) on 200 participants comparing trunk endurance (measured using the prone double straight leg raise) in participants with lower back pain to those without lower back pain, showed that there was a relationship between poor trunk endurance and lower back pain. Another study conducted by (Lopes et al., 2020) on 545 Naval Cadets showed that reduced trunk endurance measured using the prone plank test was associated with overuse injury, which included lower back pain.

The above mentioned studies demonstrate an association between trunk muscle endurance and lower back pain in golfers. It is not, however, possible to determine if the reduced trunk muscle activity was as a result of lower back pain or a precursor to developing lower back pain. If one adopts the pain adaption model of pain, the pain experienced by the golfer in their lower back, may have resulted in a reduction in muscle activation of the agonists (Hamberg-van Reenen et al., 2007). Proponents of this view state that the reason for this reduction in agonist muscle activity is to reduce movement velocity and movement excursion (Hamberg-van Reenen et al., 2007). On the other hand, it is possible to hypothesise that weakness or poor endurance in the trunk muscles may have predisposed the golfer to lower back pain (Lee et al., 1999). Therefore the mechanism of injury is still unknown. In order to establish if the reduced trunk muscle activity precluded the development of lower back pain, measurement of trunk muscle activity should ideally take place before the lower back pain occurs. For this reason all muscle activity measurements were taken prior to the golfers developing lower back pain. Golfers were then monitored for lower back pain incidence.

2.11 Performance

If lower back pain injury associations are identified, and interventions are designed to address the modifiable factors, an essential consideration is the impact that any intervention may have on elite golfer performance. Therefore performance parameters were measured in the golfers that took part in the four studies contained in this thesis.

The golf drive has often been cited as a strong determinant of overall golf performance in professional golfers (Alexander and Kern, 2005). The importance of the golf drive can be seen in the PGA (Professional Golfer's Association) Tour results with the top ten drivers in the PGA earning 30% more money than the top ten putters (Alexander and Kern, 2005). Increasing golf drive distance has been a focus of golfers and coaches as golf courses have been built longer and old courses have been lengthened thereby favouring golfers with long drives (Alexander and Kern, 2005). Improvements in club design have also seen an improvement in accuracy relative to drive distance (Wiseman and Chatterjee, 2006). This means that a golfer is able to hit the ball further with greater accuracy.

Swing characteristics and club and ball performance have been shown to influence competition performance. It is important to appreciate the expected ranges for club head speed and ball speed when looking at the results in this thesis. PGA (professional golfers association) tour average ball speed of 270 km/hour. Golfers with a handicap of zero and below have an average ball speed of 259 km/hour. Golfers with a handicap of five have a ball speed of 237 km/hour (Trackman, 2021c). PGA (professional golfers association) tour average carry distance of 252 metres. Golfers with a handicap of zero and below have an average carry distance of 230 metres. Golfers with a handicap of five have an average carry distance of 204 metres (Trackman, 2021a). PGA (professional golfers association) tour average club head speed of 182 km/hour. Golfers with a handicap of zero and below have an average ball speed of 177 km/hour. Golfers with a handicap of five have a ball speed of 163 km/hour (Trackman, 2021d).

Club and ball performance was measured in this research by observing club head speed, ball speed, carry distance, total drive distance and roll while driving at least ten balls at the driving range. These variables were captured using a 3D Doppler tracking radar for golf specific application. Both club and ball performance are important factors to an elite golfer, but competition performance is by far the most important factor. Excellent competition performance, which is measured by order of merit in a competition, is the primary objective or patient specific goal of a young elite male golfer. For this reason, competition performance using a yearly order of merit competition in which all the golfers involved in the performance study participated was used as a measure of golf performance. For more detail on how order of merit was calculated see chapter 6.

Swing biomechanical factors, especially the X-factor, have been associated with improved performance in elite golfers. In a study conducted by Myers et al. (2008) increased X-factor (torso-pelvic angular separation at the top of the back swing), maximum torso-pelvic separation, maximum upper torso rotation velocity, upper torso rotational velocity at lead arm parallel and at the last 40ms before impact, were shown to increase ball velocity. Similarly, a study conducted by Chu et al. (2010) showed that X-factor, delayed release of the arms and wrist, trunk forward and lateral tilting and weight shifting during the swing were associated with improved ball velocity. Lynn et al. (2013) showed that skilled golfers had greater pelvic acceleration and increased time between peak pelvic velocity and ball contact when compared to less skilled golfers. Pelvic acceleration was also shown to correlate with drive distance (Lynn et al., 2013)

As the young elite golfers' primary objective is to perform well in a competition, it is imperative to consider the performance impact of any swing modification even if the modification is aimed at improving longevity and reducing back pain incidence.

2.12 Confounding variables that should be controlled for when determining musculoskeletal and biomechanical injury factors

While assessing the influence that biomechanical and musculoskeletal factors may have on developing back pain in elite male golfers, it is important to control for demographic, anthropometric and training variables that have been show to increase the likelihood of developing lower back pain.

In terms of demographics and past medical history, pooled analysis showed that increasing age was predictive of lower back pain (Smith et al., 2018). Out of four studies reporting on the relationship between gender and lower back injury risk, one study, reported that being male was associated with increased risk of developing lower back pain (Smith et al., 2018). This was not, however, reflected in three other studies (Smith et al., 2018). A previous history of lower back pain was also associated with increased occurrence of lower back pain over a 12 month period (Burdorf et al., 1996).

In terms of anthropometrics, pooled results showed that increasing body mass was associated with developing lower back pain (Smith et al., 2018). Sub analysis of this data, which separated professional and recreational golfers demonstrated that the relationship between increasing body mass and increased likelihood of lower back pain was only present amongst recreational golfers and not in professional golfers (Smith et al., 2018). Hand dominance was not shown to be related to developing lower back pain therefore both left handed and right handed golfers were included in this research (Murray et al., 2009).

In terms of training, it is not possible to perform pooled analysis as there is a lack of heterogeneity in terms of how the studies monitored training (Smith et al., 2018). The majority of studies conducted on the effect of training on developing lower back pain showed no relationship between training frequency (including playing frequency, full shot practice and chipping practice) and developing lower back pain (Smith et al., 2018). A study conducted by McHardy et al. (2007) , however, found that golfers who perform zero to one hour of full shot practice per week had a lower risk of developing lower back pain than golfers who practiced more than one hour of full shot practice per week; but that weekly frequency of chipping practice, putting practice, game play or other sporting activity did not influence the risk of developing lower back pain.

2.13 Rationale for studies contributing to this research

The forces inherent to the golf drive are responsible for facilitating the ability to project the ball, which in turn is a major theoretical determinant of performance. Performance in turn is the major motivator and reason that golfers play. Repetitive forces do, however, have collateral effects such as tissue strain and damage (Edwards et al., 2020). The elite golf swing exerts significant force on the lower back, which is evidenced by the large incidence of lower back pain in elite golfers. Studies have been conducted comparing golfers with lower back pain to those without lower back pain, but a systematic review conducted by Smith et al. (2018) on lower back pain in golf concluded by saying, that the evidence supporting the relationship between swing characteristics (such as kinematics and muscle activity during the swing) and golf related lower back pain remains low, and that additional high quality prospective trials are needed to clarify the risk factors for lower back pain in golfers. The overall purpose of this research is to identify the biomechanical risk factors associated with the development of lower back pain in young elite male golfers within the necessary context of performance.

Chapter 3 and chapter 4 focused on identifying the biomechanical risk factors associated with developing lower back pain in young, elite male golfers. ‘Chapter 3: Lower quadrant swing biomechanics identifies golfers with increased risk of lower back pain, a prospective longitudinal cohort study' aims to identify the lower quadrant kinematic variables associated with developing lower back pain in young elite male golfers while controlling for the following confounding variables: demographics, injury history, training and performance. 'Chapter 4 - Changes in spinal muscle recruitment strategies during the golf swing is linked to developing lower back pain, a prospective longitudinal cohort study' aims to identify the muscle recruitment strategies associated with developing lower back pain in young elite male golfers; while at the same time controlling for the following confounding variables: demographics, injury history, training and performance. These chapters fulfil step two of the TRIPP model of injury prevention and according to this model future intervention studies should be conducted to address these factors.

The third study presented in this research is 'Chapter 5 - The efficacy of injury screening in elite golfers, a prospective longitudinal cohort study' aims to identify if screening tests are able to identify golfers at risk of injury. Ideally prevention strategies should only be targeted at individuals at risk of developing injury. This study aims to address step one of screening model proposed by Bahr (2016), which is to identify the strength of risk factor association with injury development.

The final study presented is 'Chapter 6 - Golf drive distance better predicts overall golfing performance than conventional measurements of swing characteristics, a cross-sectional study in young elite golfers'. This study aims to identify the factors associated with performance. It is essential to identify performance factors, as competition performance is the primary goal of young elite golfers (patient-centred goal). Any biomechanical or musculoskeletal intervention strategy aimed at reducing lower back pain should ideally not influence factors associated with competition performance.

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LINKING PARAGRAPH BETWEEN THE LITERATURE REVIEW AND CHAPTER 3

According to the TRIPP model of injury prevention it is essential to identify factors associated with injury (Emery and Pasanen, 2019). The movement characteristics of the golf swing have been proposed as one of the possible mechanisms of lower back pain in golfers (Smith et al., 2018). In order to understand the possible movement injury associations, a closer look at the swing of elite golfers is required, particularly the golf drive, during which large muscle force and torque is produced in order to generate maximum club head speed at impact so as to maximise golf drive distance (Hume et al., 2005). This next study, 'Lower quadrant swing biomechanics identifies golfers with increased risk of lower back pain, a prospective longitudinal cohort study', is aimed at determining the lower quadrant kinematics associated with developing lower back pain in young elite male golfers.

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CHAPTER 3: LOWER QUADRANT SWING BIOMECHANICS IDENTIFIES GOLFERS WITH INCREASED RISK OF LOWER BACK PAIN, A PROSPECTIVE LONGITUDINAL COHORT STUDY.

Abstract

Background: Elite golfers have a high incidence of lower back pain. Recent reviews have emphasised the need for investigation into how to prevent lower back pain in golfers, prompting the current study.

Study design: Prospective longitudinal cohort study.

Methods: Forty-one injury free golfers were included in this study. At baseline, lower quadrant joint angles, club and ball performance were measured whilst each golfer performed 10 drives. The golfers were then monitored for six months and were allocated into two groups depending on whether they developed lower back pain or not. The initial, baseline kinematic variables of the lower back pain group and the uninjured group were then compared.

Results: Over the six month monitoring period, 17 (41%) of the golfers developed lower back pain and 24 (59%) of the golfers did not. The lower back pain group had: four degrees less non-dominant ankle dorsiflexion at set up ($p=0.01$; effect size=0.82); six degrees less non-dominant knee flexion during the backswing ($p=0.05$; effect size=0.64); six degrees less non-dominant ankle dorsiflexion during the backswing ($p=0.01$; effect size=0.82); six degrees more dominant hip adduction during the backswing ($p=0.02$; effect size=0.79); nine degrees more dominant knee flexion at impact ($p=0.05$; effect size=0.64); six degrees more dominant hip adduction at follow through($p<0.00$; effect size=1.00).

Conclusion: Golfers who developed lower back pain during the six month monitoring period displayed distinct differences in biomechanics at baseline when all golfers were injury free. These biomechanical differences may be considered a precursor to injury.

Clinical relevance: Three dimensional motion analysis may indicate if elite golfers are at risk of developing lower back pain.

Future research: Additionally, research is needed to assess if targeted exercise and sports drills aimed at improving dominant hip abduction during backswing and follow through, as well as squatting on the non-dominant leg at set up and during the backswing may prevent lower back pain in elite golfers.

3.1 Introduction

Golf is a very popular sport with significant health benefits (Khan et al., 2012). Golf does, however, pose a moderate risk for developing lower back pain (Smith et al., 2018). This risk increases in elite golfers who have a lower back pain prevalence of around 50% (Smith et al., 2018). Lower back pain is one of the leading causes of years lived with disability (Vos et al., 2012). This considerable incidence of lower back pain and risk of disability necessitates investigation into how to prevent lower back pain in elite golfers.

The golf swing itself is often cited as a possible cause of lower back pain in golfers (Smith et al., 2018, Cole and Grimshaw, 2016). The golf swing generates considerable force (Gluck et al., 2008), with the lumbar spine sustaining a large amount of compression, axial twisting, torsion, anterior-posterior shearing and lateral flexion forces during the golf swing (Gluck et al., 2008). The following swing characteristics are frequently cited as possible mechanisms of lower back pain in golfers: increased X-factor (angular separation of the shoulder and trunk from the pelvis at the top of backswing), increased crunch factor (the product of maximum lateral flexion toward the lead side and axial plane angular velocity) (Smith et al., 2018, Cole and Grimshaw, 2016), and trunk hyperextension during follow through (Smith et al., 2018).

Movement analysis studies have been conducted to determine the point of maximum intervertebral load and spinal postures associated with increased load. A simulation study (Bae et al., 2014) conducted on ten professional golfers showed that intervertebral load was the highest at the L5/S1 region (area between the fifth lumbar vertebra and the first sacral vertebra) and gradually decreased toward T12 (12^{th} thoracic vertebra). They also showed that the intervertebral load value was the greatest for those that played with kypholordosis, followed by normal lordosis, then hypolordosis, then excessive lordosis before the impact phase; but that this order was reversed during the follow through phase (Bae et al., 2014). They also observed that the load reached a peak for all postures during the follow through phase (Bae et al., 2014). This study provides useful information regarding which postures exert the most load to the spine, but intervertebral load during sport does not necessarily equate to injury. Increased loading to a point may be beneficial, as in the case of patients with osteoporosis. The ratio of load on the spine to failure of the bone (factor of risk) varies from golfer to golfer based on their risk factors (Myers and Wilson, 1997).

Movement analysis studies have been conducted to compare golfers with lower back pain to those without lower back pain. In the cross-sectional study conducted by Cole and Grimshaw (2014) the crunch factor of 12 golfers with a history of lower back pain was compared to 15 asymptomatic controls using three Genlock Panasonic cameras (Matsushita Electric Industrial Company Ltd, Japan). They found no difference in crunch factor maximum angle or timing between those that had lower back pain and the asymptomatic control group (Cole and Grimshaw, 2014). This study was conducted on older recreational golfers and as such the results may not be applicable to young elite golfers. The mean (standard error of mean) age lower back pain group was 46 (5.15) years, mean (SD) handicap lower back pain group was 10.5 (2.43); mean (standard error of mean) age uninjured group was 39.6 (3.6) years; mean (SD) handicap uninjured group was 10.4 (1.07).

In a similar cross-sectional study conducted by Tsai et al. (2010) 16 golfers with a history of lower back pain were compared to 16 age and handicap matched asymptomatic golfers using a Vicon Motus System (Vicon, Centennial, CO). They found that trunk kinematics and maximum spinal movements during the golf swing were similar for those with lower back pain and those without lower back pain (Tsai et al., 2010). Tsai et al. (2010) did, however, observe less trunk rotation towards the trail side during the backswing in the golfers with a history of lower back pain. This study was also conducted on older recreational golfers and therefore may not be generalizable to young elite golfers (lower back pain group age mean (SD) = 48.6 (7.4) years; lower back pain group mean (SD) handicap = 9.1 (4.6); uninjured group age mean (SD) = 47.9 (8.3) years; uninjured group mean (SD) handicap = 9.5 (4.8)).

Lindsay and Horton (2002) conducted a cross section study on professional male golfers comparing six participants who always experienced lower back pain after playing or practicing to six controls who never experienced lower back pain after playing or practicing. They used a wearable triaxial electrogoniometer capable of measuring three dimensional motion of the thoraco-lumbar spine. They found that the golfers with lower back pain had a greater maximal lateral flexion towards the lead side during the backswing, and increased peak trunk lateral flexion angular velocity when compared to the uninjured golfers (Lindsay and Horton, 2002). The golfers in the study conducted by Lindsay and Horton (2002) were older than the golfers in this thesis (mean (SD) age lower back pain group = 29.2 (6.4) years;

mean (SD) age uninjured group = 32.7 (4.8) years) and therefore may not be generalisable to younger elite male golfers.

These studies are valuable in that they show how lower back pain affects swing movement in golfers. They do not, however, establish whether the change in swing pattern was as a result of pain or if the altered movement pattern was present prior to injury. This limitation was mentioned by Cole and Grimshaw (2014). According to a systematic review conducted by Smith et al. (2018) there is no clear evidence linking the development of lower back pain in golfers to swing mechanics. They go on to conclude that because of the low quality and heterogeneity of current research, more investigation into how to prevent lower back pain in golfers is required.

The present study is the first to establish baseline kinematic swing variables prior to potentially developing lower back pain. The hypothesis for this study was that there is a difference in baseline (before injury) lower quadrant kinematics between elite golfers who develop lower back pain and those that do not. All the golfers included in this study were injury free at the time of inclusion and initial assessment and were then monitored for injury over a six month period. This approach enabled injury associations to be identified, as the changes seen in swing pattern would not have been attributed to pain at the time of data collection.

3.2 Materials and Methods

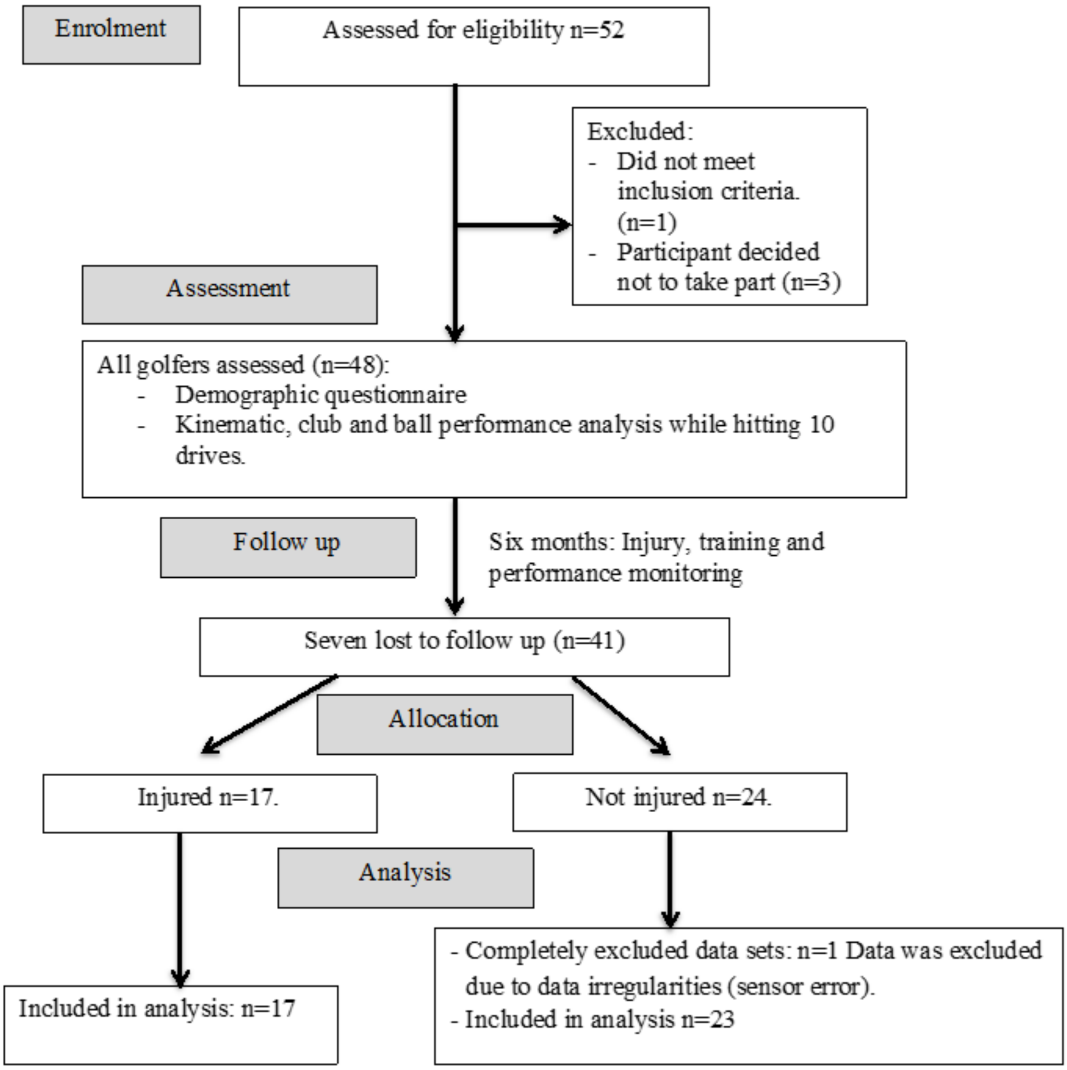

Figure 3.1: Flow diagram overview of the study

3.2.1 Study design and setting

This was a prospective longitudinal cohort study with one baseline measurement. It was performed at the Golf School of excellence, an elite golf academy in Johannesburg. A flow diagram overview showing how the final golfers were selected and the study scheme is shown in the figure (Figure 3.1).

3.2.2 Participants

In order to avoid selection bias, all golfers at the golfing school that were classified as elite players by their coaches were invited to take part in the study. To be included golfers needed to be injury free at the time of inclusion. 'Injury free' was defined as no pain or discomfort while performing activities of daily living or while playing golf. The participants needed to be: recognised as an elite player by the academy's coaches, male and between the ages of 16 and 30 years old. Junior golfers (under 18 years old) were included in the study; therefore a handicap cut off was not applied. Due to an insufficient number of available female participants and the differences in swing mechanics between elite male and elite female golfers (Egret et al., 2006), it was not possible to include female golfers in the study. Golfers were excluded from participation in the study if they had serious hip or spinal pathology. Data were collected from Feb 2017 to Jan 2018.

3.2.3 Ethics

Ethical clearance was received from the ethics committee of the University of the Witwatersrand. The study was conducted according to the ethical standards and guidelines of the Declaration of Helsinki. Identifiable information was removed from the dataset and study numbers were allocated to each participant. The link between the study numbers and identifiable information was kept in an access controlled file to which only the researcher had access.

Written informed consent was granted by participants over the age of 18 years old. Written informed assent was granted by participants under the age of 18 years old and informed written consent was granted by their legal guardians for them to take part in the study. An information sheet was provided to the parents/guardians by the Golf School of Excellence. Minors (participants under 18 years old) were only approach for inclusion in the study if their parents/guardian had given permission for them to take part in the study

Minors were included in this study as a high volume of sports participation has been shown to increase the risk of developing lower back pain in childhood and adolescence (Sato et al., 2011). Research involving minors is permitted if it is therapeutic research in the best interests of the minor (Strode and Slack, 2015). The inclusion of minors in this study was approved by the University of the Witwatersrand's Human Research Ethics Committee

3.2.4 Procedure

Each golfer attended a baseline assessment (before injury occurred). This included the completion of a demographic questionnaire which requested their full names, address, contact numbers, email address, handicap, past medical history and back injury history. Past medical history questions ascertained if the golfers had a diagnosis or history of: i) vascular conditions including angina pectoris, blood pressure problems, congestive cardiac failure, clotting abnormalities, TIA or stroke, coronary artery disease, arrhythmia, heart attack; ii) respiratory conditions including exercise induced asthma, asthma, emphysema or chronic bronchitis; iii) cancer; iv) musculoskeletal conditions such as rheumatoid arthritis, osteoarthritis, osteoporosis, fibromyalgia, chronic lower back or neck pain; v) neurological conditions such as multiple sclerosis, anxiety, depression, Polio, Parkinson's, epilepsy, seizures, or Alzheimer's; vi) gastrointestinal conditions such as stomach or duodenal ulcer, gastric acid reflux, Irritable Bowel Syndrome, diverticulitis or gall bladder problems. Due to the sensitive nature of the questions being asked, golfers were permitted to continue with the study even if they chose to omit some answers to the health screening questionnaire or declined to complete it. Giving the participants the option to omit part or all of the medical screening questionnaire was an important ethical consideration for this study. Despite this option being given, the majority of participants agreed to complete it.

The golfers' anthropometric measurements (height and body mass) were taken and they then underwent a kinematic assessment. Each participant was asked to hit 10 golf balls with their driver just as they would during a competition: where both distance and accuracy are important. Lower quadrant joint angles were measured using an Xsens inertial sensor motion analysis system (MVN Link Biomech system; Xsens Technologies B.V., Enschede, The Netherlands). Both lower limb and trunk kinematics were captured as changes in lower limb kinematics can impact upon the kinetic chain thereby affecting trunk kinematics and trunk muscle recruitment. The system consists of 17 wireless body motion trackers and one wireless club sensor (which was able to detect vibration in the club), which captured movement at a rate of 240 Hz. The Xsens system has been shown to yield data equivalent to that of other, similar kinematic systems (Poitras et al., 2019). The positions of the wireless motion trackers have been provided in Figure 3.2. The segments axes and origin were pre-defined by the Xsens system (Xsens, 2016)[2]. The Xsens system works on basis of Kalman Filtering (Xsens, 2016)[3]. Unlike laboratory golf systems, the Xsens system allows for data capture in a natural golf environment. The golfers in this study performed their golf swings at the driving range at the academy where they usually train.

Figure 3.2: Figure showing the Xsens sensor locations

During the kinematic assessment, club and ball performance was monitored using the Flightscope X2 Elite system (Flightscope X2 Elite, Flightscope (Pty) Ltd, Stellenbosch, South Africa). This is a 3D Doppler tracking radar designed for golf-specific application (Flightscope, Published 2021) as per Leach et al. (2017).

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[2] For more information on how Xsens defined these segments please refer to the MVN user manual Section 23.6 (Pg.128-134) which can be found at https://www.xsens.com/hubfs/Downloads/usermanual/MVN_User_Manual.pdf

[3] More detailed information on how Xsens processes data can be found in the MVN user manual in Section 23.8 (Pg. 134- 137) which can be found at https://www.xsens.com/hubfs/Downloads/usermanual/MVN_User_Manual.pdf.

After the initial baseline assessment, injury was monitored for six months. As most golf injuries are related to repetitive movement, the monitoring period needed to be long enough to capture these injuries. At the same time the injury needed to be related to the swing pattern at the time of assessment. The golfers included in this study were enrolled at a golf academy and in the process of refining their swing pattern. It was felt that a six-month monitoring period would allow sufficient time to capture injuries related to repetitive movement, while at the same time ensuring that the golfers swing had not changed significantly from the time of initial assessment.

Injury was monitored by prompted self-reporting. Golfers were asked to contact the research team immediately if they sustained any injury. Participants were contacted weekly to determine if they sustained an injury or not. ‘Injury’ to the back was defined according to the European guidelines for defining lower back pain (Burton et al., 2006). Lower back pain was defined as ‘pain and discomfort that was localised below the costal margin and above the inferior gluteal folds, with or without leg pain’ (Burton et al., 2006). The lower back pain did not need to result in limited golf participation. The golfers were initially offered a free physiotherapy assessment, which many of the golfers did not choose to attend. Reasons given for not attending the physiotherapy assessment were travel and training commitments. The golfers were free to seek out physiotherapy or medical care if they developed lower back pain. As this was an observational study, no attempt was made to influence their decision to seek care. Once the findings of the study have been published by peer-review journals, the Golf academy will be sent a summary of the findings in simple English as well as some recommendations regarding evidenced-based management of lower back pain. A recommendation will be made to the Golf Academy that they disseminate this information to all the golfers at the academy (current and past students).

Training was also monitored during this six-month period via a weekly training log, which each golfer was asked to complete. If the golfer forgot to complete the log, the primary researcher contacted the golfer via telephone and completed the log on behalf of the golfer. The log included a record of the number of rounds of golf each golfer played per week and the number of hours spent each week engaged in: putting practice, short game practice, range practice, flexibility training, postural training, weight training, and cardiovascular exercise.

Golfer performance was ranked using the 2017 yearly order of merit score. The majority of the golfers at the academy play in a weekly order of merit tournament, the results of which update the order of merit score. The yearly order of merit score is calculated using true score, stroke average, fairways hit, greens hit, number of putts, scrambling (this is a score that measures how often a golfer scores par or better after missing a green in regulation) and ups and downs (a golfer is said to have achieved an ‘up and down’ if he takes only two strokes to get his ball into the hole when his ball is resting around the green or in a greenside bunker).

3.2.5 Data reduction and Analysis

Kinematic data was used to determine the timing of the following swing related events: set up, top of the backswing, impact and end of follow through. Set up was determined as the point just before the club head started to move (Figure 3.3A). The top of the backswing was determined as the club head’s point of zero velocity at the top of the backswing (Figure 3.3B). Impact was determined as the point of club contact, which was determined by the vibration changes exhibited in the club at ball contact (Figure 3.3C). The end of follow through was defined as the end point of the swing. At the end of follow through, the golfer’s trunk should be facing the target in a well-balanced position (Hume et al., 2005). His lumbar spine will be in slight hyperextension and lateral flexion (Hume et al., 2005) (Figure 3.3D).

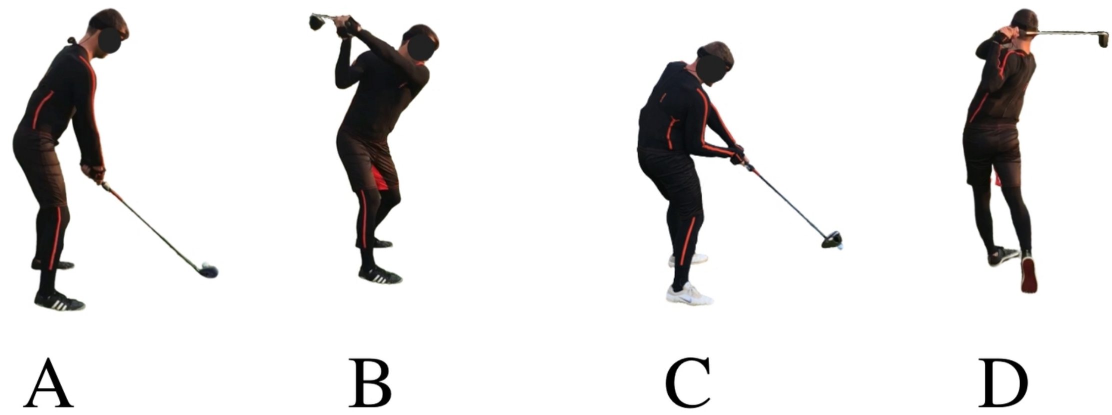


Figure 3.3: Swing related events: set up, top of the backswing, impact and end of follow through.

Joint angle measurements (averaged over 10 swings) were made for each participant for: trunk (lateral flexion, flexion, extension); X-factor; upper thoracic (lateral flexion, rotation, flexion, extension); dominant and non-dominant hip (adduction, abduction, internal rotation, external rotation, flexion, extension); dominant and non-dominant knee (adduction, abduction, internal rotation, external rotation, flexion, extension); dominant and non-dominant ankle (adduction, abduction, inversion and eversion, dorsiflexion, plantiflexion). In order to interpret the joint angles in peripheral joints: i) abduction is recorded as positive; ii) adduction is recorded as negative; iii) internal rotation recorded as positive; iv) external rotation is recorded as negative; v) flexion (and dorsiflexion) is recorded as positive; vi) extension (and plantiflexion) is recorded as negative. In order to interpret the joint angles in the spine: i) rotation towards the non-dominant side is recorded as positive; ii) rotation towards the dominant side is recorded as negative; iii) lateral flexion towards the dominant side is recorded as positive; iv) lateral flexion towards the non-dominant side is recorded as negative; v) flexion is recorded as positive; vi) extension recorded as is negative.

The Flightscope data was recorded as an average of 10 swing cycles. The Flightscope data provided measurements of: carry distance (meters), roll (meters), total distance (meters), ball speed (km/h) and club head speed (km/h).

Two participants were left-handed and played with a left-handed swing. For this reason kinematic measurements were recorded as dominant and non-dominant.

3.2.6 Statistical analysis

Data was analysed using Statistica® Version 12 (StatSoft Inc, Tulsa, USA). Categorical data (Table 3.8) was analysed using Pearson Chi Squared test or the Fisher's Exact Test (Kim, 2017) . Phi (φ) and risk ratio were used to describe the effect size of categorical data when there were two by two contingency tables (Kim, 2017, Pautz et al., 2018a).

$$\varphi = \sqrt{\frac{x^2}{n}}$$

Cramer's V was used to calculate effect size for categorical data on larger tables and was interpreted according to the following table (Table 3.1) (Kim, 2017)

Table 3.1: Interpretation of effect size for Cramer's V

Degree of freedom	Small	Medium	Large
1	0.1	0.3	0.5
2	0.07	0.21	0.35
3	0.06	0.17	0.35
4	0.04	0.13	0.22

Cramer's V was calculated as:

$$V = \sqrt{\frac{x^2}{n-df}}$$

n is the total number of observations. df is the degrees of freedom which is calculated by $(r-1)(c-1)$. Here, r is the numbers of rows of the contingency table and c is the numbers of columns of the contingency table (Kim, 2017). Risk ratios and odds ratios were also calculated for categorical data (Table 3.8).

Continuous and ranked data was tested for normality (Table 3.2- Table 3.7). Normality was determined by dividing kurtosis score by its standard error and by dividing skew by its standard error (z score) (Kim, 2013). If the z score was between -1.96 and 1.96, the data was considered to be normally distributed (Kim, 2013). The data that was normally distributed is marked with a superscript 'n' in the results tables (Table 3.2- Table 3.7). Normally distributed data were analysed using unpaired Student's t-tests was presented as means and standard deviations. Data that was not normally distributed was analysed using the Mann Whitney U-test was presented as medians and interquartile ranges. In line with the American Statistical Association's statement on the use of p-values (Wasserstein et al., 2019), a dichotomised cut-off of p<0.05 was not applied to this research study. The term 'statistically significant' or any of its variants was not used, but p-values were reported as continuous quantities (Wasserstein et al., 2019). I am aware of the developments advising authors not comment on p values between groups for randomised controlled trials (RCT) (de Boer et al., 2015). Part of the argument against using p values for between group differences is the premise that the p-values measure the likelihood that such results would have been obtained by chance. In a RCT the groups should be randomised at baseline using randomisation techniques, therefore a test to estimate this is in itself self-defeating. This study, however, is a cohort study with prospective group allocation based on lower back pain development, therefore the groups were not randomly allocated, therefore p values were used to discuss how similar these groups would have been if they had been allocated by chance. Differences between groups at baseline was presented and assessed using p values, means or medians, standard deviations or interquartile ranges, confidence intervals, effect sizes and minim clinically relevant change.

Confidence intervals and effect sizes were calculated for continuous and ranked data (Pautz et al., 2018a, Pautz et al., 2018b). Effect sizes for data that underwent Student's t-tests was calculated and presented as Cohen's (d_s), where $d_s = \frac{X_1 - X_2}{\sqrt{\frac{(n_1-1)SD_1^2+(n_2-1)SD_2^2}{n_1+n_2-2}}}$ (Pautz et al., 2018b).

Effect sizes for data that had undergone the Mann Whitney U-test was presented as probability of superiority ($P_{a,b}$), where $P_{a.b} = \frac{U}{n_a n_b}$ (Pautz et al., 2018a). Effect sizes for continuous and ranked data were interpreted as: small (≤0.2), moderate (≥0.5), and large (≥0.8) (Cohen, 1992).

As no evidence currently exists to determine the minimum clinically significant difference in joint range of motion, a clinical assessment was made by me to determine what I would consider a meaningful difference to constitute (I currently practice as a highly specialist musculoskeletal physiotherapists and I have 15 years of experience treating patients with musculoskeletal conditions and 10 years of experience treating elite golfers). In order for the change to be considered clinically meaningful it had to be five degrees or more. This viewpoint is supported by the paper published by Lindsay and Horton (2002) in which a change of 6.7 degrees was considered relatively small from a clinical perspective and was of questionable clinical significance. A five degree scale was therefore used on the graphs representing the confidence intervals to give the reader a feel for the relationship of the change to the minimum clinically relevant difference (Figure 3.4- Figure 3.17).

Missing data was not replaced. If an average already existed for the participant (e.g. nine swing cycles, but not 10 swing cycles) then an average over nine swing cycles was calculated. This was explicitly mentioned in the analysis when it occurred. If no answer was provided to the medical screening questionnaire this was omitted from analysis.

Biomechanical studies typically involve small sample sizes. This is largely due to the amount of time it takes to collect data for one participant. Instead of conducting a formal sample size to power the study, p values, confidence intervals, minimum clinically meaningful change and effect sizes were reported. This approach has been used in other biomechanical studies e.g. single leg squat study published by Olivier et al. (2019).

3.3 Results

3.3.1 Participants and incidence of injury

A total of 48 golfers met the inclusion criteria and attended the initial assessment. Seven golfers out of the 48 did not respond to the injury questions and/or complete their training logs and were therefore excluded from the study. Of the remaining 41 golfers, 17 (41%) golfers developed lower back pain over the six month monitoring period. Fourteen golfers only had one episode of lower back pain. Episodes were measured in weeks. The first episode median(IQR)=2(4) weeks. Three participants had two episodes of lower back pain. The second episode mean(SD)=2.3(0.56) weeks. The total length of back pain episodes (first and second) median(IQR)= 2(5) weeks. Three participants reported lower back pain and lower limb pain at the same time. Fifteen participants continued to play golf despite having lower back pain. One of the golfers in the lower back pain group only had one swing cycle collected (this was included in the analysis). Three of the participants from the uninjured group only had nine swings recorded (the nine swings were averaged and included in the analysis). All others completed all 10 swing cycles. The mean (SD) age was 19.8 (2.54) years, height was 1.61 (0.07) m, body mass was 77.94 (13.15) kg, and BMI was 24.26 (3.69) kg/m^2).

3.3.2 Baseline kinematics

Differences in lower quadrant joint angles between golfers who developed lower back pain during the follow up period and those who did not were determined at four time points during the golf swing, each of which is presented in the following four tables: Table 3.2, Table 3.3, Table 3.4 and Table 4.5.

Set up

At set up the lower back pain group had: i) a five degree higher trunk lateral flexion towards the dominant side (p = 0.02, effect size = 0.28) (Figure 3.4); ii) one degree higher upper thoracic lateral flexion towards the dominant side (p = 0.02, effect size = 0.28) (Figure 3.5); iii) the non-dominant knee was in three degrees more abduction (p = 0.02, effect size = 0.28) (Figure 3.6); iv) four degrees less non-dominant knee flexion (p = 0.01; effect size = 0.25) (Figure 3.7); v) four degrees less non-dominant ankle dorsiflexion (p = 0.01; effect size = 0.82) (Figure 3.8). No other differences in lower quadrant joint kinematics were observed at baseline assessment for the set up position (Table 3.2).

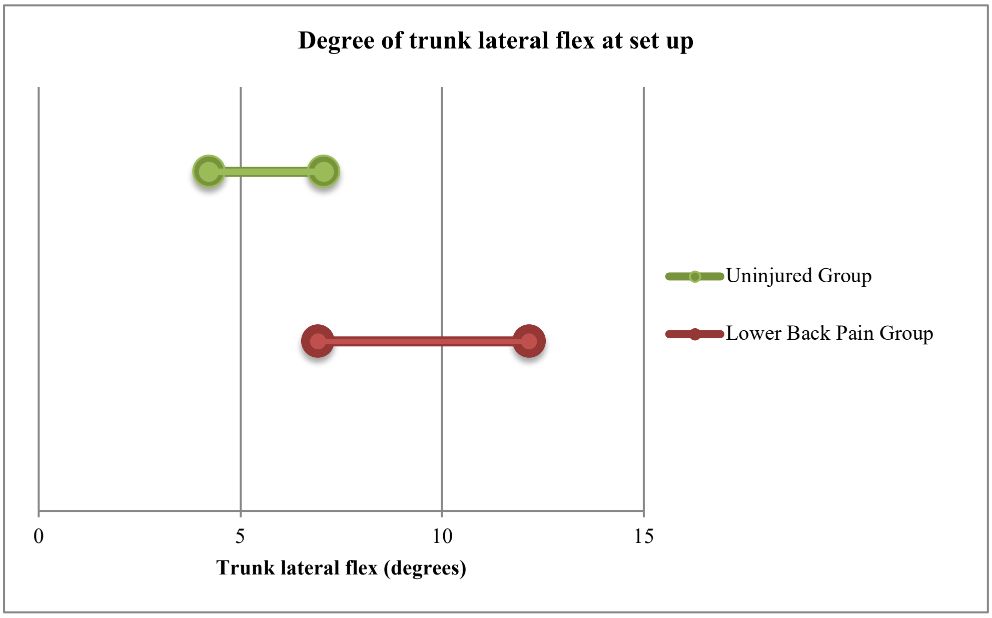
When viewing the confidence intervals for trunk lateral flexion towards the dominant side at set up (Figure 3.4), upper thoracic lateral flexion towards the dominant side at set up (Figure 3.5) non-dominant knee abduction at set up (Figure 3.6) and non-dominant knee flexion (Figure 3.7) the differences between the confidence intervals for the lower back pain group and the uninjured group did not exceed the clinically meaningful value of 5 degrees. The effect sizes of these differences were also small, therefore the differences between the groups in terms of these variables was not likely to have clinical significance.

When viewing the confidence intervals for non-dominant ankle dorsiflexion at set up (Figure 3.8) the differences between the confidence intervals for the lower back pain group and the uninjured group did not exceed the clinically meaningful value of 5 degrees, but the effect size was large, therefore this finding should be viewed cautiously, especially as a minimum clinically meaningful difference in range of motion in young, elite male golfers has not been established through rigorous research.

Table 3.2: Table to compare lower back pain group (n=17) versus uninjured group's (n=23) lower quadrant kinematics at baseline assessment for the set up position

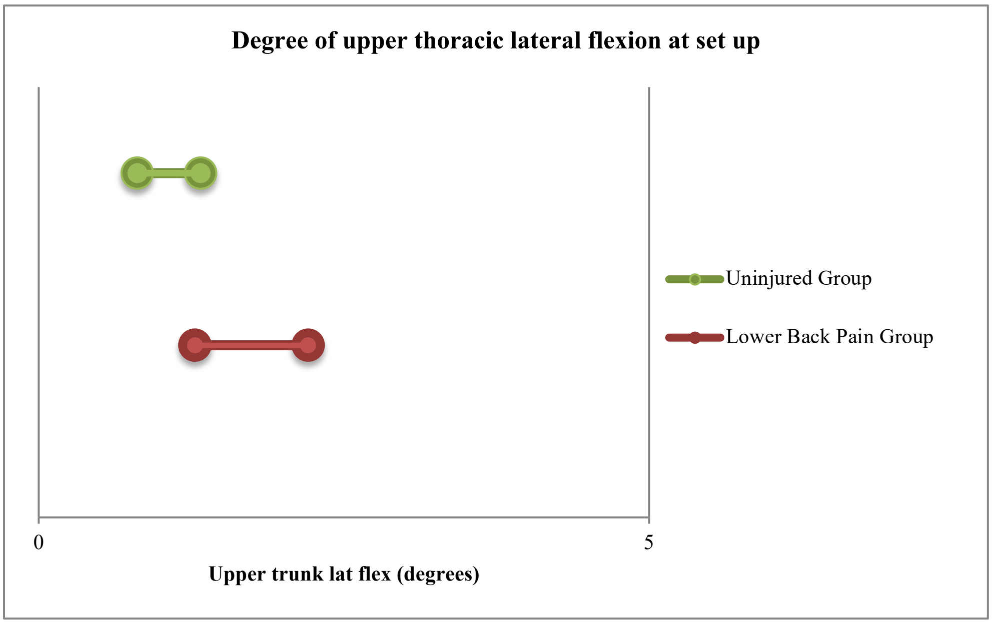
Joint angle (degrees)	Uninjured group n=23			Lower back pain group n=17			Comparison between groups at baseline		
	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper	p value	Effect size	Effect size
Trunk lateral flex non-dominant and dominant side	6.40 (4.07)	4.22	7.07	11.08 (7.64)	6.93	12.16	0.02	0.28	small
X-factor non-dominant and dominant side[n]	9.25 (6.53)	6.42	12.07	10.80 (6.12)	7.65	13.95	0.45	0.24	small
Trunk flex/ext[n]	22.83 (5.74)	20.35	25.31	19.74 (6.74)	16.28	23.21	0.13	0.5	medium
Upper thoracic lat flex non-dominant and dominant side	1.15 (0.71)	0.81	1.33	2.01 (1.31)	1.28	2.21	0.02	0.28	small
Upper thoracic rot non-dominant and dominant side[n]	1.59 (1.15)	1.09	2.08	1.84 (1.09)	1.27	2.40	0.49	0.22	small
Upper thoracic flex/ext[n]	4.01 (1.01)	3.57	4.45	3.45 (1.18)	2.85	4.06	0.12	0.51	medium
Dominant hip abd/add[n]	15.73 (6.69)	12.83	18.62	13.52 (3.35)	11.80	15.25	0.18	0.4	small
Dominant hip int/ext[n]	-4.32 (4.06)	-6.07	-2.56	-4.12 (4.44)	-6.4	-1.84	0.89	0.05	small
Dominant hip flex/ext[n]	25.2 (7.46)	21.98	28.43	25 (7.62)	21.08	28.92	0.93	0.03	small
Dominant knee abd/add	-2.4 (5.72)	-2.85	0.95	-0.77 (5.55)	-2.74	1.3	0.53	0.44	small
Dominant knee int/ext [n]	6.24 (3.7)	4.64	7.84	6.42 (6.85)	2.89	9.94	0.92	0.03	small
Dominant knee flex/ext	27.04 (11.37)	23.89	30.48	28.3 (5.13)	24.59	29.99	0.96	0.49	small
Dominant ankle abd/add[n]	-5.15 (5.99)	-7.74	-2.56	-5.09 (3.96)	-7.12	-3.05	0.97	0.01	small
Dominant ankle inversion/eversion	0.4 (5.89)	-2.38	4.55	2.74 (7.94)	-2.98	9.05	0.21	0.38	small
Dominant ankle dorsiflex/plantiflex[n]	10.79 (5.12)	8.58	13.01	9.76 (5.33)	7.02	12.5	0.54	0.2	small
Non-dominant hip abd/add[n]	13.04 (4.48)	11.1	14.97	10.23 (7.32)	6.46	13.99	0.14	0.48	small
Non-dominant hip int/ext	-12.71 (10.69)	-14.99	-7.95	-15.01 (13.21)	-18.82	-3.59	0.48	0.43	small
Non-dominant hip flex/ext[n]	25.94 (7.04)	22.9	28.98	20.49 (10.46)	15.11	25.87	0.06	0.63	medium
Non-dominant knee abd/add	-1.19 (5.12)	-2.51	1.63	1.82 (5.68)	-0.39	6.38	0.02	0.28	small
Non-dominant knee int/ext[n]	7.53 (4.6)	5.54	9.52	9.71 (7.09)	6.07	13.36	0.24	0.38	small
Non-dominant knee flex/ext	25.31 (9.4)	22.65	28.45	21.24 (12.32)	9.56	23.04	0.01	0.25	small
Non-dominant ankle abd/add[n]	-6.49 (5.71)	-8.96	-4.02	-6.58 (6.72)	-10.04	-3.13	0.96	0.02	small
Non-dominant ankle inversion/ eversion	1.85 (5.73)	-1.37	5.62	1.32 (12.55)	-6.89	4.04	0.48	0.43	small
Non-dominant ankle dorsiflex/plantiflex[n]	6.48 (5.72)	4	8.95	2.12 (4.74)	-0.32	4.55	0.01	0.82	large

Table notes: n =number of participants ; SD = standard deviation; IQR = interquartile range; [n] =normally distributed data; p values calculated using Mann Whitney U test and T tests depending on normality.



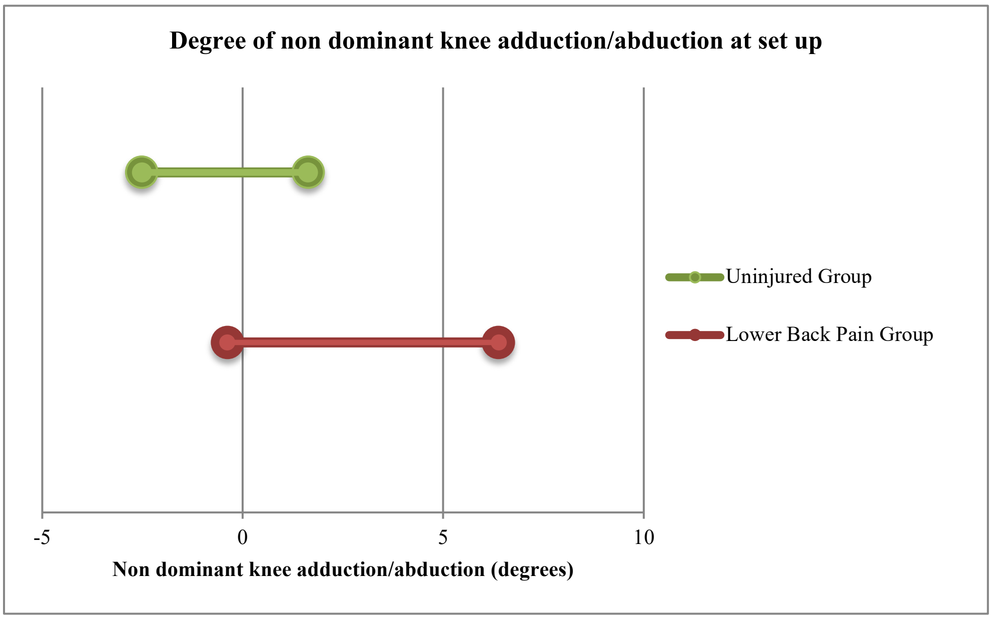
Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.4: Graph representing the difference in 95% confidence intervals for the degree of trunk lateral flexion at the set up position between the uninjured group and the lower back pain group at baseline assessment



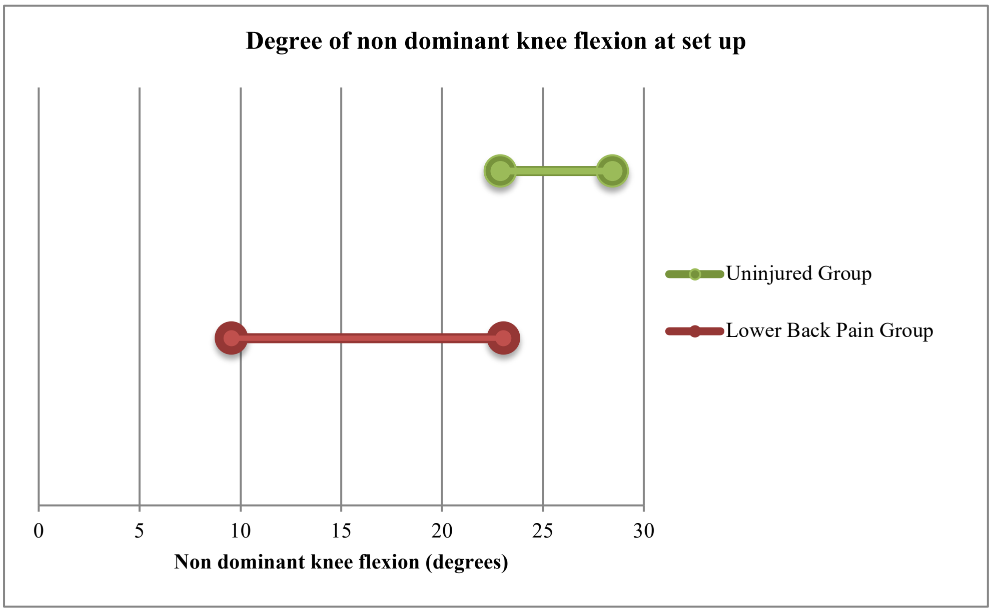
Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.5: Graph representing the difference in 95% confidence intervals for the degree of upper thoracic lateral flexion at the set up position between the uninjured group and the lower back pain group at baseline assessment



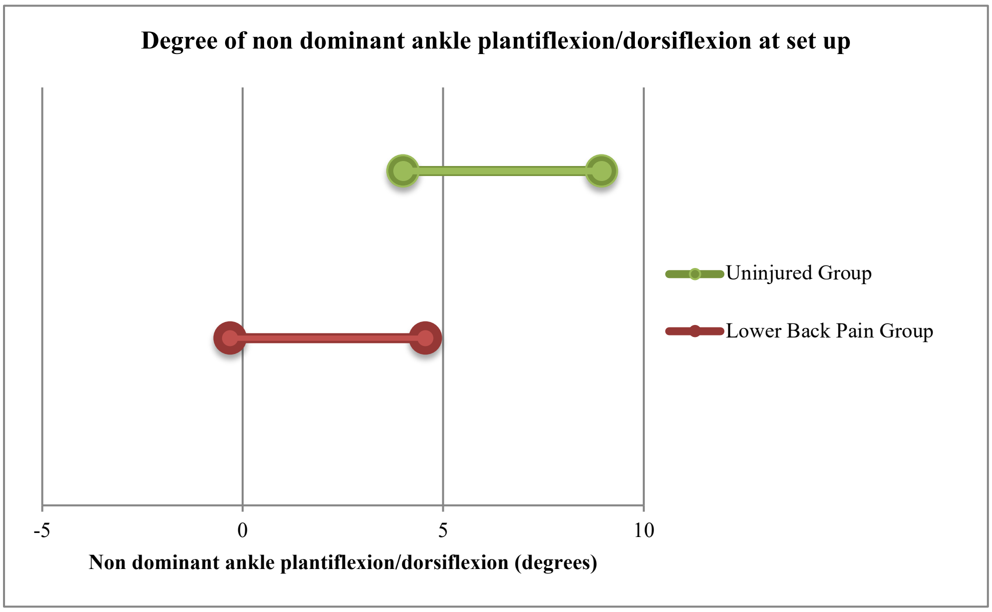
Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.6: Graph representing the difference in 95% confidence intervals for the degree of non-dominant knee adduction/abduction at the set up position between the uninjured group and the lower back pain group at baseline assessment



Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.7: Graph representing the difference in 95% confidence intervals for the degree of non-dominant knee flexion at the set up position between the uninjured group and the lower back pain group at baseline assessment



Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.8: Graph representing the difference in 95% confidence intervals for the degree of non-dominant ankle plantiflexion and dorsiflexion for the set up position between the uninjured group and the lower back pain group at baseline assessment

Top of the backswing

At the baseline assessment at the top of the backswing the lower back pain group had: i) six degrees more dominant hip adduction ($p = 0.02$; effect size = 0.79); ii) eight degrees less non-dominant knee adduction ($p = 0.003$; effect size = 0.23); iii) six degrees less non-dominant knee flexion ($p = 0.05$; effect size = 0.64); iv) seven degrees more non-dominant ankle adduction ($p = 0.04$; effect size = 0.31); v) six degrees less non-dominant ankle dorsiflexion ($p = 0.01$; effect size = 0.82). No other differences in lower quadrant joint kinematics were observed at the top of the backswing at baseline assessment (Table 3.3).

When viewing the confidence intervals for dominant hip adduction (Figure 3.9) there was not much overlap of confidence intervals, the mean difference was above the clinically meaningful difference and the effect size was moderate. This indicates that this was a clinically relevant difference.

When viewing the confidence intervals for non-dominant knee adduction (Figure 3.10) there was not much overlap of confidence intervals, the mean difference was above the clinically meaningful difference, but the effect size was small. This indicates that the difference may not have been clinically relevant.

When viewing the confidence intervals for non-dominant knee flexion (Figure 3.11) there was moderate overlap of confidence intervals but this may be due to the large confidence interval in the lower back pain group. The mean difference was above the clinically meaningful difference and the effect size was moderate. This indicates that this was a clinically relevant difference.

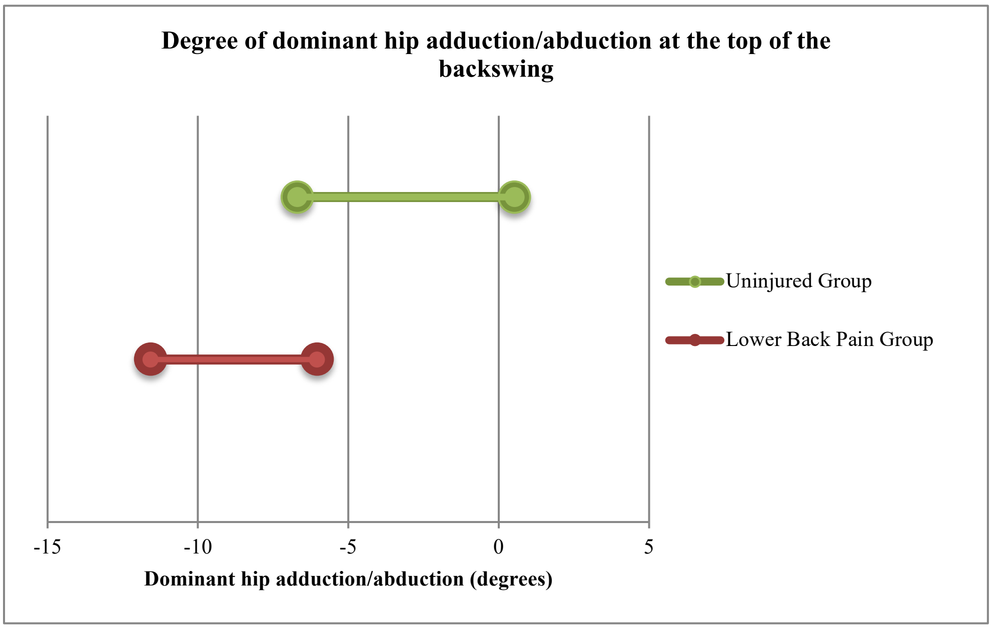
When viewing the confidence intervals for non-dominant ankle adduction (Figure 3.12) there was not much overlap of confidence intervals, the mean difference was above the clinically meaningful difference, but the effect size was small. This indicates that the difference may not have been clinically relevant.

When viewing the confidence intervals for non-dominant ankle dorsiflexion (Figure 3.13) there was not much overlap of confidence intervals, the mean difference was above the clinically meaningful difference and the effect size was large. This indicates that this was a clinically relevant difference.

Table 3.3: Table to compare lower back pain group (n=17) versus uninjured group's (n=23) lower quadrant kinematics at baseline at the top of the backswing

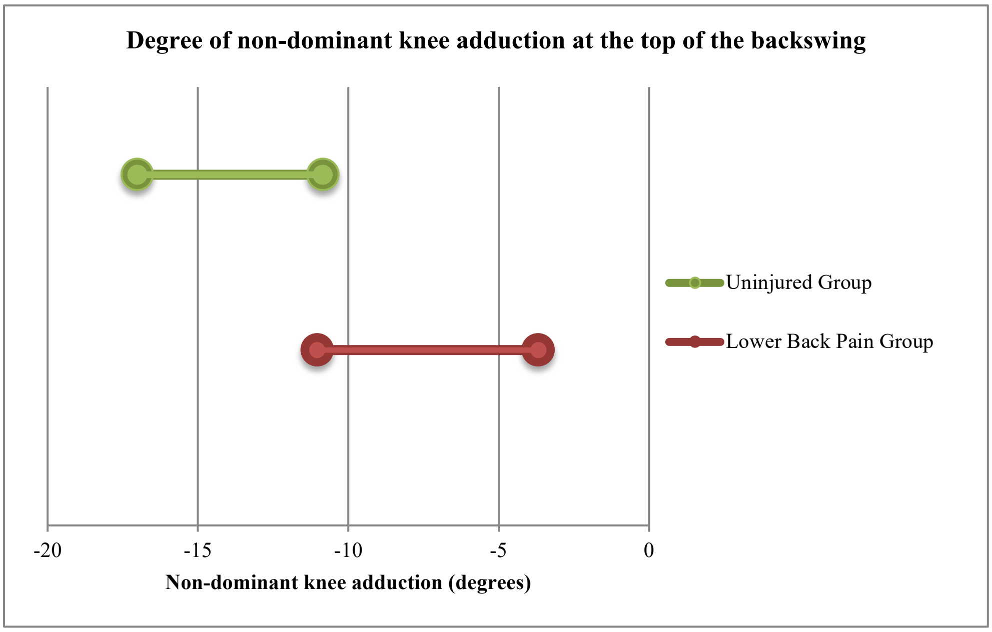
Joint angle (degrees)	Uninjured group n=23			Lower back pain group n=17			Comparison between groups at baseline		
	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper	Mean (SD) or Median (IQR)	95% CI lower	95% CI Upper	p value	Effect size value	Effect size
Trunk lateral flex non-dominant and dominant side	-6.95 (8.51)	-9.39	-4.57	-9.22 (4.54)	-11.4	-5.22	0.18	0.37	small
X-factor non-dominant and dominant side^{n}	-37.22 (9.65)	-41.39	-33.04	-37.04 (7.75)	-41.03	-33.05	0.95	0.02	small
Trunk flex/ext^{n}	10.47 (6.16)	7.81	13.14	8.14 (7.9)	4.08	12.2	0.3	0.34	small
Upper thoracic lat flex non-dominant and dominant side	-1.26 (1.36)	-1.74	-0.9	-1.66 (1.01)	-2.06	-1.01	0.18	0.37	small
Upper thoracic rot non-dominant and dominant side^{n}	-6.51 (1.68)	-7.23	-5.78	-6.49 (1.38)	-7.2	-5.77	0.96	0.01	small
Upper thoracic flex/ext ^{n}	1.74 (1.1)	1.27	2.22	1.32 (1.43)	0.59	2.06	0.30	0.34	small
Dominant hip abd/add^{n}	-3.08 (8.35)	-6.7	0.53	-8.81 (5.38)	-11.58	-6.05	0.02	0.79	medium
Dominant hip int/ext^{n}	11.87 (4.41)	9.96	13.78	12.97 (4.83)	10.49	15.45	0.46	0.24	small
Dominant hip flex/ext^{n}	36.14 (8.19)	32.6	39.68	36.21 (6.44)	32.9	39.52	0.98	0.01	small
Dominant knee abd/add^{n}	-3.32 (4.28)	-5.17	-1.47	-1.53 (4.67)	-3.93	0.87	0.22	0.40	small
Dominant knee int/ext^{n}	15.12 (6.42)	12.35	17.9	13.9 (7.25)	10.17	17.62	0.58	0.18	small
Dominant knee flex/ext^{n}	29.06 (9.64)	24.89	33.23	31.28 (8.46)	26.93	35.63	0.45	0.24	small
Dominant ankle abd/add	-3.32 (10.98)	-7.17	-1.54	-4.57 (4.32)	-7.38	-2.26	0.50	0.43	small
Dominant ankle inversion/ eversion	15.47 (9.49)	10.15	18.71	12.72 (17.07)	6.44	18.86	0.59	0.45	small
Dominant ankle dorsiflex/ plantiflex	2.45 (13.72)	1.88	8.2	7.55 (6.53)	2.68	9.84	0.53	0.44	small
Non-dominant hip abd/add^{n}	32.88 (6.58)	30.04	35.73	33.78 (7.66)	29.84	37.72	0.69	0.13	small
Non-dominant hip int/ext^{n}	-20 (7.98)	-23.45	-16.55	-21.07 (7.39)	-24.87	-17.28	0.67	0.14	small
Non-dominant hip flex/ext	12.26 (11.15)	8.15	17.77	11.49 (12.64)	6.79	15.44	0.32	0.40	small
Non-dominant knee abd/add	-14.15 (6.72)	-17.02	-10.85	-6.39 (7.87)	-11.04	-3.68	0.003	0.23	small
Non-dominant knee int/ext^{n}	-14.22 (6.81)	-17.16	-11.27	-18.8 (8.53)	-23.19	-14.41	0.07	0.60	medium
Non-dominant knee flex/ext^{n}	46.81 (8.51)	43.13	50.49	41.28 (8.73)	36.79	45.76	0.05	0.64	medium
Non-dominant ankle abd/add	-5.43 (14.31)	-10.94	-4.2	-12.57 (9.45)	-16.37	-9.69	0.04	0.31	small
Non-dominant ankle inversion/ eversion^{n}	-9.39 (10.67)	-14	-4.78	-13.85 (11.06)	-19.54	-8.17	0.21	0.41	small
Non-dominant ankle plantiflex/dorsiflex^{n}	24.43 (6.46)	21.63	27.22	18.8 (7.47)	14.97	22.64	0.01	0.82	large

Table notes: n =number of participants ; SD = standard deviation; IQR = interquartile range; ^{n} =normally distributed data; p values calculated using Mann Whitney U test and T tests depending on normality.



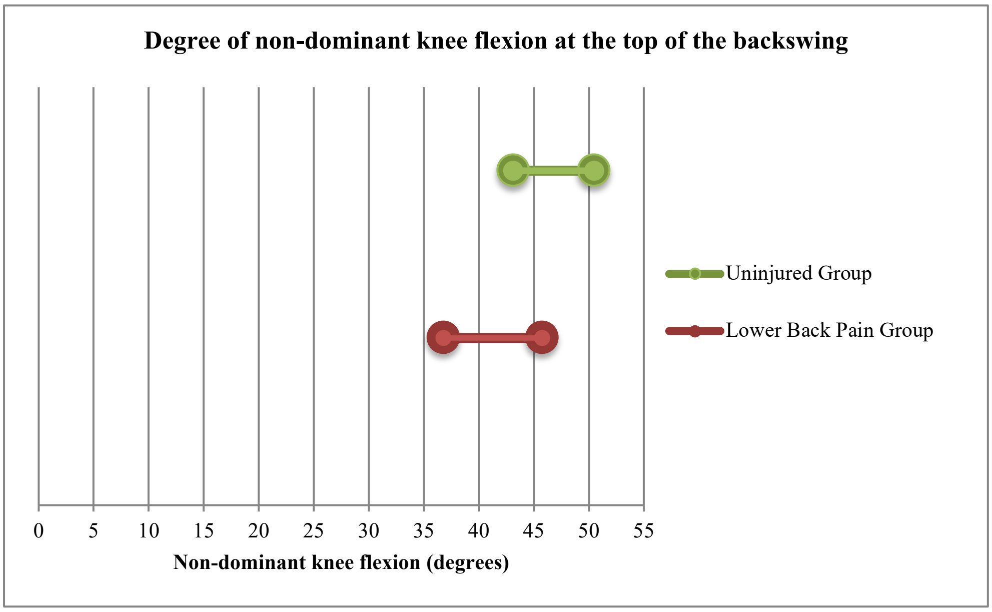
Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.9: Graph representing the difference in 95% confidence intervals for the degree of dominant hip adduction/abduction at the top of the backswing between the uninjured group and the lower back pain group at baseline assessment



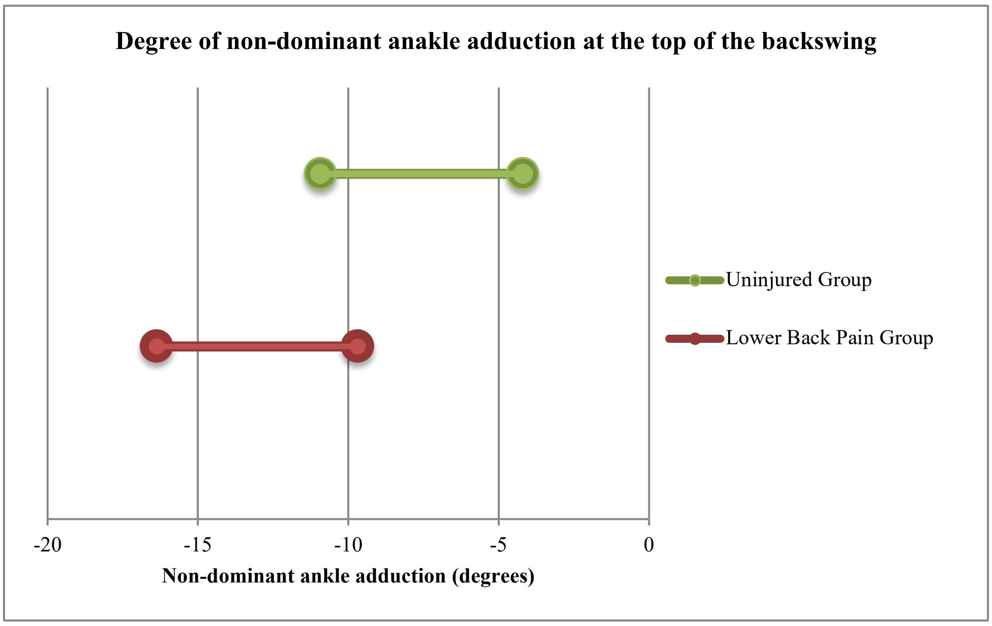
Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.10: Graph representing the difference in 95% confidence intervals for the non-dominant knee adduction/ abduction at the top of the backswing between the uninjured group and the lower back pain group at baseline assessment



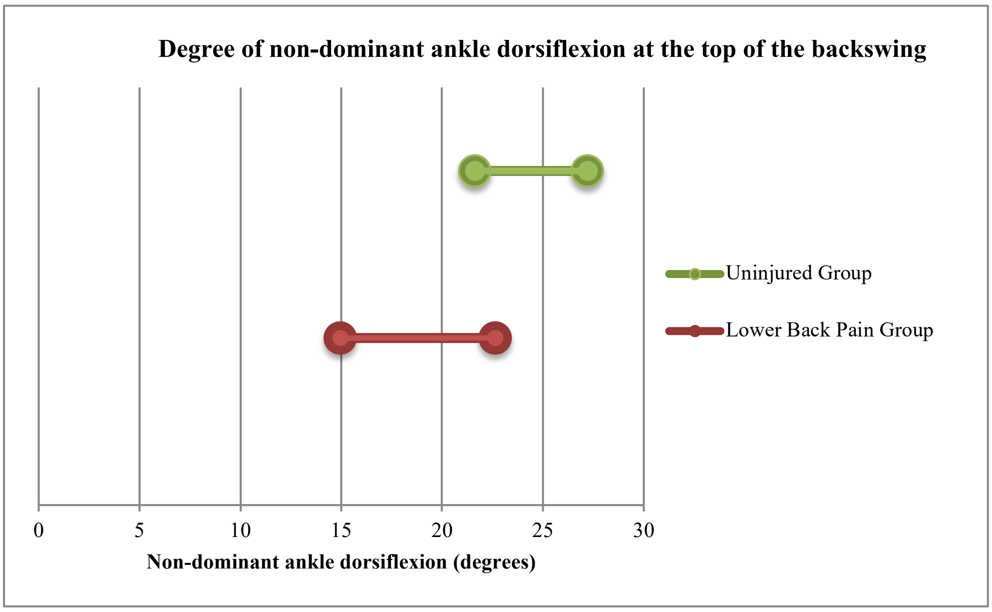
Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.11: Graph representing the difference in 95% confidence intervals for the degree of non-dominant knee flexion at the top of the backswing between the uninjured group and the lower back pain group at baseline assessment



Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.12: Graph representing the difference in 95% confidence intervals for the degree of non-dominant ankle adduction at the top of the backswing between the uninjured group and the lower back pain group at baseline assessment



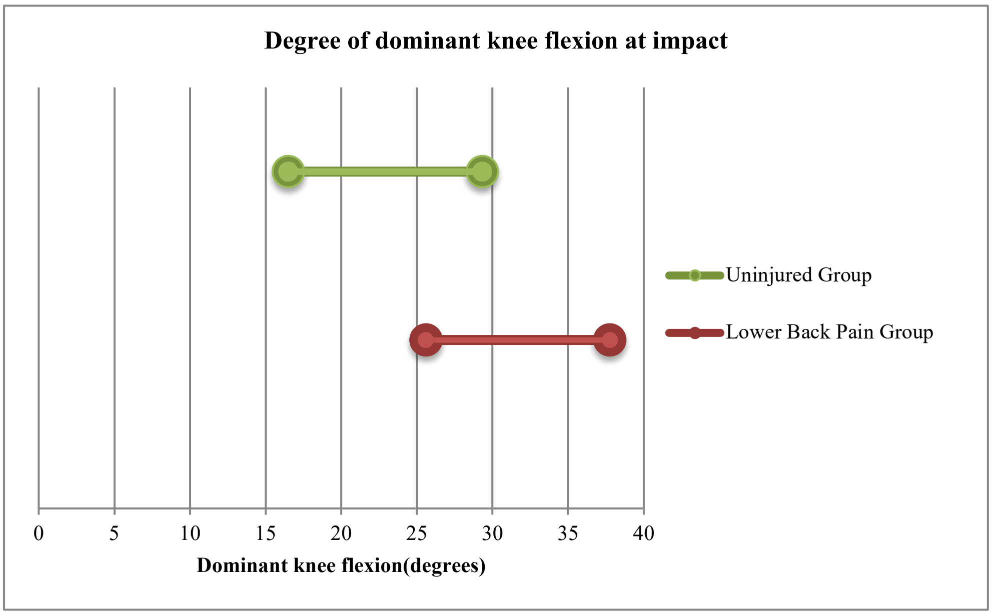
Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.13: Graph representing the difference in 95% confidence intervals for the degree of non-dominant ankle dorsiflexion at the top of the backswing between the uninjured group and the lower back pain group at baseline assessment

Impact

At baseline assessment at impact the lower back pain group had: i) nine degrees more dominant knee flexion ($p = 0.05$; effect size = 0.64). No other difference in lower quadrant joint kinematics were observed at impact during baseline assessment (Table 3.4).

When viewing the confidence intervals for dominant knee flexion (Figure 3.14) there was moderate overlap of confidence intervals but this may be due to the large confidence interval in both the lower back pain group and the uninjured group. The mean difference was well above the clinically meaningful difference and the effect size was moderate. This indicates that this was a clinically relevant difference.



Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.14: Graph representing the difference in 95% confidence intervals for the degree of dominant knee flexion at impact between the uninjured group and the lower back pain group at baseline assessment

Table 3.4: Table to compare lower back pain group (n=17) versus uninjured group's (n=23) lower quadrant kinematics at baseline at the point of impact.

Joint angle (degrees)	Uninjured group n=23			Lower back pain group n=17			Comparison between groups at baseline		
	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper	Mean (SD) or Median (IQR)	95% CI lower	95% CI Upper	p value	Effect size value	Effect size
Trunk lateral flex non-dominant and dominant side^{n}	23.37 (5.51)	20.99	25.75	21.79 (4.63)	19.41	24.17	0.35	0.31	small
X-factor non-dominant and dominant side^{n}	-18.09 (6.9)	-21.07	-15.11	-15.83 (5.94)	-18.89	-12.78	0.28	0.35	small
Trunk flex/ext^{n}	20.15 (7.5)	16.91	23.39	20.4 (6.61)	16.99	23.8	0.91	0.03	small
Upper thoracic lat flex non-dominant and dominant side^{n}	3.96 (0.98)	3.53	4.38	3.67 (0.8)	3.26	4.09	0.34	0.31	small
Upper thoracic rot non-dominant and dominant side^{n}	-3.36 (1.21)	-3.88	-2.83	-2.88 (1.08)	-3.44	-2.33	0.21	0.41	small
Upper thoracic flex/ext^{n}	3.74 (1.31)	3.18	4.31	3.76 (1.17)	3.16	4.36	0.97	0.01	small
Dominant hip abd/add	27.85 (7.78)	25.78	31.75	26.2 (8.61)	20.8	28.53	0.14	0.36	small
Dominant hip int/ext^{n}	-15.62 (7.43)	-18.83	-12.4	-14.24 (5.78)	-17.21	-11.27	0.53	0.2	small
Dominant hip flex/ext^{n}	-15.08 (12.56)	-20.51	-9.64	-16.25 (9.68)	-21.23	-11.27	0.75	0.1	small
Dominant knee abd/add^{n}	5.68 (4.57)	3.71	7.66	6.73 (6.11)	3.59	9.87	0.54	0.2	small
Dominant knee int/ext	-1.4 (6.71)	-4.12	1.28	-1.35 (7.87)	-2.93	5.72	0.37	0.41	small
Dominant knee flex/ext^{n}	22.91 (14.83)	16.5	29.32	31.68 (11.84)	25.59	37.77	0.05	0.64	medium
Dominant ankle abd/add	-4.86 (15.53)	-12.398	0.298	-11.992 (16.727)	-19.603	-4.607	0.2	0.38	small
Dominant ankle inversion/ eversion^{n}	-9.306 (13.576)	-15.18	-3.44	-9.3 (11.89)	-15.42	-3.18	1	0.00	small
Dominant ankle dorsiflex/ plantiflex^{n}	-8.9 (15.091)	-15.42	-2.37	-7.84 (12.69)	-14.37	-1.32	0.82	0.07	small
Non-dominant hip abd/add^{n}	-7.74 (5.14)	-9.96	-5.52	-6.16 (5.58)	-9.03	-3.29	0.36	0.3	small
Non-dominant hip int/ext^{n}	-8.42 (9.65)	-12.59	-4.25	-9.59 (6.5)	-12.93	-6.25	0.67	0.14	small
Non-dominant hip flex/ext^{n}	2.18 (8.2)	-1.37	5.72	0.19 (8.6)	-4.23	4.61	0.46	0.24	small
Non-dominant knee abd/add^{n}	-5.35 (4.06)	-7.11	-3.59	-4 (5.25)	-6.7	-1.3	0.37	0.29	small
Non-dominant knee int/ext^{n}	9.68 (5.42)	7.33	12.02	13.41 (7.89)	9.35	17.47	0.08	0.57	medium
Non-dominant knee flex/ext^{n}	17.92 (12.06)	12.71	23.14	17.33 (9.5)	12.44	22.21	0.87	0.05	small
Non-dominant ankle abd/add^{n}	-4.37 (11.67)	-9.43	0.67	-5.31 (5.89)	-8.34	-2.28	0.74	0.1	small
Non-dominant ankle inversion/ eversion^{n}	21.91 (8.52)	18.23	25.59	18.94 (12.24)	12.65	25.24	0.37	0.29	small
Non-dominant ankle dorsiflex/ plantiflex^{n}	-7.34 (13.15)	-13.03	-1.65	-12.85 (15.37)	-20.75	-4.94	0.23	0.39	small

Table notes: n =number of participants ; SD = standard deviation; IQR = interquartile range; ^{n} =normally distributed data; p values calculated using Mann Whitney U test and T tests depending on normality.

Follow through

At baseline assessment at follow through the lower back pain group had: i) six degrees more dominant hip adduction ($p < 0.003$; effect size = 1.00); ii) six degrees more dominant ankle adduction ($p = 0.05$; effect size = 0.32); iii) One degree less non-dominant ankle inversion ($p < 0.00$; effect size = 0.26). No other differences in lower quadrant joint kinematics were observed at the top of the backswing during baseline assessment (Table 3.5).

When viewing the confidence intervals for dominant hip adduction (Figure 3.15) there was no overlap of confidence intervals, the mean difference was above the clinically meaningful difference and the effect size was large. This indicates that this was a clinically relevant difference.

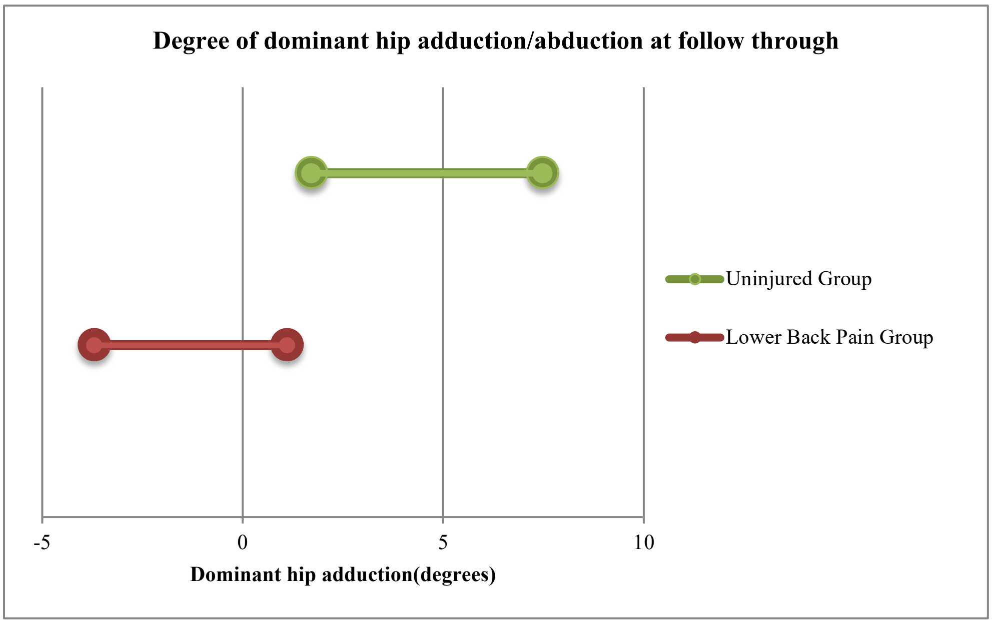
When viewing the confidence intervals for dominant ankle adduction (Figure 3.16) there was large overlap of confidence intervals, the mean difference was above the clinically meaningful difference, but the effect size was small. This indicates that this was probably not a clinically relevant difference.

When viewing the confidence intervals for non-dominant ankle inversion (Figure 3.17) there was a large overlap of confidence intervals, the mean difference was below the clinically meaningful difference, and the effect size was small. This indicates that this was a not a clinically relevant difference.

Table 3.5: Table to compare lower back pain group (n=17) versus uninjured group's (n=23) lower quadrant kinematics at baseline at the point of follow through

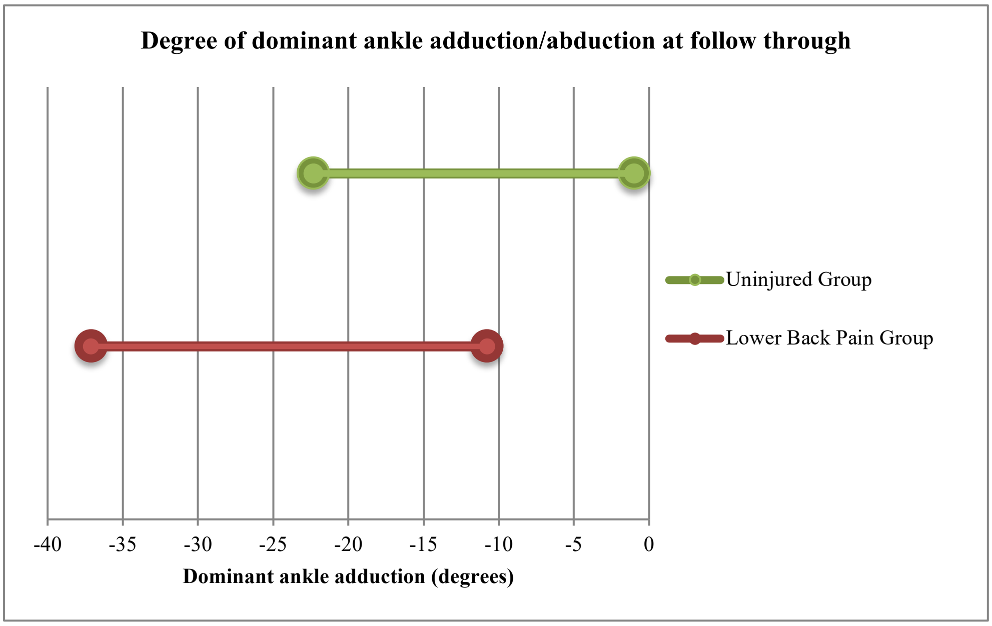
Joint angle (degrees)	Uninjured group n=23			Lower back pain group n=17			Comparison between groups at baseline		
	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper	Mean (SD) or Median (IQR)	95% CI lower	95% CI Upper	p value	Effect size value	Effect size
Trunk lateral flex non-dominant and dominant side^{n}	-1.21 (6.4)	-3.98	1.56	-4.45 (4.7)	-6.87	-2.04	0.09	0.56	medium
X-factor non-dominant and dominant side^{n}	31.5 (6.58)	28.65	34.34	30.62 (6.06)	27.5	33.73	0.67	0.14	small
Trunk flex and ext^{n}	-16.25 (7.12)	-19.33	-13.17	-17.45 (9.01)	-22.08	-12.82	0.64	0.15	small
Upper thoracic lat flex non-dominant and dominant side^{n}	-0.37 (1.15)	-0.86	0.13	-0.97 (0.85)	-1.41	-0.53	0.08	0.58	medium
Upper thoracic rot non-dominant and dominant side ^{n}	5.55 (1.16)	5.05	6.05	5.38 (1.06)	4.83	5.92	0.63	0.15	small
Upper thoracic flex and ext ^{n}	-2.85 (1.26)	-3.4	-2.31	-3.04 (1.58)	-3.85	-2.22	0.68	0.13	small
Dominant hip abd/add ^{n}	4.59 (6.67)	1.71	7.48	-1.3 (4.68)	-3.71	1.1	0.003	1	large
Dominant hip int/ext ^{n}	-6.21 (6.17)	-8.88	-3.55	-2.86 (6.07)	-5.98	0.26	0.1	0.55	medium
Dominant hip flex/ext	-23.21 (6.91)	-26.51	-20.09	-21.97 (12.32)	-27.62	-18.99	0.53	0.44	small
Dominant knee abd/add ^{n}	-0.03 (3.46)	-1.53	1.46	-1.87 (3.6)	-3.72	-0.02	0.11	0.52	medium
Dominant knee int/ext ^{n}	-2.82 (10.08)	-7.18	1.55	-1.63 (9.64)	-6.58	3.33	0.71	0.12	small
Dominant knee flex/ext ^{n}	25.89 (6.43)	23.11	28.68	22.64 (5.99)	19.56	25.72	0.11	0.52	medium
Dominant ankle abd/add	-12.36 (21.8)	-22.32	-1	-18.36 (29.11)	-37.12	-10.77	0.05	0.32	small
Dominant ankle inversion/eversion	-6.37 (34.03)	-9.66	10.81	9.36 (46.23)	-7.18	19.91	0.36	0.41	small
Dominant ankle dorsiflex/ plantiflex	-42.356 (23.069)	-46.971	-19.288	-40.409 (15.165)	-49.070	-10.634	0.55	0.44	small
Non-dominant hip abd/add ^{n}	-12.938 (4.755)	-14.995	-10.882	-11.711 (5.529)	-14.554	-8.869	0.46	0.24	small
Non-dominant hip int/ext ^{n}	8.089 (8.044)	4.610	11.567	11.262 (6.603)	7.867	14.657	0.19	0.42	small
Non-dominant hip flex/ext ^{n}	5.378 (6.334)	2.639	8.117	4.565 (8.223)	0.337	8.793	0.73	0.11	small
Non-dominant knee abd/add	-2.486 (5.838)	-4.811	-0.227	-1.443 (4.417)	-4.185	0.873	0.55	0.44	small
Non-dominant knee int/ext ^{n}	15.502 (7.136)	12.416	18.588	16.716 (8.390)	12.402	21.030	0.62	0.16	small
Non-dominant knee flex/ext	17.811 (9.070)	14.275	21.990	14.932 (4.993)	12.999	17.501	0.20	0.38	small
Non-dominant ankle abd/add ^{n}	-4.668 (9.064)	-8.588	-0.749	-0.512 (7.24)	-4.235	3.210	0.13	0.50	medium
Non-dominant ankle eversion/ inversion	30.085 (1.365)	26.093	30.381	29.315 (10.382)	19.184	28.483	0.00	0.26	small
Non-dominant ankle plantiflex/ dorsiflex	1.207 (13.600)	-3.389	4.717	2.114 (8.864)	0.026	7.385	0.36	0.41	small

Table notes: n =number of participants ; SD = standard deviation; IQR = interquartile range; ^{n} =normally distributed data; p values calculated using Mann Whitney U test and T tests depending on normality.



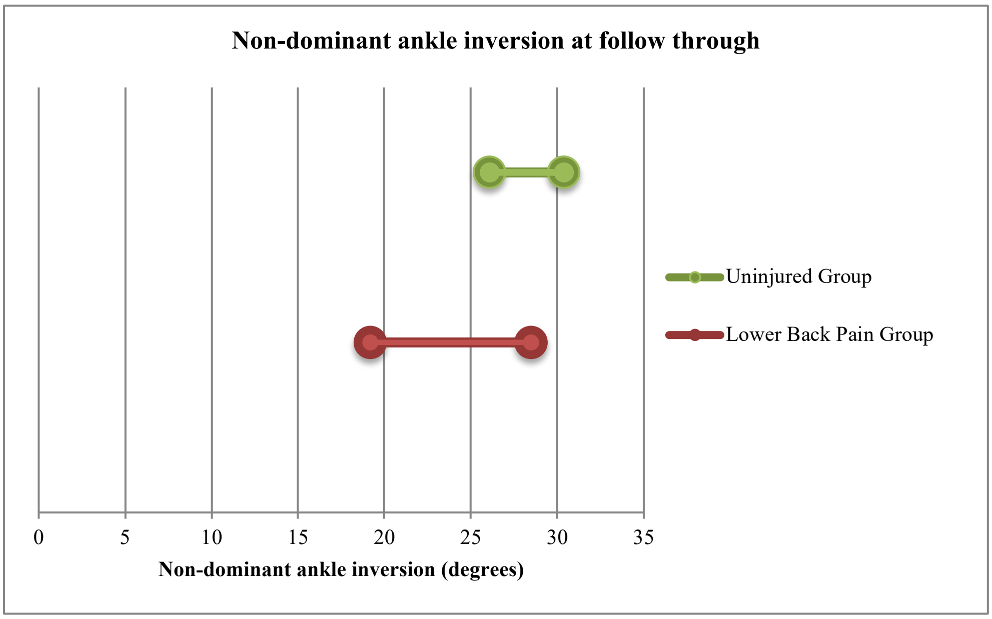
Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.15: Graph representing the difference in 95% confidence intervals for the degree of dominant hip adduction/abduction at follow through between the uninjured group and the lower back pain group at baseline assessment



Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.16: Graph representing the difference in 95% confidence intervals for the degree of dominant ankle adduction at follow through between the uninjured group and the lower back pain group at baseline assessment



Graph notes: The scale chosen for the x-axis is divided into 5 degree intervals, which represents the minimum clinically significant interval.

Figure 3.17: Graph representing the difference in 95% confidence intervals for the degree of non-dominant ankle inversion at follow through between the uninjured group and the lower back pain group at baseline assessment

3.3.3 Performance and training

There was no difference in order of merit, handicap, carry distance, roll, total distance, ball speed, club head speed, putting practice per week, short game practice per week, range practice per week, flexibility training per week, postural training per week, weight training per week, cardio training per week and rounds of golf per week amongst golfers who did develop lower back pain and the uninjured group (Table 3.6). It should be noted that not all the golfers played in the yearly order of merit and that one of the Flightscope recording did not record. It should be noted that one participant completed only 17 weeks of his training log and declined to complete any further training logs. He was, however, prepared to continue to be contacted for his injury monitoring. An average training for this participant was provided for week 18- week 26.

Table 3.6: Table to compare lower back pain group versus uninjured group's confounding variables in terms of training and performance

Variable	Uninjured group				Lower back pain group				Comparison between groups	
	n	Mean (SD) or Median (IQR)	95% CI Lower	95% CI Upper	n	Mean (SD) or Median (IQR)	95% CI Lower	95% CI Upper	P value	Effect size
Order of merit (place)	16	21.5 (26)	15.54	30.71	10	19 (32.25)	12.03	35.97	0.94	0.49
Handicap (score)	23	1 (3)	1.15	3.55	17	2 (2.5)	1	3	0.93	0.30
Carry distance (metres)^{n}	23	216.26 (19.70)	207.74	224.78	16	218.07 (22.69)	205.98	230.16	0.79	0.09
Roll distance (metres)	23	12.05 (10.97)	10.55	21.66	16	18.55 (22.20)	13.02	30.20	0.29	0.40
Total distance (metres)^{n}	23	232.32 (15.43)	225.64	238.99	16	239.64 (20.32)	228.82	250.46	0.21	0.42
Ball speed (km/hr)	23	238.80 (13.9)	219.1	244.79	16	240.26 (22.07)	217.98	247.43	0.97	0.49
Club head speed (km/hr)	23	177.40 (9.6)	164.7	183.34	16	175.32 (13.8)	160.77	181.23	0.25	0.39
Putting practice per week (hr/week)	23	3.96 (6.63)	3.1	6.78	17	3.08 (3.12)	2.26	3.97	0.37	0.41
Short game practice per week (hr/week)	23	4 (6.65)	3.31	6.93	17	2.94 (3.44)	2.12	4.07	0.19	0.38
Range practice per week (hr/week)	23	7 (7.63)	5.22	10.16	17	3.33 (4.54)	2.4	7.37	0.06	0.32
Flexibility training per week (hr/week)	23	1.15 (2)	0.97	2.43	17	0.92 (1.13)	0.69	1.44	0.50	0.43
Postural training per week (hr/week)	23	0.65 (1.27)	0.37	1.4	17	0.2 (0.42)	-0.22	1.69	0.45	0.43
Weight training per week (hr/week)	23	2 (2.94)	1.43	4.32	17	1.81 (2.69)	0.93	2.4	0.46	0.43
Cardio per week (hr/week)	23	1.65 (2.12)	1.39	3.08	17	1.25 (0.96)	0.78	1.59	0.07	0.33
Rounds of golf played per week (rounds per week)^{n}	23	2.87 (1.35)	2.29	3.45	17	2.53 (1.38)	1.82	3.23	0.43	0.25

Table notes: n =number of participants ; SD = standard deviation; IQR = interquartile range; ^{n} =normally distributed data; p values calculated using Mann Whitney U test and T tests depending on normality or type of data.CI were represented in the table as calculated, but it should be noted that the minimum lower CI for all variables, except for handicap is zero.

3.3.4 Baseline anthropometrics, medical history and lower back pain history

There was a difference in body mass (p=0.02; effect size =0.77) and BMI (p=0.05; effect size =0.64) between the uninjured and the group with back pain. The back pain group had a lower body mass and BMI than the uninjured group (Table 3.7). The difference in confidence intervals between the uninjured group and the lower back pain group is clinically meaningful as a large percentage of the uninjured groups confidence interval was above the recommended BMI of 25 (Ashwell, 2011) whereas all of the lower back pain group's confidence interval for BMI was below the recommended BMI of 25. This clinically meaningful difference combined with the high effect size indicates that this variable may have influenced the findings of this study.

There was no difference between the lower back pain and uninjured groups in terms of their age and height (Table 3.7).

Table 3.7: Differences between group's baseline anthropometrics

Variable	Uninjured group n=23			Lower back pain group n=17			Comparison between groups at baseline	
	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper CI	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper CI	P value	Effect size
Age (years)	19 (3)	18.46	20.59	21 (3.5)	18.8	21.55	0.32	0.40
Height (meters)^n	1.8 (0.08)	1.77	1.84	1.78 (0.05)	1.75	1.8	0.25	0.39
Body mass (Kg)^n	82.01 (12.82)	76.47	87.56	72.42 (11.79)	66.36	78.49	0.02	0.77
BMI (Kg/m^2)^n	25.24 (3.57)	23.69	26.78	22.94 (3.52)	21.13	24.76	0.05	0.64

Table notes: n =number of participants ; SD = standard deviation; IQR = interquartile range; ^{n} =normally distributed data; p values calculated using Mann Whitney U test and T tests depending on normality or type of data..

There was no difference between the lower back pain group and the uninjured group in terms of history of smoking, vascular conditions, respiratory conditions, cancer, musculoskeletal conditions, gastrointestinal conditions or lumbar spine injury in the six months prior to taking part in the study (Table 3.8).

In terms of prior lower back pain:

- Thirteen golfers had experienced lower back pain in the month prior to inclusion.
- Five golfers had missed golf training days as a result of lower back pain.
- Twenty-one training days were missed in total as a result of lower back pain.
- Three golfers missed training as a result of prior lower back pain
- Reasons given included: poor posture, overtraining, S-shaped spine, swing faults, bending, insidious onset, carrying of golf bag, playing rugby, muscle spasm, tiredness and high swing speed.
- Numerical pain score out of 10 mean (SD)= 5(2)
- Seven golfers saw a physiotherapist for their lower back pain
- One golfer saw his general practitioner (GP) for his lower back pain.
- The golfers who saw a physiotherapist or GP reported being diagnosed with: muscle spasm and poor vertebral alignment (physiotherapy diagnosis for one golfer).
- The golfers had undergone the following treatments: strapping, RICE (rest, ice, compression, elevation techniques), massage, physiotherapy, strapping, anti-inflammatory topical plasters and foam rolling

It should be noted that four golfers from the uninjured group declined to complete the medical screening and injury history questionnaire and two golfers from the uninjured group only part completed their medical history and injury history screening. One golfer from the lower back pain group only part completed his medical screening and injury history questionnaire.

Table 3.8: Comparison of baseline health history and history of injury between the lower back injured group and the uninjured group

Variable	Un-injured group total n assessed	Un-injured group n(%) with condition or health risk factor	Lower back pain group total n assessed	Lower back pain group n(%) with health condition or risk factor	p value at baseline	Odds ratio (did not have condition/ did have condition)	Relative risk for back pain	Effect size for between group difference
Participants who smoked at least 100 cigarettes in their lifetime	19	9 (47.37%)	17	7 (41.37%)	0.75	1.29	0.87	0.06
Participants with a history or diagnosis of any vascular condition	19	1 (5.26%)	16	1 (6.25%)	1	0.83	1.1	0.02
Participants with a history or diagnosis of a respiratory or lung condition	17	5 (29.41%)	16	3 (18.75%)	0.68	1.81	0.72	0.12
Participants with a history of cancer	19	1 (5.26%)	16	1 (6.25%)	1	0.83	1.1	0.02
Participants with a musculoskeletal condition	19	1 (5.26%)	16	3 (18.75%)	0.31	0.24	1.79	0.02
Participants with a history or diagnosis of a neurological condition	19	3 (15.79%)	16	2 (12.5%)	1	1.31	0.86	0.05
Participants with a history or diagnosis of a gastrointestinal conditions	19	3 (15.79%)	16	2 (12.5%)	1	1.31	0.86	0.05
Participants with a history of Lx injury in the last 6 months	19	8 (42.11%)	16	9 (56.25%)	0.51	1.77	0.75	0.14

Table notes: n =number of participants ; p values were calculated using Chi squared or fishers exact tests.

3.4 Discussion

3.4.1 Discussion of results

The incidence of lower back pain in this study was 41% and is comparable to that reported by Smith et al. (2018). They stated that longitudinal studies on young elite golfers showed an injury incidence of 57.1% over a one year monitoring period. The reduced incidence of lower back pain in this study may be due to the reduced monitoring period (six months as opposed to one year). Nonetheless, this lower back pain incidence is concerning, especially as it is occurring in young golfers and lower back pain has been shown to have a significant impact on later years lived with disability (Vos et al., 2012).

Only differences that were considered clinically meaningful will be discussed in this section. See the results section (section 3.3) for an explanation of which results were considered clinically meaningful.

At set up the injured group had reduced squatting on the dominant leg, as evidenced by the reduced flexion seen in the non-dominant knee and reduced dorsiflexion in the non-dominant ankle. During the backswing, golfers who had a reduced squat on the non-dominant leg had an increased risk of developing lower back pain. This was substantiated by the reduced knee and ankle flexion in the injured group compared to the uninjured group. During the backswing the injured golfers also had increased dominant hip adduction. This may indicate a weakness of the dominant hip primary abductors. The primary hip abductors include the *gluteus medius* muscle, *gluteus minimus* muscle, a small portion of *gluteus maximus* and the *tensor fascia lata* (Neumann, 2010). It is important to note that there was no difference in X-factor rotation between those that developed lower back pain and those that did not. This is contrary to the current view that increased X-factor is a probable mechanism of lower back pain in elite golfers.

At impact, the lower back pain group had increased dominant knee flexion. This would suggest that the injured group utilised potential energy by dropping their centre of mass on the dominant side, thereby converting potential energy into kinetic energy. This would have resulted in a unilateral dominant side compressive force.

The crunch factor is often cited as a possible mechanism of lower back pain in golfers (Smith et al., 2018). The crunch factor is a function of lumbar spine lateral flexion and angular velocity at impact (Cole and Grimshaw, 2014). In this study there was no difference in trunk lateral flexion at impact between those that developed lower back pain and the uninjured group. This would imply that this element of the crunch factor, namely, trunk lateral flexion, was not associated with developing lower back pain. This finding is consistent with pooled analysis performed by Smith et al. (2018) that showed that trunk extension and active trunk motion during the swing was not associated with lumbar spine injury in elite golfers.

At follow-through, the lower back pain group's dominant hip was in adduction whereas the uninjured group's hip was in abduction. As previously mentioned, this may be indicative of poor control of the primary hip abductors on the dominant side.

3.4.2 Training implications for elite golfers

The findings of this study highlight the usefulness of 3D kinematic analysis in order to identify golfers at risk of developing lower back pain. We would cautiously recommend that elite golfers should have: i) at set up: at least four degrees of non-dominant ankle dorsiflexion; ii) at the top of the backswing: at most 6 degrees of dominant hip adduction; at least 43 degrees of non-dominant knee flexion; at least 22 degrees of non-dominant ankle flexion; iii) at impact dominant knee flexion should be at most 29 degrees; iv) at follow through: at least 2 degrees of dominant hip abduction. These values were based on the 95% confidence interval for the uninjured group. These values should be noted with caution, as this is a small sample size. These values could initially be used as a guide and then refined or confirmed by future studies.

3.4.3 Clinical Implications

An application of the data presented here may be to suggest techniques to target the deficiencies associated with lower back pain found in the present study. These suggested interventions could then be tested in intervention studies to see if they reduce lower back pain incidence. Accordingly, if an elite golfer has reduced non-dominant knee flexion at set up and during the backswing, exercises such as single leg squats on the non-dominant leg may improve his ability to maintain a more non-dominant leg flexion set up and during the backswing.

If a golfer has increased dominant leg adduction during the backswing or the follow through, strengthening of primary hip abductors on the dominant side may help to correct this. Exercises such as single leg squats on the dominant side with the other leg at 90 degrees flexion have been shown to recruit *gluteus medius* (Olivier et al., 2019). It is best to perform these dominant leg hip abductor strengthening exercises in standing as this will help facilitate transference of motor learning (Rosalie and Müller, 2012).

If a golfer has good control and strength during a single leg squat on both the dominant and the non-dominant side, yet he still has reduced non-dominant squat at set up or during the backswing and/or increased dominant leg adduction during the follow through, then the problem may not be present in his strength or ability to perform the movement, but in his motor learning. Improved swing techniques can be taught using teaching aids or visual feedback (such as video analysis) to facilitate non-dominant knee and ankle dorsiflexion during the backswing, reduce dominant knee flexion during impact or increase dominant hip abduction during the backswing and/or follow through.

3.4.4 Future research

The associations found in this study should be tested using intervention studies to establish if they were merely associations with injury or possible mechanisms of lower back pain. Examples of such intervention studies may include: interventions aimed at facilitating non-dominant knee and ankle flexion during the backswing, reducing dominant knee flexion during impact, increasing dominant hip abduction during the backswing or during follow through. These intervention studies should be directed at young elite male golfers who have screened as at a high risk of developing lower back pain based on their kinematics (see training implications for elite golfers section 3.4.2).

3.4.5 Limitations

The assessment was performed outdoors. Weather conditions such as wind speed may have influenced Flightscope measurements, despite these having been taken within the first few centimetres of ball contact. This setting was chosen above a laboratory setting as it was felt that this would reduce the anxiety that junior golfers may experience during the assessment.

There were differences in baseline body mass and body mass index, which may have affected the results. This study showed an association between reduced body mass index and increased injury risk. This is interesting as the uninjured group had a BMI above the recommended BMI of 25 (Ashwell, 2011). This requires further investigation and the health risks associated with a BMI above 25 should be kept in mind when considering interventions aimed at addressing this risk factor in young elite male golfers. In terms of how the differences between groups in terms of BMI may have impacted the study results, it should be noted that there have been conflicting findings regarding the relationship between BMI and lower back pain in golfers. A systematic review by Smith et al. (2018) showed that golfers with lower back pain were heavier than golfers without lower back pain. In contrast a longitudinal study by Evans et al. (2005) on trainee professional golfers showed that golfers who with a BMI below 25.7kg/m2 were at greater risk of developing lower back pain. Whilst I acknowledge the possible impact that BMI had on the results, I would like to argue that it is not possible to account for all confounding variables and that it is not yet clear as to how BMI affects injury risk in golfers.

In terms of possible confounding variables related to medical history and injury history, there was no difference between groups in terms of medical history, but the lower back pain group did have a higher rate of lower back pain in the six months prior to assessment (56%) than the uninjured group (42%). It could be argued that the kinematic differences may be as a result of previous lower back pain episodes. It is true that spinal movement may change as a result of previous lower back pain episodes (van Dieën et al., 2017), but this does not detract from the observation that these particular movements were associated with developing lower back pain and were not as a result of pain at the time (they were not an antalgic movement pattern).

3.5 Conclusion

Golfers at risk of developing lower back pain had: reduced non-dominant leg squat at set up and at the top of the backswing, increased dominant knee flexion during impact and decreased dominant hip abduction during the backswing and during follow through. The incidence of back pain was 41%. This high incidence of lower back pain in a young golfing population highlights the need for more intervention studies to be performed into how to reduce lower back pain in elite golfers.

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LINKING PARAGRAPH BETWEEN CHAPTER 3 AND CHAPTER 4

The previous study highlighted the role that swing kinematics played in the development of lower back pain in elite golfers. The next lower back pain risk factor that requires attention, is the muscle recruitment strategies used during the swing. Lumbar musculature is responsible for much of the force generation for the swing action and for protecting the lumbar structure against injury (Gluck et al., 2008). The following study, 'Changes in muscle recruitment strategies during the golf swing is linked to developing lower back pain, a prospective longitudinal cohort study', aims to identify the muscle recruitment factors associated with developing lower back pain in young elite male golfers.

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CHAPTER 4: CHANGES IN MUSCLE RECRUITMENT STRATEGIES DURING THE GOLF SWING IS LINKED TO DEVELOPING LOWER BACK PAIN, A PROSPECTIVE LONGITUDINAL COHORT STUDY

Abstract

Background: Muscle activity has been linked to developing lower back pain in golfers. Cross-sectional research has been conducted comparing golfers with lower back pain to those without lower back pain. This is the first study that presents electromyographic (EMG) measurements prior to the development of lower back pain in young elite golfers.

Study design: Prospective longitudinal cohort study with one baseline time point.

Methods: Thirty four injury free elite golfers were included. Muscle activity from *latissimus dorsi, rectus abdominis, external oblique* and *erector spinae* muscles were recorded during 10 drive golf swings prior to injury (baseline assessment). Lower back pain, training and performance were monitored over a six month period.

Results: Seventeen participants (41%) developed lower back pain. At impact the lower back pain group's dominant *latissimus dorsi* (median (IQR)=11.22 (22.27)% MVC) was more active than the uninjured group (median (IQR)=5.25 (10.39)% MVC; p=0.01; effect size=0.25). Throughout the swing the lower back pain group's dominant *rectus abdominis* (mean (SD)=17.85 (9.92)% MVC) and dominant *latissimus dorsi* (median (IQR)=11.25 (10.89)% MVC) were more active than the uninjured group (dominan*t rectus abdominis* mean (SD)=11.47 (7.27)% MVC, p=0.04, effect size= 0.74; dominant *latissimus dorsi* median (IQR)=3.29 (7.13)% MVC, p=0.02, effect size =0.26).

Discussion: Elite golfers who developed lower back pain had higher dominant *latissimus dorsi* and dominant *rectus abdominis* activity during the swing compared to uninjured golfers. The increased muscle recruitment appears to be a centralised movement strategy and was not as a result of pain at the time of measurement.

4.1 Introduction

On average professional golfers have an injury prevalence of around 88%, with the majority of these injuries occur in the lower back (Cabri et al., 2009). The golf swing generates considerable strain on spinal architecture (McHardy et al., 2006). Lumbar musculature is responsible for much of the force generation for the swing action and for protecting the lumbar structure against injury (Gluck et al., 2008). Two systematic reviews on golf injury concluded that more research is required to determine the neuromuscular mechanisms of golf injury, including why some players are more likely to sustain injury than others (Smith et al., 2018, Cabri et al., 2009).

The first step in establishing lower back pain mechanisms is to identify the possible factors associated with back pain (Emery and Pasanen, 2019). Differences in muscle activity may be a potential factor, however, there is a lack of muscle activity data on the golf swing (Silva et al., 2013).

EMG (electromyography) studies have been performed on the lumbar spine musculature to establish differences in EMG activity between golfers who have already have lower back pain and those without lower back pain (Horton et al., 2001, Cole and Grimshaw, 2008a). These cross-sectional studies compared participants who had already developed lower back pain at the time of the EMG data collection to participants without lower back pain.

Horton et al. (2001) showed no difference in the abdominal muscle activity between six male professional golfers (mean (SD) age=29.2 (6.4) years) who currently had lower back pain and six professional male golfers who did not have lower back pain (mean (SD) age=32.7 (4.8) years). They did, however, observe a delay in *external oblique* muscle onset at the start of the backswing in golfers who had lower back pain (Horton et al., 2001). These EMG measurements were taken once the golfer had already developed lower back pain and therefore it is not possible to determine if these changes occurred as a result of the lower back pain (antalgic movement pattern) or were a risk factor associated with developing lower back pain. The golfers in the study conducted by Horton et al. (2001) were older than the golfers assessed in this research and the results may therefore not be generalizable to young elite male golfers.

In another study published by Cole and Grimshaw (2008a) it was shown that *erector spinae* muscle activation prior to initiation of the backswing occurred earlier in participants with lower back pain. They proposed that this earlier activation strategy may compensate for dysfunctional deep spinal stabilisers (Cole and Grimshaw, 2008a). Pooled analysis of studies conducted by Cole and Grimshaw (2008a) and Horton et al. (2001) showed no relationship between the timing of lead side *external oblique* onset relative to the backswing and lower back pain (Smith et al., 2018).

In a similar study Cole and Grimshaw (2008b) compared 12 male golfers who experienced lower back pain while playing golf to 18 asymptomatic controls. The golfers were divided into high handicap players and low handicap players. They showed that golfers with a low handicap and lower back pain had reduced *erector spinae* activity at the top of the backswing and at impact compared to the low handicap control group. They also observed that the low handicap and lower back pain group had greater *external oblique* activity throughout the swing. The low handicap golfers in this study were defined as those with a handicap below 12, which implies a mixture of elite and non-elite golfers, and therefore the results of this study cannot be generalised to elite golfers (Cole and Grimshaw, 2008b).

Silva et al. (2015) conducted a study that used non-linear machine learning to show that lead *biceps femoris* activity was the primary distinguishing factor between five golfers with lower back pain compared to seven golfers without lower back pain. This study was conducted on middle aged non-elite golfers (lower back pain group age mean (SD) = 58.6(10.9) years; lower back pain group handicap mean (SD) = 16(7.5); uninjured group age mean (SD) = 48.1(11.7) years; uninjured group handicap = 14.5(4.4)) and therefore also cannot be generalised to elite male golfers.

These studies provide valuable insight into the differences between golfers with lower back pain and those without lower back pain. They do not, however, reveal whether these changes in muscle recruitment occurred as a result of lower back pain or if they were associated factors in the development of lower back pain. To identify the precipitating lower back pain associations, baseline measurements should take place before the onset of pain.

The aim of this study was therefore to determine whether there is a difference in baseline spinal muscle recruitment, measured by EMG, between golfers that developed lower back pain and those that did not. To achieve this, a large sample of golfers were recruited for initial measurements and were monitored for lower back pain incidence. The incidental occurrence of lower back pain onset in a large proportion of participants allowed for retrospective comparisons to be made. The hypothesis for this study is that there is a difference in baseline EMG between golfers who develop lower back pain and those that do not. The observed retrospective baseline associations may point to possible lower back pain mechanisms in young elite golfers.

4.2 Materials and Methods

4.2.1 Study design and setting

This study was descriptive and followed a prospective longitudinal cohort design with one baseline measurement. For a flow diagram overview of the study please see Figure 4.1. It was conducted at a private golf academy in Johannesburg. All of the participants were identified as elite golfers by the academy's coaches prior to their participation. Data were collected from Feb 2017 to Jan 2018.

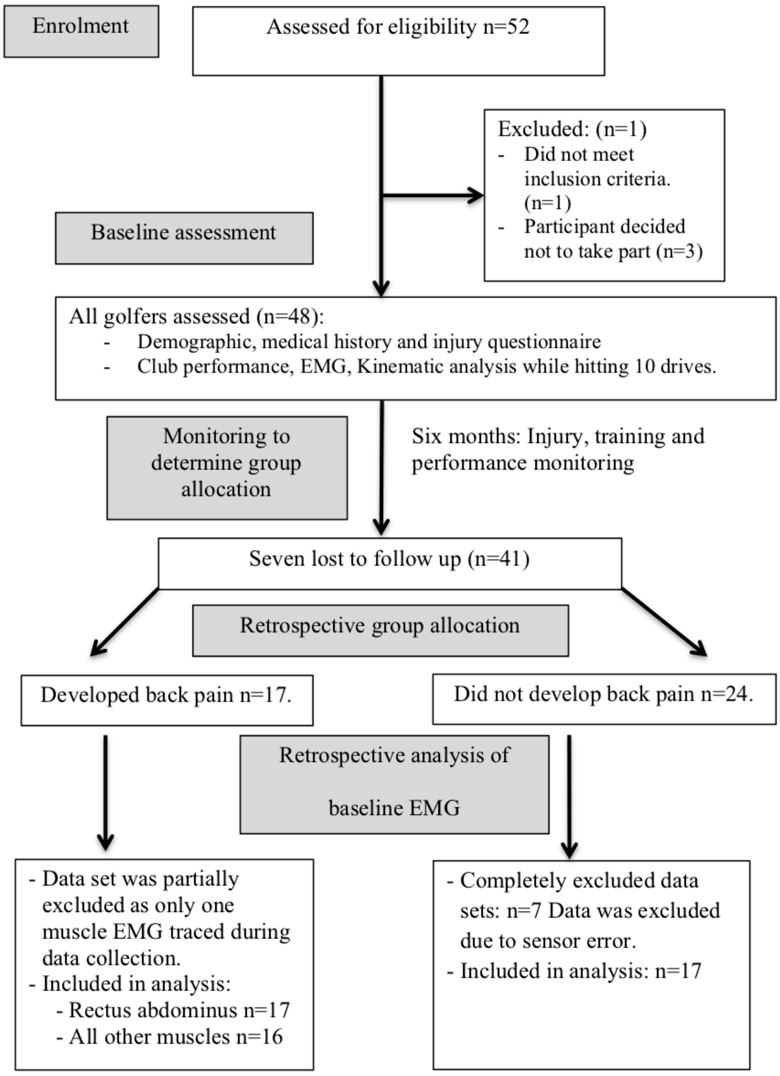

Figure 4.1: Study Cohort Diagram

4.2.2 Participants

All elite golfers at the golf academy were invited to take part in the study. This approach helped to avoid selection bias. To be included, participants needed to be injury free at the time of enrolment. 'Injury free' was defined as having no current pain or discomfort while performing activities of daily living or sport (including golf). Participants also needed to be identified as elite golfer by the academy's coaches, male and between the ages of 16-30 years. As some junior golfers (under 18 years old) were included in the study, a golf handicap cut-off was not applied. The mean (SD) handicap of the golfers selected was 2 (1.94). Male golfers were selected as only a small number of female golfers were enrolled at the academy at the time of the study. Female golfers were not included in the analysis as elite women golfers have been shown to have different swing mechanics to male golfers (Zheng et al., 2008). Golfers were excluded from the study if they had a known serious spinal or hip pathology. This included any participants who had undergone spinal or hip surgery, or had sustained spinal or hip fractures.

4.2.3 Ethics

Ethical clearance for this study was granted by the Ethics Committee of the University of the Witwatersrand, Johannesburg, South Africa. The study was performed according to the ethical standards and guidelines of the Declaration of Helsinki. Identifiable information was removed from the dataset and study numbers were allocated to each participant. The link between the study numbers and identifiable information was kept in an access controlled file to which only the researcher had access.

Written informed consent was granted by participants over the age of 18 years old. Written informed assent was granted by participants under the age of 18 years old and informed written consent was granted by their legal guardians for them to take part in the study. An information sheet was provided to the parents/guardians by the Golf School of Excellence. Minors (participants under 18 years old) were only approach for inclusion in the study if their parents/guardian had given permission for them to take part in the study

Minors were included in this study as a high volume of sports participation has been shown to increase the risk of developing lower back pain in childhood and adolescence (Sato et al., 2011). Research involving minors is permitted if it is therapeutic research in the best interests of the minor (Strode and Slack, 2015). The inclusion of minors in this study was approved by the University of the Witwatersrand's Human Research Ethics Committee

4.2.4 Instrumentation and Outcome Measures

Muscle activity of the dominant and non-dominant sides of each of the following muscles was measured: *rectus abdominis*, *external oblique*, *erector spinae* and *latissimus dorsi*. Anatomical descriptions of these muscles has been provided in Appendix 13 The Delsys Trigno wireless EMG system (Analogue and Digital Version) (Delsys Inc, Natick, Massachusetts, USA) which is able to sample up to 4000 samples per second. These muscles were chosen as they have been shown to be very active during the golf swing (McHardy and Pollard, 2005, Jobe et al., 1986, Marta et al., 2013). They are also large, superficial muscles, making surface EMG measurements possible. There is evidence to show that measurement of *latissimus dorsi* activity can be contaminated by *erector spinae* activity and therefore fine wire EMG is preferable when measuring *latissimus dorsi* activation (i.e. *lastissimus dorsi* surface EMG = *latissimus dorsi* muscle activity + *erector spinae* muscle activity) (Smith et al., 2018). However, we elected not to use fine wire EMG measurements as it was felt that the insertion of the wire may have altered swing biomechanics due to increased anxiety (especially amongst junior golfers) (McLenon and Rogers, 2019) and possible pain at the insertion site. This position is strengthened by the study performed by Lee et al. (2020) which showed that fine-wire insertion into the lumbar *multifidus* muscle resulted in reduced spinal extensor maximum strength performance in participants with persistent lower back pain. In order to account for contamination of the measurement of the *latissimus dorsi* muscle activity by the *erector spinae* muscle activity, independent *erector spinae* muscle EMG measurements were also made.

A Delsys Trigger Module (Delsys Inc., Natick, United States of America) was used to synchronize EMG and kinematic data collection. Simultaneous EMG, kinematic and club data were collected in order to determine the following during each participants 10 golf swings: the point of set up, the top of the backswing, impact and the end of follow through during each swing cycle. Set up was defined as the point just before the club head started to move into the backswing. The top of the backswing was defined as the point of zero velocity at the top of the backswing. Impact was defined as the point of club contact with the ball, which was determined by the vibration changes exhibited in the club at ball contact. The end of follow through was defined as the end point of the swing. At the end of follow through, the golfer's trunk should be facing the target in a well-balanced position (Hume et al., 2005). His lumbar spine will be in slight hyperextension and lateral flexion (Hume et al., 2005). Kinematic data was obtained using the Xsens inertial sensor motion analysis system (MVN Link Biomech system; Xsens Technologies B.V., Enschede, The Netherlands). The Xsens system consists of 17 wireless body motion trackers and one wireless club sensor (able to detect vibration in the club), which captures movement at a rate of 240 Hz. The Xsens system and Delsys Trigno system have been shown to yield equivalent data to that of other kinematic and electromyographic systems.(Poitras et al., 2019)

Club and ball performance was measured using the flight scope Flightscope X2 Elite system (Flightscope X2 Elite, Flightscope (Pty) Ltd, Stellenbosch, South Africa), a 3D Doppler tracking radar for golf specific application (Flightscope, Published 2021) as per Leach et al. (2017).

Golfer performance was ranked using the 2017 yearly order of merit score. Most of the golfers at the academy played in a weekly order of merit tournament, which contributed to a yearly order of merit score. The yearly order of merit score took into account true score, stroke average, fairways hit, greens hit, number of putts, scrambling (this is a score that measures how often a golfer scores par or better after missing a green in regulation) and ups and downs (a golfer is said to have achieved an 'up and down' if he takes only two strokes to get his ball into the hole when his ball is resting around the green or in a greenside bunker).

Lower back pain was monitored for six months. As most golf injuries are related to repetitive movement, the monitoring period needed to be long enough to capture these injuries. At the same time the injury needed to be related to the swing pattern at the time of assessment. The golfers included in this study were enrolled at a golf academy and in the process of refining their swing pattern. It was felt that a six-month monitoring period would allow sufficient time to capture injuries related to repetitive movement, while at the same time ensuring that the golfers swing had not changed significantly from the time of initial assessment. Lower back pain injury was monitored through prompted self-reporting. ‘Lower back pain’ was defined according to the European guidelines as ‘pain and discomfort that was localised below the costal margin and above the inferior gluteal folds, with or without leg pain’ (Burton et al., 2006). The lower back pain was self-reported and did not need to limit golf participation or score above a certain pain level. The golfers were initially offered a free physiotherapy assessment, which many of the golfers did not choose to attend. Reasons given for not attending the physiotherapy assessment were: travel and training commitments. The golfers were free to seek out physiotherapy or medical care if they developed lower back pain. As this was an observational study, no attempt was made to influence their decision to seek care. Once the findings of the study have been published by peer-review journals, the Golf academy will be sent a summary of the findings in simple English as well as some recommendations regarding evidenced-based management of lower back pain. A recommendation will be made to the Golf Academy that they disseminate this information to all the golfers at the academy (current and past students).

4.2.5 Procedure

Anthropometric measurements (height and body mass) were taken for each participant. Each golfer also completed a demographic and health questionnaire, which included: full name, address, contact numbers, email address, golf handicap, history of smoking, past medical history and incidence of back pain in the past six months. Past medical history questions established if the golfers had a diagnosis or history of: i) vascular conditions including angina pectoris, coronary artery disease, arrhythmia, blood pressure problems, congestive cardiac failure, clotting abnormalities, TIA or stroke, heart attack; ii) respiratory conditions including exercise induced asthma, asthma, emphysema or chronic bronchitis; iii) cancer; iv) musculoskeletal conditions such as osteoporosis, chronic lower back or neck pain, rheumatoid arthritis, osteoarthritis or fibromyalgia; v) neurological conditions such as anxiety, depression, multiple sclerosis, Polio, Parkinson’s, epilepsy, seizures, or Alzheimer's; vi) gastrointestinal conditions such as stomach or duodenal ulcer, Irritable Bowel Syndrome, gastric acid reflux, diverticulitis or gall bladder problems. Due to the sensitive nature of the medical screening questions, golfers were permitted to omit questions they did not want to answer or to forgo the medical screen. Giving the participants the option to omit part or all of the medical screening questionnaire was an important ethical consideration for this study. Despite this option being given, the majority of participants agreed to complete it.

Standard abrasion and swab techniques were used prior to EMG sensor placement with double sided tape. The following placement positions were used (figure 4.2): i) *rectus abdominis* 3cm lateral to the umbilicus (in the centre of the muscle belly) (McGill et al., 1996); ii) *external oblique*: 15 cm lateral to the umbilicus; iii) *erector spinae:* 1cm lateral to the spinous process at L5; iv) *latissimus dorsi*: lateral to T9 over the muscle belly (Vera-Garcia et al., 2010) (Figure 4.2). After EMG placement, a test muscle contraction was performed to confirm good position and skin-electrode contact.

Figure 4.2: EMG placement

The participant then performed a maximum isometric voluntary contraction (MVC) for 3-5 seconds. Three MVC measurements per muscle were recorded with a two minute rest period between each MVC recording. There is much debate regarding the position used to capture maximum voluntary contractions for trunk musculature and no single test has been shown to illicit maximum muscle activity (Vera-Garcia et al., 2010). For this reason although specific tests were performed in order to illicit a maximum voluntary contraction for each muscle, all eight EMG electrodes continued to record throughout all the MVC tests and the movement that produced the highest MVC was used. This is in line with the recommendations made by Vera-Garcia et al. (2010). The tests chosen for the MVC were based on the Seniam guidelines were possible. When not possible they were based on the muscles primary function. The primary function of the *rectus abdominis* muscle is to compress the abdominal content, flex the vertebral column and tense the abdominal wall (Drake et al., 2009), therefore to obtain *Rectus abdominis* MVC the golfer sat in crook (sitting with feet facing forwards, knees and hips flexed) with his trunk slightly extended with his feet supported. He was asked to attempt to perform a sit up against resistance applied by the research assistant. When acting on one side, the *External oblique* laterally flexes the trunk towards the same side and turns the anterior part of the abdomen towards the opposite side, therefore to obtain *External oblique* MVC the golfer sat in a crook with his trunk slightly extended and his feet supported. He then rotated his thorax. He was then asked to attempt to rotate back to midline while the research assistant applied resistance. Seniam recommends that the clinical test for erector spinae is to lift the trunk from the prone position (Seniam, 2021), therefore in order to obtain *erector spinae* MVC the golfer was placed in prone and asked to extend his back while the research assistance applied a counter resistance. The primary function of *latissimus dorsi* is adduct, extend and medially rotate of the upper limb (Drake et al., 2009). As such *latissimus dorsi* MVC was obtained with the golfer position in prone (lying on his stomach). The golfer was then asked to extend his shoulder and keep his elbow straight while a counter resistance was applied by the research assistant. All MVC were performed at approximately mid muscle length.

With the EMG sensors in place, the golfers were then fitted with the Xsens inertial sensor motion analysis system. A Delsys Trigger Module (Delsys Inc., Natick, United States of America) was used to synchronize EMG and kinematic data collection, while the specified frequency filters were used to reduce any influence of high frequency variations in EMG signals. With the Flightscope monitor set up, the golfers approach their usual driving range and were asked to hit the golf ball 10 times with their driver.

They were asked to hit the ball as they would during a competition where both distance and accuracy were important. This set up allowed for simultaneous EMG, kinematic, club performance and ball flight performance measurements to take place.

Following the initial assessment, training and injury were monitored for a period of six months. Golfers were asked to immediately contact the research team if they sustained any injury. Participants were contacted weekly to ascertain if they had developed lower back pain or not. Training was monitored weekly through a training log, which the golfer was expected to fill in weekly. If the golfer forgot to complete the log, the primary researcher contacted the golfer via telephone and completed the log on behalf of the golfer. The training log monitored time spent putting, practicing short game, range practice, flexibility training, postural training, weight training, cardiovascular training, and number of rounds of golf played. The training log was recorded in Microsoft Excel and analysed to determine if there were training differences between the injured and non-injured golfers.

4.2.6 Data reduction and Analysis

The raw EMG data were filtered using Delsys EMG works software (Delsys Inc., Natick, USA) (Butterworth four order filter; bandpass ripple of 3dB; attenuation of 40dB; bandpass; first corner frequency of 20 Hz; second corner frequency of 500 Hz). These data were then normalised to maximum voluntary contraction (MVC) and was presented as root mean . The swing events (set up, top of the backswing, impact and follow through) were determined for each golfer using the kinematic data. The corresponding muscle activation (%MVC) at each swing event was recorded. The muscle activation at each swing event for each golfer was then averaged so that each participant had one average muscle activation (%MVC) at each swing event for each muscle (This information was used in the analysis presented in Table 4.2-4.3).

$$AVE\ MVC = \frac{\sum \%MVC\ at\ each\ swing\ event_i}{Total\ number\ of\ swings}$$

AVE MVC= Average muscle activation at each swing event

The average muscle activation at each swing (%MVC) was also averaged to a unity average (average muscle recruitment throughout the swing) as adapted from Yang and Winter (1984) and presented as a unity average (This information was used in the analysis presented in Table 4.4). The unity average for each muscle for each golfer was calculated according to the following equation:

$$Unity\ average = \frac{AVE\ MVC\ SU + AVE\ MVC\ BS + AVE\ MVC\ IM + AVE\ MVC\ FT}{4}$$

Two participants were left-handed and played with a left-handed swing. For this reason EMG measurements were recorded as dominant and non-dominant, rather than left and right (e.g. EMG measurements for dominant *latissimus dorsi,* is the EMG recording for left *latissimus dorsi* in the left-handed golfer). The Flightscope data was averaged over10 swing cycles.

4.2.7 Statistical analysis

Data was analysed using Statistica® Version 12 (StatSoft Inc., Tulsa, USA). Categorical data (Table 4.7) was analysed using Pearson Chi Squared test or the Fisher's Exact Test (Kim, 2017) . Phi (φ) and risk ratio were used to describe the effect size of categorical data when there were two by two contingency tables (Kim, 2017, Pautz et al., 2018a).

$$\varphi = \sqrt{\frac{x^2}{n}}$$

Cramer's V was used to calculate effect sizes of categorical data on larger tables and was interpreted according to the following table (Table 4.1) (Kim, 2017). Cramer's V is calculated as:

$$V = \sqrt{\frac{x^2}{n-df}}$$

n is the total number of observations. df is the degrees of freedom which is calculated by $(r-1)(c-1)$. Here, r is the numbers of rows of the contingency table and c is the numbers of columns of the contingency table (Kim, 2017).

Table 4.1: Interpretation of effect size for Cramer's V

Degree of freedom	Small	Medium	Large
1	0.1	0.3	0.5
2	0.07	0.21	0.35
3	0.06	0.17	0.35
4	0.04	0.13	0.22

Continuous and ranked data was tested for normality (Table 4.2-Table 4.6). Normality was determined by dividing kurtosis score by its standard error and by dividing skew by its standard error (z score) (Kim, 2013). If the z score was between -1.96 and 1.96, the data was considered to be normally distributed (Kim, 2013). The data that was normally distributed is marked with a superscript 'n' in the results tables (Table 4.2-Table 4.6). Normally distributed data were analysed using unpaired Student's t-tests was presented as means and standard deviations. Data that was not normally distributed and ranked data was analysed using the Mann Whitney U-test and was presented as medians and interquartile ranges.

In line with the American Statistical Association's statement on the use of p-values (Wasserstein et al., 2019), a dichotomised cut-off of p<0.05 was not applied to this research study. The term 'statistically significant' or any of its variants was not used, but p-values were reported as continuous quantities (Wasserstein et al., 2019), instead effect sizes, p-values, confidence intervals and minimum clinically meaningful difference were taken into consideration when showing a difference between groups in this paper (Pautz et al., 2018a, Pautz et al., 2018b).

Determining clinically meaningful difference for trunk EMG muscle activation was difficult to determine, as relatively small differences in spinal muscle recruitment can have large effects on spinal stability (Reeves et al., 2019). A contraction of only 2% MVC of the paraspinal muscles is sufficient to support the spine in an upright posture (Reeves et al., 2019). Therefore clinical relevance was kept in mind, but a cut off value for clinical meaningful difference was not applied to this study.

Effect sizes for data that underwent Student's t-tests was calculated and presented as Cohen's (d_s), where $d_s = \frac{X_1 - X_2}{\sqrt{\frac{(n_1-1)SD_1^2+(n_2-1)SD_2^2}{n_1+n_2-2}}}$ (Pautz et al., 2018b). Effect sizes for data that had undergone the Mann Whitney U-test was presented as probability of superiority ($P_{a,b}$), where $P_{a.b} = \frac{U}{n_a n_b}$ (Pautz et al., 2018a). Effect sizes for continuous and ranked data were interpreted as: small (≤0.2), moderate (≥0.5), and large (≥0.8) (Cohen, 1992).

Missing data was not replaced. If an average already existed for the participant (e.g. nine swing cycles, but not 10 swing cycles) then an average over nine swing cycles was calculated. This was mentioned in the analysis when it occurred. If no answer was provided to the medical screening questionnaire this was omitted from analysis.

As many variables were simultaneously tested, the risk of obtaining false positives was increased. In order to account for this a Bonferroni correction could be performed, but as this was a relatively small sample size, the Bonferroni correction would unfairly increase the probability of a false negative (VanderWeele and Mathur, 2019).

Biomechanical studies typically involve small sample sizes. This is largely due to the amount of time it takes to collect data for one participant. Instead of conducting a formal sample size to power the study, p values, confidence intervals, minimum clinically meaningful change and effect sizes were reported. This approach has been used in other biomechanical studies e.g. single leg squat study published by Olivier et al. (2019).

4.3 Results

4.3.1 Participants and incidence of injury

Forty-eight golfers met the inclusion criteria and attended the initial assessment. Seven of these golfers were later excluded from the study as they did not respond to the injury questions and/or complete their training logs and as such it was not possible to determine their training load and injury status. Of the remaining 41 injury-free golfers tested at baseline, 17 golfers developed back pain, while 24 golfers did not develop back pain over the six-month monitoring period. Fourteen golfers only had one episode of lower back pain. Episodes were measured in weeks. The first episode median(IQR)=2(4) weeks. Three participants had two episodes of lower back pain. The second episode mean(SD)=2.3(0.56) weeks. The total length of back pain episodes (first and second) median(IQR)= 2(5) weeks. Three participants reported lower back pain and lower limb pain at the same time. Fifteen participants continued to play golf despite having lower back pain. EMG data from seven participants from the uninjured group were excluded due to sensor error. It is possible that the sensor error occurred as a result of the high speed nature of the movement and the wide range of action. EMG data for one of the back-pain participants was incomplete and only non-dominant *rectus abdominis* was recorded. EMG data for only one swing cycle was captured for one of the back-pain participants. Overall, 34 participants data sets were analysed in the study. The mean (SD) or median (IQR) for the whole group's age = 19.88 years (2.68), height = 1.79 m (0.07), body mass =77.96 kg (13.58), and BMI = 24.25 kg/m^2 (3.77).

4.3.2 Baseline muscle activity

At baseline assessment, at the point of impact the lower back pain group's dominant *latissimus dorsi* muscle activation (mean (SD) 11.22 (22.27)% MVC) was six per cent higher than the uninjured group (mean (SD) 5.25 (10.39)% MVC; p=0.01, effect size =0.25) (Table 4.3). There is some overlap of confidence interval between the lower back pain group and the uninjured group. The confidence interval for the lower back group is also relatively large (Figure 4.3). This overlap of confidence intervals combined with the small effect size indicates that this difference should be viewed with caution.

At baseline assessment, muscle activity throughout the swing for the lower back pain group's dominant *rectus abdominis* muscle (mean (SD)= 17.85 (9.92)% MVC) was six per cent higher than the uninjured group (mean (SD)=11.47 (7.27)% MVC; p=0.04; effect size =0.74) (Table 4.4). There was some overlap of confidence intervals for the lower back pain group and uninjured group's dominant *rectus abdominis* muscle activation throughout the swing (Figure 4.4). The effect size for this difference was moderate and the mean difference at baseline was fairly high, therefore this difference should be viewed as a clinically meaningful difference.

At baseline assessment, muscle activity throughout the swing for the lower back pain group's dominant *latissimus dorsi* muscle (median (IQR)= 11.25 (10.89)% MVC) was eight per cent higher than the uninjured group (median (IQR)= 3.29 (7.13)% MVC; p=0.02; effect size=0.26) (Table 4.4). There was some overlap of confidence intervals for the lower back pain group and uninjured group for dominant *latissimus dorsi* muscle activation throughout the swing (Figure 4.5). This confidence interval overlap combined with the small effect size indicates that this difference should be viewed cautiously.

There were no other differences in baseline muscle activation between the back pain group and the uninjured group observed at set up, top of the back swing, impact or follow through (Table 4.2- Table 4.4).

Table 4.2: Table to compare lower back pain group versus uninjured group's percentage MVC at baseline for non-dominant *recuts abdominis*, dominant *rectus abdominis*, non-dominant *external oblique*, dominant *external oblique*, non-dominant *erector spinae* and dominant *erector spine*, non-dominant *latissimus dorsi* and dominant *latissimus dorsi* at set up and at the top of the backswing.

Muscle activity (% MVC)	Uninjured n=17			Lower back pain (n=17 non-dominant rectus abdominis, all other muscles n=16)			Comparison between groups at baseline	
	median (IQR) or mean (SD)	95% CI lower	95% CI upper	median (IQR) or mean (SD)	95% CI lower	95% CI upper	p-value	Effect size
Set up								
Non-dominant rectus abdominis	1.50 (1.50)	0.99	3.08	1.47 (1.37)	0.72	3.27	0.47	0.43
Dominant rectus abdominis [n]	1.35 (1.04)	0.81	1.88	1.31 (0.85)	0.86	1.76	0.91	0.04
Non-dominant external oblique	2.97 (3.30)	2.51	4.64	3.14 (1.85)	1.81	7.25	0.85	0.48
Dominant external oblique	3.05 (3.04)	2.23	4.73	3.12 (4.71)	2.17	5.02	0.93	0.49
Non-dominant erector spinae [n]	12.33 (8.13)	8.15	16.50	12.43 (6.77)	8.83	16.04	0.97	0.01
Dominant erector spinae	9.13 (9.41)	5.50	12.35	10.00 (9.25)	6.58	13.73	0.66	0.45
Non-dominant latissimus dorsi	3.14 (4.12)	2.41	4.87	2.14 (2.13)	1.74	3.41	0.33	0.40
Dominant latissimus dorsi	1.26 (3.64)	1.02	3.36	3.18 (3.31)	1.64	6.80	0.05	0.30
Top of the backswing								
Non-dominant rectus abdominis	7.18 (11.74)	6.25	18.41	11.51 (9.25)	6.98	23.76	0.61	0.45
Dominant rectus abdominis	21.13 (20.85)	13.58	30.20	30.46 (37.17)	22.19	52.07	0.10	0.33
Non-dominant external oblique	11.63 (7.11)	8.69	15.44	11.30 (9.23)	8.54	19.63	0.68	0.46
Dominant external oblique	28.28 (17.67)	20.39	33.18	25.22 (29.00)	18.48	42.88	1.00	0.50
Non-dominant erector spinae	22.95 (48.23)	17.30	66.80	23.94 (19.76)	16.30	31.36	0.71	0.46
Dominant erector spinae	28.92 (47.95)	15.27	86.23	30.37 (36.86)	7.40	123.41	1.00	0.50
Non-dominant latissimus dorsi	28.28 (31.66)	18.65	40.13	16.46 (34.04)	14.67	39.44	0.66	0.45
Dominant latissimus dorsi	6.12 (15.83)	4.29	23.26	15.41 (27.89)	12.53	29.32	0.06	0.40

Table notes: [n] indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations. Data not normally distributed presented as medians and interquartile range (IQR). p values were calculated using T tests and Mann Whitney U tests depending on normality.

Table 4.3: Table to compare lower back pain group versus uninjured group's percentage MVC at baseline for non-dominant *recuts abdominis*, dominant *rectus abdominis*, non-dominant *external oblique*, dominant *external oblique*, non-dominant *erector spinae* and dominant *erector spine*, non-dominant *latissimus dorsi* and dominant *latissimus dorsi* at impact and follow through

Muscle activity (% MVC)	Uninjured n=17			Lower back pain (n=17 non-dominant rectus abdominis, all other muscles n=16)			Comparison between groups at baseline	
	median (IQR) or mean (SD)	95% CI lower	95% CI Upper	median (IQR) or mean (SD)	95% CI Lower	95% CI Upper	P value	Effect size
Impact								
Non-dominant rectus abdominis	10.79 (13.33)	8.09	23.95	20.96 (32.83)	14.98	39.79	0.11	0.34
Dominant rectus abdominis ^{n}	19.38 (14.32)	12.02	26.74	29.75 (17.18)	20.59	38.90	0.07	0.66
Non-dominant external oblique	12.70 (10.32)	10.27	22.49	17.01 (23.33)	12.87	26.44	0.56	0.44
Dominant external oblique ^{n}	20.74 (11.90)	14.62	26.85	18.40 (7.54)	14.38	22.42	0.51	0.23
Non-dominant erector spinae	24.56 (45.55)	1.41	101.02	21.24 (31.91)	13.92	45.86	0.90	0.49
Dominant erector spinae	13.491 (15.89)	3.24	50.46	9.04 (39.10)	8.26	42.88	0.74	0.46
Non-dominant latissimus dorsi	11.97 (12.79)	9.27	18.19	7.00 (15.93)	4.43	22.56	0.40	0.41
Dominant latissimus dorsi	5.25 (10.39)	3.62	13.26	11.22 (22.27)	9.23	26.88	0.01	0.25
Follow through								
Non-dominant rectus abdominis	3.21 (5.06)	2.75	6.36	3.56 (5.94)	2.97	7.82	0.79	0.47
Dominant rectus abdominis	3.72 (2.61)	2.17	4.37	2.28 (2.49)	1.83	4.64	0.61	0.44
Non-dominant external oblique	5.67 (7.32)	5.07	10.36	6.92 (7.98)	5.56	10.77	0.79	0.47
Dominant external oblique	8.51 (8.02)	6.71	12.53	5.48 (5.48)	4.49	10.14	0.20	0.37
Non-dominant erector spinae	8.89 (23.17)	7.08	27.25	4.70 (10.51)	1.10	23.15	0.47	0.42
Dominant erector spinae	5.68 (6.31)	2.96	13.87	2.67 (2.83)	1.94	5.96	0.22	0.37
Non-dominant latissimus dorsi	22.98 (38.76)	13.30	32.90	5.41 (23.84)	6.07	22.05	0.18	0.36
Dominant latissimus dorsi	1.09 (1.68)	0.62	3.19	2.10 (3.64)	1.72	4.00	0.06	0.31

Table notes: ^{n} indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations. Data not normally distributed presented as medians and interquartile range. p values were calculated using T tests and Mann Whitney U tests depending on normality.

Table 4.4: Table to compare lower back pain group versus uninjured group's percentage MVC at baseline for non-dominant recuts abdominis, dominant rectus abdominis, non-dominant external oblique, dominant external oblique, non-dominant erector spinae and dominant erector spine, non-dominant latissimus dorsi and dominant latissimus dorsi throughout the swing (unity values)

Muscle activity (% MVC)	Uninjured n=17			Lower back pain (n=17 non-dominant rectus abdominis, all other muscles n=16)			Comparison between groups at baseline	
	median (IQR) or mean (SD)	95% CI lower	95% CI Upper	median (IQR) or mean (SD)	95% CI Lower	95% CI Upper	P value	Effect size
Unity Values								
Non-dominant rectus abdominis	7.04 (7.12)	5.06	12.41	11.43 (8.11)	7.48	17.59	0.15	0.35
Dominant rectus abdominis ^{n}	11.47 (7.27)	7.73	15.21	17.85 (9.92)	12.57	23.14	0.04	0.74
Non-dominant external oblique	9.56 (5.01)	7.32	12.54	9.93 (10.52)	7.82	15.40	0.71	0.46
Dominant external oblique ^{n}	15.15 (6.81)	11.65	18.66	15.00 (7.88)	10.80	19.20	0.95	0.02
Non-dominant erector spinae	24.04 (32.33)	10.44	50.94	15.50 (12.84)	12.59	26.55	0.68	0.46
Dominant erector spinae	12.65 (24.32)	9.79	37.68	13.52 (22.67)	7.76	44.77	0.96	0.49
Non-dominant latissimus dorsi	15.57 (18.31)	11.96	22.97	9.62 (16.82)	7.52	21.07	0.23	0.38
Dominant latissimus dorsi	3.29 (7.13)	2.84	10.31	11.25 (10.89)	7.65	15.38	0.02	0.26

Table notes: ^{n} indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations. Data not normally distributed presented as medians and interquartile range. Unity values were calculated as an average of the values from set up, top of the back swing, impact and follow through for each muscle. p values were calculated using T tests and Mann Whitney U tests depending on normality.

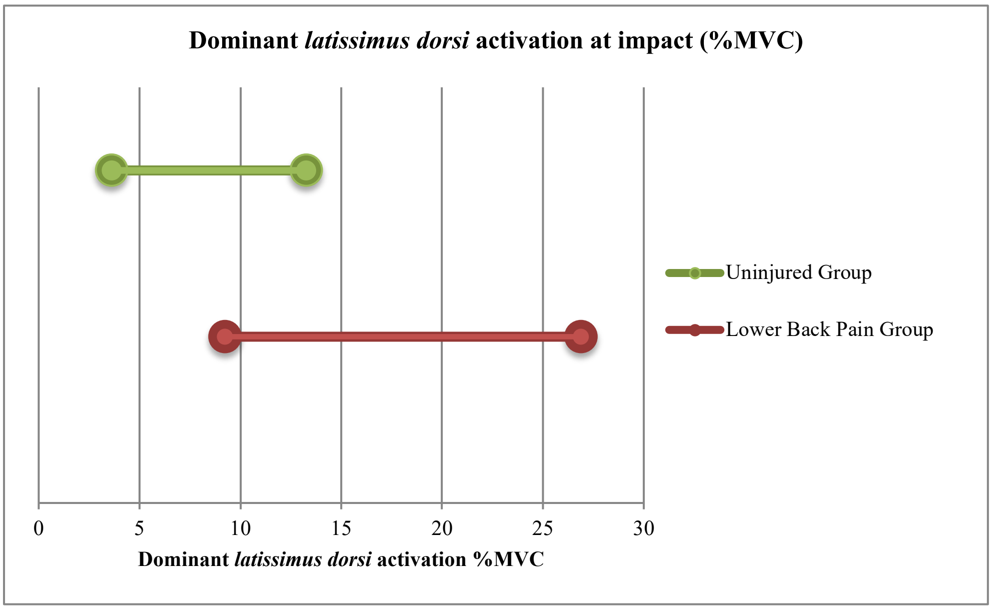

Figure 4.3: Graph representing the difference in 95% confidence intervals for dominant *latissimus dorsi* activation at impact during baseline assessment for the lower back pain group compared to the uninjured group

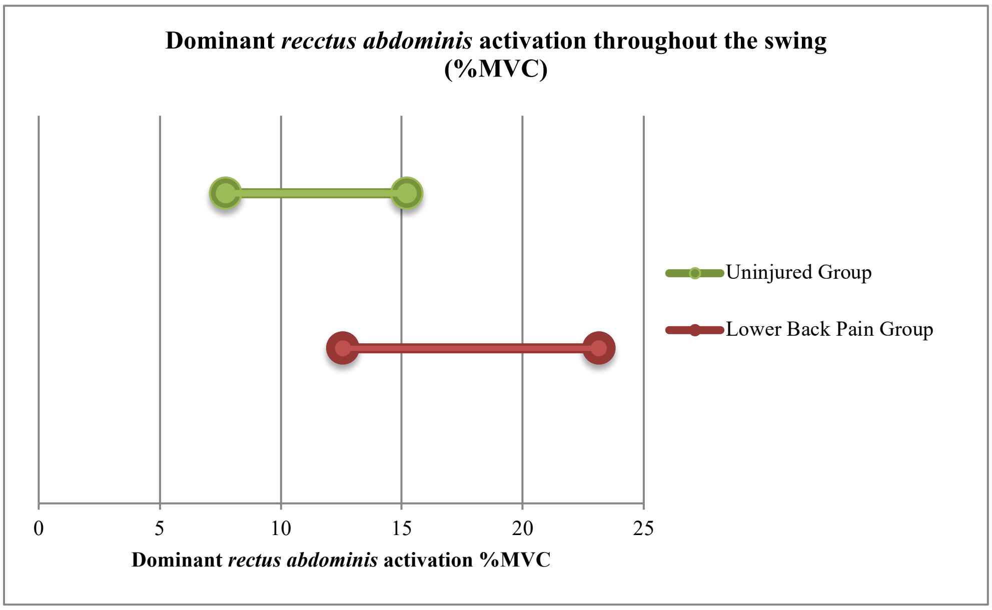

Figure 4.4: Graph representing the difference in 95% confidence intervals for dominant *rectus abdominis* activation throughout the swing during baseline assessment for the lower back pain group compared to the uninjured group

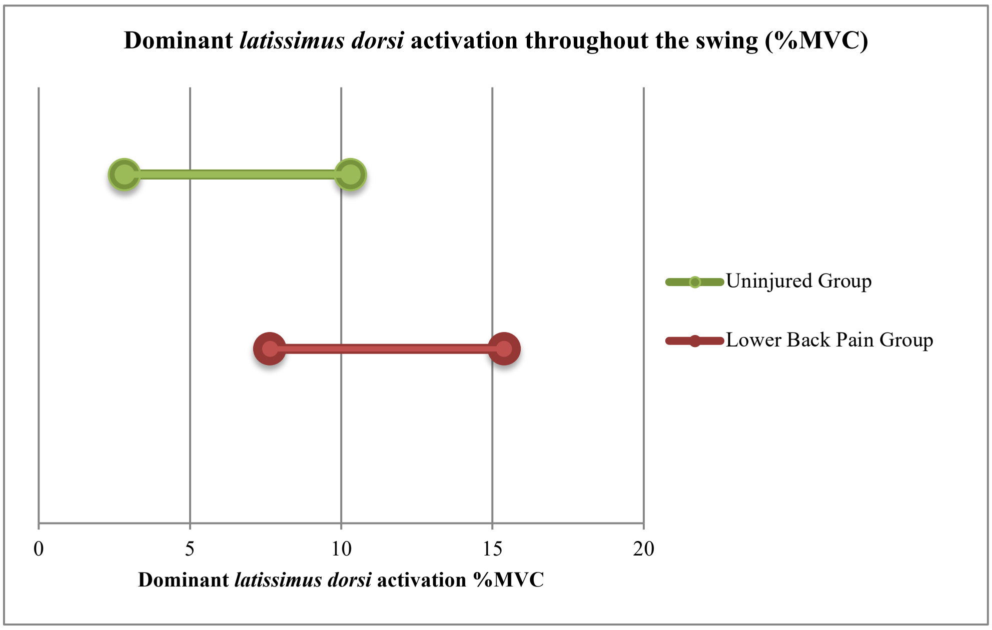

Figure 4.5: Graph representing the difference in 95% confidence intervals for dominant *latissimus dorsi* activation throughout the swing comparing during baseline assessment for the lower back pain group compared to the uninjured group

4.3.3 Performance and training

In terms of performance there was no difference between the uninjured and the lower back pain group's yearly order of merit or handicap (Table 4.5). In terms of training in the six months following the baseline assessment there was no difference between the uninjured and the lower back pain group's putting practice, short game practice, range practice, rounds of golf played, flexibility training, postural training, weight training, cardiovascular training, carry distance, roll, total distance , ball speed or club head speed (Table 4.5). It must be noted that one of the participant's Flightscope recording did not record during the assessment and was therefore not included in the analysis.

Table 4.5: Table to compare confounding training and performance variables means, standard deviations, medians, interquartile range between injured and uninjured groups

Confounding variable	Uninjured group				Lower back pain group				Between group comparison	
	n	Mean (SD) or median (IQR)	Lower CI	Upper CI	n	Mean (SD) or median (IQR)	Lower CI	Upper CI	p value	Effect size
Order of merit (position)	12	21.5 (19)	15.75	30.25	10	19 (32.25)	12.03	35.97	0.96	0.44
Handicap	17	1 (3)	1.13	3.57	17	2 (2.5)	1	3	0.89	0.40
Putting practice (hours per week)	17	3.96 (5.42)	2.46	6.35	17	3.08 (3.12)	2.26	3.97	0.54	0.44
Short game practice (hours per week)	17	4 (6.56)	2.73	7.28	17	2.94 (3.44)	2.12	4.07	0.32	0.39
Range practice (hours per week)	17	7 (6.43)	4.59	10.69	17	3.33 (4.54)	2.5	7.37	0.09	0.33
Rounds of golf played (per week) ^{n}	17	2.83 (1.42)	2.1	3.56	17	2.53 (1.38)	1.82	3.23	0.52	0.22
Flexibility training (hours per week) ^{n}	17	1.88 (1.83)	0.94	2.82	17	1.07 (0.74)	0.69	1.44	0.10	0.6
Postural training (hours per week)	17	0.65 (1.58)	0.3	1.68	17	0.2 (0.42)	-0.22	1.69	0.56	0.44
Weight training (hours per week)	17	2 (3.2)	1.05	4.92	17	1.81 (2.69)	0.93	2.4	0.59	0.44
Cardiovascular training (hours per week)	17	1.65 (2.06)	1.22	2.79	17	1.25 (0.96)	0.78	1.59	0.08	0.31
Carry distance (meters)^{n}	17	215.49 (17.1)	206.69	224.28	16	218.07 (22.69)	205.98	230.16	0.71	0.13
Roll (meters)	17	12.6 (11.65)	9.63	23.49	16	18.55 (22.2)	13.02	30.2	0.44	0.42
Total distance (meters)	17	233.1 (17.82)	224.64	239.38	16	241.26 (30.11)	228.81	250.46	0.28	0.39
Ball speed (km/hr)	17	239.81 (10.45)	236.72	246.49	16	240.27 (22.07)	217.98	247/43	0.54	0.43
Club head speed (km/h)	17	182.5 (10.08)	177.3	185.14	16	175.32 (13.8)	160.77	181.23	0.05	0.30

Table notes: ^{n} indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations. Data not normally distributed presented as medians and interquartile range. p values were calculated using T tests and Mann Whitney U tests depending on normality and data type.

4.3.4 Baseline anthropometrics, medical history and history of lower back pain

The lower back pain group's body mass (p=002; effect size= 0.88) and BMI (p=0.04; effect size = 0.73) were lower than the uninjured group. This difference in BMI had a large effect size for body mass and a medium effect size for BMI. The confidence interval for BMI for the uninjured group was largely above 25 kg/m^2 (CI=23.68-27.42 kg/m^2), which is considered overweight (Ashwell, 2011). The confidence interval for BMI for the lower back pain group was all below 25 kg/m^2 (CI=21.13-24.76 kg/m^2). This difference in BMI between groups was clinically meaningful and may have affected the outcome of this study. There was no difference in terms of age or height (Table 4.6).

Table 4.6: Table of anthropometrics at baseline[4]

Variable	Uninjured Group n=17			Lower back pain group n=17			Between group comparison at baseline	
	Mean (SD) or median (IQ)	Lower CI	Upper CI	Mean (SD) or median (IQR)	Lower CI	Upper CI	p value	Effect size
Age (years)	19.00 (3)	18.18	21	21 (3.5)	18.8	21.55	0.36	0.41
Height (m) ^{n}	1.81 (0.08)	1.77	1.85	1.78 (0.05)	1.75	1.80	0.18	0.47
Body mass (kg) ^{n}	83.49 (13.27)	76.66	90.31	72.42 (11.79)	66.36	78.49	0.02	0.88
BMI (kg/m^2) ^{n}	25.55 (3.64)	23.68	27.42	22.94 (3.52)	21.13	24.76	0.04	0.73

Table notes: ^{n} indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations. Data not normally distributed presented as medians and interquartile range. p values were calculated using T tests and Mann Whitney U tests depending on normality

[4] The reason for the low variance between Table 3.7 and Table 4.6 in this thesis, may indicate that the participants were fairly homogenous in body shape, which would be expected for elite athletes of a specific age range in a particular sport. E.g. young male elite gymnasts are likely to have similar body morphology.

In terms of baseline demographics, smoking and past medical history there was no difference between groups in terms of: smoking, vascular conditions, respiratory conditions, cancer history, musculoskeletal conditions, neurological conditions, gastrointestinal problems or history of lumbar spine injury in the last 6 months (Table 4.7). Although all the participants were injury free at the time of inclusion, 13 golfers had a history of lower back pain in the six months prior to assessment. Eleven of the golfers had experienced lower back pain in the last month. Five golfers reported having missed golf training due to lower back pain. Twenty-one golf training days in total had been missed due to lower back pain. Three golfers reported having missed golf tournaments due to lower back pain. Six golf tournaments in total had been missed due to lower back pain. Reasons given by the golfers for their previous lower back pain were: poor posture, spinal shape, swing faults, bending, tiredness, stiffness, overtraining, high swing speeds, insidious onset and sport. Mean (SD) pain intensity scored on the numerical pain scale for prior lower back pain = 5(2). In terms of management for their previous lower back pain: six golfers saw a physiotherapist; one saw their general practitioner (GP). Other treatment included: RICE (rest, ice, compression and elevation) techniques, strapping, non-steroidal anti-inflammatory plasters, foam rolling and stretching. Out of the six golfers who saw a GP or a physiotherapist for their previous lower back pain, three were diagnosed with muscle spasm and one with abnormal vertebral movement (physiotherapy diagnosis).

It should be noted that two participants from the uninjured group declined to complete the previous medical history and smoking questionnaire, and two participants from the same group only part completed the questionnaire. One participant from the lower back pain group only part completed his assessment.

Table 4.7: Comparison of baseline health history and history of injury between the lower back injured group and the uninjured group

Variable	No lower back pain group n	No lower back pain group n(%) with condition or health risk factor	Lower back pain group n	Lower back pain group n(%)with condition or health risk factor	p value for between group difference at baseline	Odds ratio	Relative risk for lower back pain	Effect size
Participants who smoked at least 100 cigarettes in their lifetime	15	6 (40%)	17	7 (41.18%)	1	0.95	1.02	0.01
Participants with a history or diagnosis of any vascular condition	15	0 (0%)	16	1 (6.25)%	1	NA	NA	0.18
Participants with a history or diagnosis of a respiratory or lung condition	13	3 (23.08%)	16	3 (18.75%)	1	1.3	0.89	0.05
Participants with a history of cancer	15	1 (6.67%)	16	1 (6.25%)	1	1.07	0.97	0.01
Participants with a musculoskeletal condition	15	1 (6.67%)	16	3 (18.75%)	0.32	0.31	1.56	0.18
Participants with a history or diagnosis of a neurological condition	15	2 (13.33%)	16	2 (12.5%)	1	0.13	0.96	0.01
Participants with a history or diagnosis of a gastrointestinal conditions	15	3 (20%)	16	2 (12.5%)	0.65	1.75	0.74	0.1
Participants with a history of Lx injury in the last 6 months	15	4 (26.67%)	16	9 (56.25%)	0.15	3.54	0.47	0.3

Table notes: As risk ratio =cumulative incidence in lower back pain group/ cumulative incidence non back pain group, it was not possible to calculate this for vascular conditions as no participants in the no back pain group had a vascular condition. p values calculated using Pearson Chi Square Test or Fishers Exact Test.

4.4. Discussion

4.4.1 Discussion of results

The lower back pain incidence was 41%. A high injury incidence is well established in young elite golfers. Evans et al. (2005) reported an 85.71% of incidence of lower back pain in young elite golfers over a 10 month monitoring period. The present study's incidence may have been reduced compared to Evans et al. (2005) due to the shortened monitoring period. Lower back pain injuries in golfers commonly include facet joint capsule trauma and internal disc disruption (Gluck et al., 2008). This large incidence in lower back pain over a six month period highlights the need for targeted lower back pain prevention strategies for this group of golfers.

The results of our study support the proposed hypothesis that there is a difference in baseline EMG between golfers who go on to develop lower back pain and those who do not. Golfers with greater activation of *dominant latissimus dorsi* muscle and dominant *rectus abdominis* muscle during the swing were at risk of developing lower back pain. When active *latissimus dorsi* and *rectus abdominis,* are able to exert a compressive force on the lumbar spine (Granata and Marras, 1995). The increased lumbar compression observed in injured golfers was unilateral in nature, as the increased muscle activation occurred only on the dominant side. This unilateral increase in spinal compressive load will add to the already high spinal load previously reported during the golf swing. Lim et al. (2012) reported that a golf swing produces a mean peak compressive load equivalent to six times bodyweight; a mean peak anterior load equivalent to 1.6 times bodyweight and a mean peak medial shear load equivalent to 0.6 times bodyweight.

It is interesting to note that in this study the golfers who went on to develop lower back pain had increased trunk muscle activity before the onset of pain. Changes in lumbar spine muscle recruitment patterns have been observed in patients with lower back pain and these include; greater co-contraction, redistribution of muscle activity and delayed postural control of deep trunk muscle (Devecchi et al., 2021). Van Dieën et al. (2019) proposes that there are two divergent motor control patterns that individuals can adopt after having developed lower back pain and these include: 'tight control' and 'loose control'. The 'tight control' motor adaptation involves increased co-contraction of the trunk musculature which results in reduced lumbar movement, but this improved control comes at the expense of increased spinal loading (Van Dieën et al., 2019). The 'loose control' motor adaptation involves reduced muscle activity in order to reduce spinal load, but this comes at the expense of spinal movement control(Van Dieën et al., 2019). The golfers who developed lower back pain appear to have adopted a 'tight control' movement strategy (Van Dieën et al., 2019). This is evidenced by the greater activation of dominant *latissimus dorsi* muscle and dominant *rectus abdominis* muscle during the swing.

The increased activation seen in dominant *rectus abdominis* in the lower back pain group is well supported by the moderate effect size and the mean difference of six per cent, which despite being small may be clinically relevant. Relatively small changes in spinal muscle recruitment can have large effects on spinal stability. To this end, a contraction of only two per cent MVC of the paraspinal muscles is sufficient to support the spine in an upright posture (Reeves et al., 2019).

The increased activation of dominant *latissimus dorsi* in the lower back pain group should be noted with caution, as the effect sizes of the differences were small, but in defence of the data presented regarding dominant *latissimus dorsi* activation: little is known about the minimum clinically relevant change for EMG measurements and the median change for dominant *latissimus dorsi* was six per cent at impact and eight per cent throughout the swing. As mentioned above this may well be a meaningful clinical change.

In terms of surface EMG *latissimus dorsi* muscle activity contamination by *erector spinae* muscle activity, the lower back pain group's surface EMG *latissimus dorsi* measurements was higher than the injury free group, but there was no difference between the groups *erector spinae* EMG measurements. Therefore according to the previously discussed equation (*lastissimus dorsi* surface EMG = *latissimus dorsi* muscle activity + *erector spinae* muscle activity), the increased muscle activity could be attributed to *latissimus dorsi* muscle activity.

It could be argued that the muscle recruitment differences may be as a result of previous lower back pain episodes. It is true that spinal muscle activation may change as a result of previous lower back pain episodes (Devecchi et al., 2021), but this does not detract from the observation that these particular muscle recruitment patterns were associated with developing lower back pain and were not as a result of pain at the time (they were not an antalgic movement pattern). It should further be noted that there was no difference between groups in terms of history of lower back pain.

4.4.2 Clinical implications

Reducing dominant *latissimus dorsi* and *rectus abdominis* muscle activation during the swing may be protective and might reduce the incidence of lower back pain in elite golfers. Future observation studies could be done into ways of offloading dominant *latissimus dorsi* and *rectus abdominis* e.g. muscle biofeedback training focused on teaching the golfer to reduce muscle activation of *latissimus dorsi* and *rectus abdominis* in specific positions during the swing. Intervention studies could then be performed comparing golfers who use the offloading techniques to those that do not. If lower back pain incidence reduces in the golfers who are taught the offloading techniques compared to those that do not, then increased dominant *latissimus dorsi* and *rectus abdominis* muscle activity during the swing could be confirmed as injury mechanism in young elite male golfers.

4.4.3 Limitations

Not all of the EMG data collected was analysed. This was due to sensor error.

In terms of baseline characteristics and baseline factors that could influence injury there was a difference in body mass and BMI between the injured and the uninjured group. The uninjured group had a higher BMI than the lower back pain group. This difference in baseline BMI should be considered as a possible confounding variable for this study.

In terms of how the differences between groups in terms of BMI may have impacted the study results, it should be noted that there have been conflicting findings regarding the relationship between BMI and lower back pain in golfers. A systematic review by Smith et al. (2018) showed that a golfers with lower back pain were heavier than golfers without lower back pain. In contrast a longitudinal study by Evans et al. (2005) on trainee professional golfers showed that golfers who with a BMI below 25.7 kg/m2 were at greater risk of developing lower back pain. It is also important to note that only BMI and body mass were considered different between the two groups. Other important confounding variables, which included training, medical history, history of lower back pain, club performance and overall performance were similar between the two groups. Whilst I acknowledge the possible impact that BMI had on the results, I would like to argue that as in real life scenarios, it is not possible to account for every confounding variable.

A second EMG assessment was not performed after injury and it is therefore not possible to tell if dominant *latissimus dorsi* and dominant *rectus abdominis* muscle activation continued to be higher in the lower back pain group compared to the uninjured group, after injury. This was outside the scope of this study. The purpose of this study was to identify baseline characteristics associated with the development of lower back pain. It should also be noted that EMG studies on golfers with injury have been performed (Horton et al., 2001, Cole and Grimshaw, 2008a) and comparisons to these findings can be made.

The study was conducted on young male elite golfers and therefore cannot be generalised to young female elite golfers.

4.5 Conclusion

Elite golfers who developed lower back pain had increased dominant *latissimus dorsi* and dominant *rectus abdominis* muscle activity during their golf swing. This may have resulted in increased dominant side spinal compression. The increased muscle recruitment of dominant *latissimus dorsi* and dominant *rectus abdominis* muscles appears to be a centralised movement strategy and was not as a result of pain at the time of measurement.

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LINKING PARAGRAPH CHAPTER 4 AND CHAPTER 5

The first two studies demonstrated that both swing kinematics and muscle recruitment strategies during the swing were associated with developing lower back pain in young elite golfers. The kinematic and EMG equipment used in chapter 3 and chapter 4 is expensive and requires advanced skills to operate. As such not many golf academies, especially in developing countries, will have access to these kinds of assessment tools to identify golfers at risk of developing lower back pain. Identifying golfers at risk of lower back pain is essential so that targeted prevention interventions can be directed towards them. Functional movement screening has been proposed as a possible cost effective means of screening golfers at risk of developing injury. The next study, Chapter 5 - 'The efficacy of injury screening in elite golfers, a prospective longitudinal cohort study' aims to determine if commonly used functional movement screening tools were able to identify golfers at risk of developing lower back pain. Although this chapter is focused on lower back pain injury screening, screening for the neck/thoracic spine, upper limb and lower limb injuries was also performed and can be found in Appendix 14. Ideally, the findings from this study would form the basis of future research projects to develop an intervention to address the identified deficiencies.

CHAPTER 5: THE EFFICACY OF INJURY SCREENING IN ELITE GOLFERS, A PROSPECTIVE LONGITUDINAL COHORT STUDY.

Abstract

Background: Injury prevention is a growing focus for golfers in general and for elite golfers in particular. Functional movement screening has been proposed as a possible cost effective means of identifying underlying risk factors and is widely utilised by therapists, trainers and coaches.

Purpose/ hypothesis: To establish whether results from functional movement screening was associated with subsequent injury.

Study design: Prospective longitudinal cohort study with one baseline time point.

Methods: Forty-one injury-free golfers underwent an injury screen. The golfers were monitored for injury for six months for lower back pain.

Results: Seventeen golfers developed lower back pain (41%). Screening tests were able to differentiate golfers that developed lower back pain and those who did not included: rotational stability test on the non-dominant side ($p=0.01$, effect size=0.27), rotational stability test on the dominant side ($p=0.03$; effect size= 0.29) and plank score ($p=0.03$; effect size=0.24).

Conclusion: Out of 30 screening tests only three tests were able to identify golfers at risk of developing injury. All three of these tests had weak effect sizes.

Clinical Relevance: Functional movement screening was not effective in identifying elite golfers at risk of injury in this study.

5.1 Introduction

Elite golfers have a moderate risk of injury, the majority of which occur in the lower back (Robinson et al., 2019, Smith et al., 2018). Lower back pain prevalence amongst professional golfers is estimated to be around 50% (Smith et al., 2018). There has been increasing awareness regarding preventing injury in elite golfers. Several studies have shown an association between certain swing kinematics and developing lower back pain (Smith et al., 2018). The tools used to assess swing kinematics are expensive and require advanced skill to utilise. Functional movement screening has been proposed as a possible cost effective alternative to identifying these underlying risk factors and is widely utilised by therapists, trainers and coaches in the golf industry (Titleist Performance Institute, 2021b). There is, however, conflicting evidence regarding the usefulness of functional movement screening in identifying athletes at risk of injury (Moran et al., 2017).

According to a systematic review by Bahr (2016) there are three essential steps required to develop and validate a screening tool. The first step is to identify the strength of risk factor association with injury development (Bahr, 2016). In support of this first step, several studies have shown an association between trunk and hip muscle performance in golfers and back pain (Smith et al., 2018). In particular, decreased *transverse abdominis* endurance, decreased trunk strength in all planes, reduced trunk rotational endurance towards the lead side, reduced isokinetic trunk extension and reduced isometric hip adduction have been associated with lower back pain in golfers (Smith et al., 2018). Asymmetry between the left and right side endurance during the side bridge has been shown to be associated with the development of lower back pain (Evans et al., 2005), while pooled analysis of trunk extension range of motion did not correlate with developing lower back pain (Smith et al., 2018). Similarly pooled analysis of lead and trail hip external rotation range of motion and internal rotation was also not associated with developing lower back pain (Smith et al., 2018). Side to side hip internal rotation range of motion asymmetry has been shown to be greater in golfers with lower back pain (Smith et al., 2018). These studies give theoretical support to the concept that functional movement screening tools may be able to identify golfers at risk of developing lower back pain.

The second step in developing an injury screening tool involves screening athletes with the same test, but in this second scenario, a predetermined cut-off score is used to separate athletes at risk of injury from the rest (Bahr, 2016). The athletes should then be monitored for injury over a set period of time. Step three involves conducting a randomised controlled trial in which athletes at risk are identified using the screening tool and then randomly put into a control or a treatment group (Bahr, 2016). The treatment group is given an intervention aimed at addressing the risk factor identified by the tool. The athletes should then be monitored and if injury incidence reduces in the treatment group, the screening tool can be validated for that patient group (Bahr, 2016).

The aim of this study was to address the first step of the screening development process proposed by Bahr (2016); to determine if commonly used screening tools were associated with developing pain in the lower back in young elite male golfers.

5.2. Materials and Methods

5.2.1 Study design and setting

This study followed a prospective longitudinal cohort design and had one baseline measurement. The study took place at an elite golf academy in Johannesburg, South Africa. A flow diagram overview of the study is shown in Figure 5.1

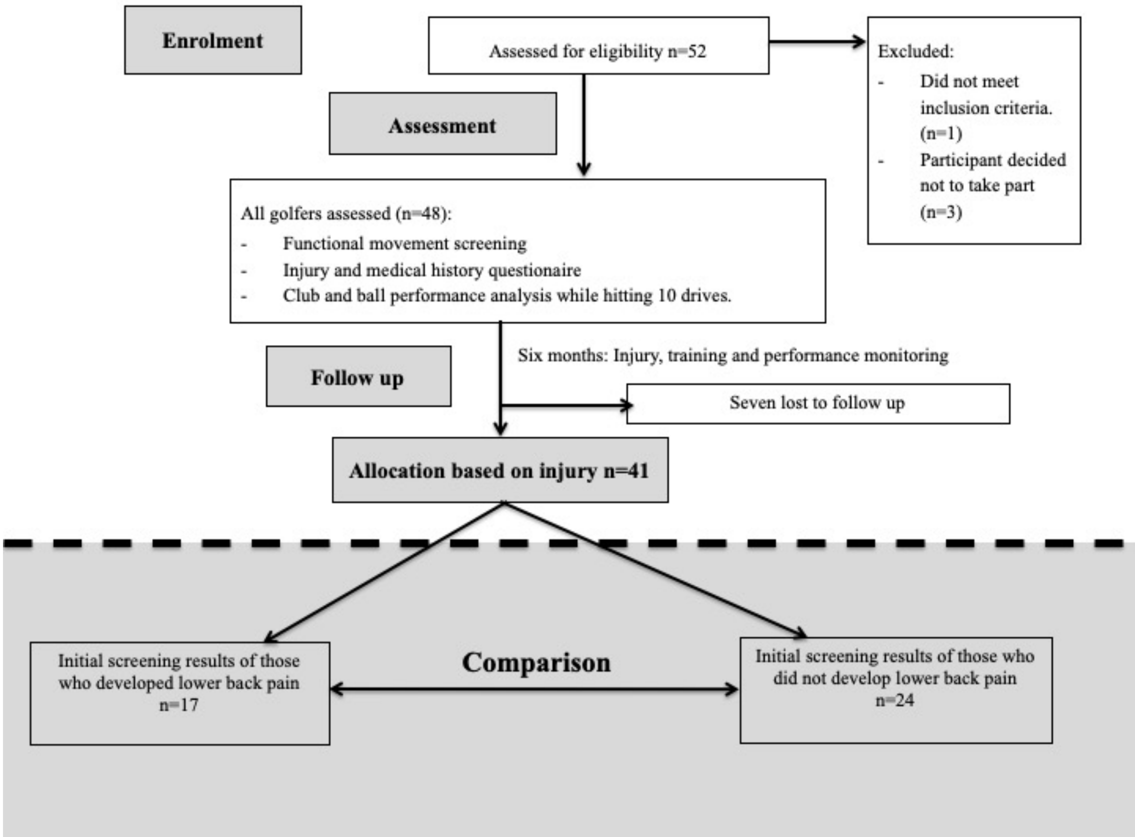

Figure 5.1: Cohort diagram for this study

5.2.2 Participants

To prevent selection bias all injury free golfers at the golf academy, that were also classified as elite players by their coaches were invited to take part in the study. 'Injury free' was defined as not experiencing pain or discomfort while playing golf or performing activities. Further inclusion criteria included being male and between the ages of 16 and 30 years old. Due to limited access to female golfers, we were unable to focus the study on female golfers. It was also not possible to combine female golfers functional screen measurements with male golfers measurements as women golfers are likely to have reduced strength compared to male golfers (Ramos et al., 1998). Golfers under 18 years old (Junior golfers) were included in the study, therefore a handicap cut off was not applied. Data were collected from Feb 2017 to Jan 2018.

5.2.3 Ethics

The Ethics Committee of the University of the Witwatersrand granted Ethical Clearance. The study was undertaken according to the ethical standards and guidelines of the Declaration of Helsinki. Identifiable information was removed from the dataset and study numbers were allocated to each participant. The link between the study numbers and identifiable information was kept in an access controlled file to which only the researcher had access.

Written informed consent was granted by participants over the age of 18 years old. Written informed assent was granted by participants under the age of 18 years old and informed written consent was granted by their legal guardians for them to take part in the study. An information sheet was provided to the parents/guardians by the Golf School of Excellence. Minors (participants under 18 years old) were only approach for inclusion in the study if their parents/guardian had given permission for them to take part in the study

Minors were included in this study as a high volume of sports participation has been shown to increase the risk of developing lower back pain in childhood and adolescence (Sato et al., 2011). Research involving minors is permitted if it is therapeutic research in the best interests of the minor (Strode and Slack, 2015). The screening performed and analysis was similar to what they would typically undergo at the Golf School of Excellence. The inclusion of minors in this study was approved by the University of the Witwatersrand's Human Research Ethics Committee.

5.2.4 Procedure

Each golfer attended a baseline assessment. This baseline assessment included: a demographic questionnaire, a medical history questionnaire, anthropometric measurements, a functional screening and a swing performance assessment.

The following demographic details were obtained: full name, address, contact numbers, email address, handicap. The medical screening and injury history questionnaire ascertained if the golfers had a history of: vascular conditions, respiratory diseases, cancer; musculoskeletal concerns, neurological conditions, gastrointestinal complaints or injury six months prior to inclusion in the study. Due to the sensitive nature of the medical screening and injury questionnaire, golfers were permitted to omit certain questions or forgo this questionnaire and still take part in the study. Giving the participants the option to omit part or all of the medical screening questionnaire was an important ethical consideration for this study. Despite this option being given, the majority of participants agreed to complete it.

At this initial assessment the golfers underwent a swing performance assessment. All participants were asked to hit the golf ball with their driver 10 times as they would during a competition where both distance and accuracy were important. Club and ball performance was monitored using the Flightscope X2 Elite system (Flightscope X2 Elite, Flightscope (Pty)Ltd, Stellenbosch, South Africa). This is a 3D Doppler tracking radar designed for golf specific application (Flightscope, Published 2021) as per Leach et al. (2017).

The initial assessment also included a functional movement screen. The functional movement screen was conducted by the primary researcher and two physiotherapy research assistants (all three assessors were qualified physiotherapists). The functional movement screen included the following tests (with their various sub-divisions), 30 in total: single leg squat, deep overhead squat; hurdle step, in line lunge, straight leg raise, trunk stability push up; spinal extension test, rotational stability test, spinal flexion clearing test, pelvic tilt test, pelvic rotation test, torso rotation test, toe touch test, 90 90 shoulder test, single leg bridge, lower quarter rotation test, seated trunk rotation test, plank, sit ups, push ups, and oblique sit ups. Each of the tests have been described in Appendix 15. The scoring for the tests has been provided in Appendix 16. These tests were selected for their utility for screening golfers before and during the season for risk of injury (Titleist Performance Institute, 2021b).

After the initial baseline assessment, the golfers were monitored for six months for injury. As most golf injuries are related to repetitive movement, the monitoring period needed to be long enough to capture these injuries. At the same time the injury needed to be related to the swing pattern at the time of assessment. The golfers included in this study were enrolled at a golf academy and in the process of refining their swing pattern. It was felt that a six-month monitoring period would allow sufficient time to capture injuries related to repetitive movement, while at the same time ensuring that the golfers swing had not changed significantly from the time of initial assessment.

Participants were asked to contact the research team immediately if any injury was sustained. Golfers were also contacted weekly to determine if they developed lower back pain or not during the previous week. If the researcher was unable to contact the golfer to determine if he was injured or not over the six month period he was excluded from the study. 'Injury' to the lower back was defined as 'pain and discomfort that was localised below the costal margin and above the inferior gluteal folds, with or without leg pain' (Burton et al., 2006). The injury did not need affect golf participation. The golfers were initially offered a free physiotherapy assessment, which many of the golfers did not choose to attend. Reasons given for not attending the physiotherapy assessment were: travel and training commitments. The golfers were free to seek out physiotherapy or medical care if they developed lower back pain. As this was an observational study, no attempt was made to influence their decision to seek care. Once the findings of the study have been published by peer-review journals, the Golf academy will be sent a summary of the findings in simple English as well as some recommendations regarding evidenced-based management of lower back pain. A recommendation will be made to the Golf Academy that they disseminate this information to all the golfers at the academy (current and past students).

Training was also monitored during this six-month period via a weekly training log, which was completed by the golfer. If the golfer forgot to complete the log, the primary researcher contacted the golfer via telephone and completed the log on behalf of the golfer. This log included the number of hours spent week engaged in putting practice, short game practice, range practice, flexibility training, postural training, weight training and cardiovascular exercise. The participants were also asked to report on the number of golf rounds played per week. Golfer performance was ranked using the 2017 yearly order of merit score. The majority of the golfers played in a weekly order of merit tournament, which resulted in a yearly order of merit score. The yearly order of merit score was calculated using the following parameters: true score, stroke average, fairways hit, greens hit, number of putts, scrambling (this is a score that measures how often a golfer scores par or better after missing a green in regulation) and ups and downs (a golfer is said to have achieved an 'up and down' if he takes only two strokes to get his ball into the hole when his ball is resting around the green or in a greenside bunker).

5.2.5 Data reduction and Analysis

The following functional tests were scored using a categorical scale: single leg squat, deep overhead squat, hurdle step, in line lunge, trunk stability push up, spinal extension test; rotational stability test, spinal flexion clearing test, pelvic tilt test, pelvic rotation test, torso rotation test, toe touch test, 90 90 shoulder test, single leg bridge, lower quarter rotation test, seated trunk rotation test and plank. The straight leg raise test was recorded using a goniometer. The sit up, push ups, and oblique sit ups were recorded as a total number completed per exercise within the set time frame.

The flight scope data was recorded as an average of 10 swing cycles. The flight scope data recorded carry distance (meters), roll (meters), total distance (meters), ball distance (meters) and club head speed (km/h).

Two golfers were left-handed and played golf left-handed. For this reason measurements were recorded as dominant and non-dominant.

5.2.6 Statistical analysis

Data was analysed using Statistica® Version 12 (StatSoft Inc, Tulsa, USA). Categorical data (Table 5.2, Table 5.7, Table A14.1, Table A14.6, Table A14.7, Table 14.12, Table 14.13, Table A14.18) was presented as frequencies and analysed using Pearson Chi Squared test or the Fisher's Exact Test (Kim, 2017) . Phi (φ) and risk ratio were used to describe the effect size of categorical data when there were two by two contingency tables (Kim, 2017, Pautz et al., 2018a).

$$\varphi = \sqrt{\frac{x^2}{n}}$$

Cramer's V was used to calculate effect size on larger tables and was interpreted according to the table (Table 5.1) (Kim, 2017). This is calculated using the equation:

$$V = \sqrt{\frac{x^2}{n-df}}$$

n is the total number of observations. df is the degrees of freedom which is calculated by $(r-1)(c-1)$. Here, r is the numbers of rows of the contingency table and c is the numbers of columns of the contingency table (Kim, 2017).

Table 5.1: Interpretation of effect size for Cramer's V

Degree of freedom	Small	Medium	Large
1	0.1	0.3	0.5
2	0.07	0.21	0.35
3	0.06	0.17	0.35
4	0.04	0.13	0.22

Continuous and ranked data was tested for normality (Table 5.4-5.6, Table A14.3-A14.5, Table A14.9-A14.11, Table A14.15-A.17). Normality was determined by dividing kurtosis score by its standard error and by dividing skew by its standard error (z score) (Kim, 2013). If the z score was between -1.96 and 1.96, the data was considered to be normally distributed (Kim, 2013). The data that was normally distributed is marked with a superscript ‘n’ in the results tables (Table). Normally distributed data were analysed using unpaired Student’s t-tests was presented as means and standard deviations. Data that was not normally distributed or was ranked data was analysed using the Mann Whitney U-test and was presented as medians and interquartile ranges.

In line with the American Statistical Association’s statement on the use of p-values (Wasserstein et al., 2019), a dichotomised cut-off of p<0.05 was not applied to this research study. The term ‘statistically significant’ or any of its variants was not used, but p-values were reported as continuous quantities (Wasserstein et al., 2019). Information displaying differences between groups confounding variable at baseline was presented using p values, means or medians, standard deviations or interquartile ranges, confidence intervals and effect sizes.

Effect sizes for data that underwent Student’s t-tests was calculated and presented as Cohen’s (d_s), where $d_s = \frac{X_1 - X_2}{\sqrt{\frac{(n_1-1)SD_1^2+(n_2-1)SD_2^2}{n_1+n_2-2}}}$ (Pautz et al., 2018b). Effect sizes for data that had undergone the Mann Whitney U-test was presented as probability of superiority ($P_{a,b}$), where $P_{a.b} = \frac{U}{n_a n_b}$ (Pautz et al., 2018a). Effect sizes for continuous and ranked data were interpreted as: small (≤0.2), moderate (≥0.5), and large (≥0.8) (Cohen, 1992). (Effect sizes, p-values, confidence intervals and minimum clinically meaningful change were taken into consideration when showing a difference between groups in this paper (Pautz et al., 2018a, Pautz et al., 2018b).

Missing data was not replaced or estimated. If a participant declined to take part in a screening test or answer a question in the medical screening questionnaire, no data for that test was entered.

Biomechanical studies typically involve small sample sizes. This is largely due to the amount of time it takes to collect data for one participant. Instead of conducting a formal sample size to power the study, p values, confidence intervals, minimum clinically meaningful change and effect sizes were reported. This approach has been used in other biomechanical studies e.g. single leg squat study published by Olivier et al. (2019).

5.3 Results

5.3.1 Participants and incidence of injury

Forty-eight participants underwent the baseline assessment. Seven participants completed the screen but were lost to follow up. The total number of participants included in the final analysis was 41. The whole group's median (IQR) age was 19.80 (3) years ; mean (SD) BMI was 24.35 (3.68) kg/m^2 and the median (IQR) handicap was 2 (3). Seventeen participants (41%) developed lower back pain. Fourteen golfers only had one episode of lower back pain. Episodes were measured in weeks. The first episode median(IQR)=2(4) weeks. Three participants had two episodes of lower back pain. The second episode mean(SD)=2.3(0.56) weeks. The total length of back pain episodes (first and second) median(IQR)= 2(5) weeks. Three participants reported lower back pain and lower limb pain at the same time. Fifteen participants continued to play golf despite having lower back pain.

It should also be noted that nine participants developed pain in their upper limb (upper limb injury incidence of 22%). Three of which were to the shoulder region and six in the elbow, wrist and hand region. Nine participants developed pain in their lower limb, three of which occurred in the hip region, two in the knee and four in the ankle (lower limb injury incidence of 22%). Five participants developed pain in the neck and thoracic region (neck and thoracic spine injury incidence of 12%).

5.3.2 Screening tests for lower back pain

There was a difference between the group that developed lower back pain and those that did not in the following screening tests: rotational stability test non dominant side (p=0.01, effect size=0.27), rotational stability test dominant side (p=0.03; effect size= 0.29) and plank score (p=0.03; effect size=0.29) (Table 5.3). The lower back pain group performed better in rotational stability tests and in the plank test. There were no differences observed in any other screening tests for lower back pain (Table 5.2-Table 5.4).

Table 5.2: Page 1 of 4: Table comparing categorical screening test results for those with lower back pain compared to those without lower back pain

Variable	Score	No lower back pain group n	No lower back pain group per category n(%)	Lower back pain group n	Lower back pain group per category n(%)	Comparison between no lower back pain and lower back pain group p value	Comparison between no lower back pain and lower back pain group effect size
Spinal extension clearing test	Pain	24	0 (0%)	17	3 (17.65%)	0.06	0.33
	No pain		24 (100%)		14 (82.35%)		
Spinal flexion clearing test	Pain	24	0 (0%)	17	1 (5.88%)	0.42	0.19
	No pain		24 (100%)		16 (94.12%)		
Pelvic tilt test posture at set up score	Neutral	24	22 (91.67%)	17	15 (88.24%)	0.41	0.06
	Excessive anterior		1 (4.17%)		1 (5.88%)		
	Excessive posterior		1 (4.17%)		1 (5.88%)		
Pelvic tilt at set up posture amount of motion score	Normal motion	24	15 (62.5%)	16	11 (68.75%)	0.41	0.27
	Limited posteriorly		2 (8.33%)		2 (12.5%)		
	Limited anteriorly		0 (0%)		1 (6.25%)		
	Both limited		7 (29.17%)		2 (12.5%)		
Pelvic tilt at set up posture quality of movement	Smooth movement	24	5 (20.83%)	17	4 (23.53%)	1	0.03
	Shaking movement		19 (79.17%)		13 (76.47%)		
Pelvic rotation stability score	Good stability	24	21 (87.5%)	17	15 (88.24%)	0.53	0.23
	Limited stability left side		2 (8.33%)		1 (5.88%)		
	Limited stability right side		1 (4.17%)		0 (0%)		
	Both sides stability limited		0 (0%)		1 (5.88%)		
Pelvic rotation test mobility score	Full mobility	24	15 (62.5%)	17	31 (76.47%)	0.49	0.24
	Limited mobility to the left side		1 (4.17%)		0 (0%)		
	Limited mobility to the right side		2 (8.33%)		0 (0%)		
	Both sides showed limited mobility		6 (25%)		4 (23.53%)		
Pelvic rotation quality of movement score	Good rotatory movement	24	16 (66.67%)	17	11 (64.71%)	1	0.02
	Lateral movement more than rotatory movement		8 (3333%)		6 (35.29%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared Test and Fishers exact test.

Table 5.2 Continued Page 2 of 4: Table comparing categorical screening test results for those with lower back pain compared to those without lower back pain

Variable	Score	No lower back pain group n	No lower back pain group per category n(%)	Lower back pain group n	Lower back pain group per category n(%)	Comparison between no lower back pain and lower back pain group p value	Comparison between no lower back pain and lower back pain group effect size
Torso rotation test stability score	Good stability	24	24 (100%)	17	16 (94.12%)	0.41	0.19
	Limited to the left side		0 (0%)		1 (5.88%)		
Torso rotation test mobility score	Full mobility	24	20 (83.33%)	17	§6 (94.12%)	0.65	0.20
	Limited mobility to the left side		1 (4.17%)		0 (0%)		
	Limited mobility to the right side		1 (4.17%)		0 (0%)		
	Both sides showed limited mobility		2 (8.33%)		1 (5.88%)		
Torso rotation test quality of movement	Good rotatory movement	23	20 (86.96%)	17	17 (100%)	0.25	0.25
	More lateral movement		3 (13.04%)		0 (0%)		
Toe touch test score	Full range of motion	24	13 (54.17%)	16	12 (75%)	0.37	0.28
	Decreased toe touch on the left		2 (8.33%)		2 (12.5%)		
	Decreased toe touch on the right		1 (4.17%)		0 (0%)		
	Decreased toe touch bilaterally		8 (33.33%)		2 (12.5%)		
90 90 non dominant in standing	Standing <spine	24	2 (8.33%)	16	3 (18.75%)	0.5	0.19
	standing=spine		11 (45.83%)		8 (50%)		
	Standing>spine		11 (45.83%)		5 (31.25%)		
90 90 dominant in standing	Standing <spine	24	2 (8.33%)	17	3 (17.65%)	0.62	0.15
	standing=spine		14 (58.33%)		8 (47.06%)		
	Standing>spine		8 (33.33%)		6 (35.29%)		
90 90 shoulder in set up posture dominant score	Standing <spine	24	1 (4.17%)	17	4 (23.53%)	0.13	0.32
	standing=spine		11 (45.83%)		8 (47.06%)		
	Standing>spine		12 (50%)		5 (29.41%)		
90 90 shoulder in set up posture non-dominant score	Standing <spine	24	3 (12.5%)	17	4 (23.53%)	0.65	0.15
	standing=spine		14 (58.33%)		9 (52.94%)		
	Standing>spine		7 (29.17%)		4 (23.53%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared Test and Fishers exact test.

Table 5.2: Continued Page 3 of 4: Table comparing categorical screening test results for those with lower back pain compared to those without lower back pain

Variable	Score	No lower back pain group n	No lower back pain group per category n(%)	Lower back pain group n	Lower back pain group per category n(%)	Comparison between no lower back pain and lower back pain group p value	Comparison between no lower back pain and lower back pain group effect size
Single leg bridge non-dominant score	Glutes normal	24	20 (83.33%)	17	12 (70.59%)	0.4	0.21
	Glutes weak		4 (16.67%)		4 (23.53%)		
	Glutes inhibited		0 (0%)		1 (5.88%)		
Single leg bridge dominant score	Glutes normal	24	22 (91.67%)	17	13 (76.47%)	0.3	0.24
	Glutes weak		2 (8.33%)		3 (17.65%)		
	Glutes inhibited		0 (0%)		1 (5.88%)		
Lower quarter rotation non-dominant: internal rot score	>60 degrees	24	11 (45.83%)	17	8 (47.06%)	0.32	0.24
	=60 degrees		11 (45.83%)		5 (29.41%)		
	<60 degrees		2 (8.33%)		4 (23.53%)		
Lower quarter rotation non-dominant: external rot score	>60 degrees	24	19 (79.17%)	17	15 (88.24%)	0.73	0.12
	=60 degrees		3 (12.5%)		1 (5.88%)		
	<60 degrees		2 (8.33%)		1 (5.88%)		
Lower quarter rotation dominant: internal rot score	>60 degrees	24	1 (41.67%)	17	12 (70.59%)	0.14	0.31
	=60 degrees		11 (45.83%)		3 (17.65%)		
	<60 degrees		3 (12.5%)		2 (11.76%)		
Lower quarter rotation dominant: external rot score	>60 degrees	23	15 (65.22%)	17	14 (82.35%)	0.34	0.23
	=60 degrees		6 (26.09%)		3 (17.65%)		
	<60 degrees		2 (8.7%)		0 (0%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared Test and Fishers exact test.

Table 5.2: Continued Page 4 of 4: Table comparing categorical screening test results for those with lower back pain compared to those without lower back pain

Variable	Score	No lower back pain group n	No lower back pain group per category n(%)	Lower back pain group n	Lower back pain group per category n(%)	Comparison between no lower back pain and lower back pain group p value	Comparison between no lower back pain and lower back pain group effect size
seated trunk rot non-dominant score	>45 degrees	24	21 (87.5%)	17	17 (100%)	0.32	0.24
	=45 degrees		1 (4.17%)		0 (0%)		
	<45 degrees		2 (8.33%)		0 (0%)		
Seated trunk rot dominant score	>45 degrees	23	20 (86.96%)	17	17 (100%)	0.3	0.25
	=45 degrees		1 (4.35%)		0 (0%)		
	<45 degrees		2 (8.7%)		0 (0%)		
Seated trunk rot non-dominant: club behind back score	>45 degrees	24	21 (87.5%)	17	17 (100%)	0.32	0.24
	=45 degrees		1 (4.17%)		0 (0%)		
	<45 degrees		2 (8.33%)		0 (0%)		
Seated trunk rot dominant: club behind back score	>45 degrees	24	22 (91.67%)	17	17 (100%)	0.47	0.19
	=45 degrees		1 (4.17%)		0 (0%)		
	<45 degrees		1 (4.17%)		0 (0%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared Test and Fishers exact test.

Table 5.3: Table comparing ranking screening test results for those with lower back pain compared to those without lower back pain

Variable	No lower back pain group percentage per category n=24			Lower back pain group percentage per category n=17			Comparison between no lower back pain and lower back pain group p value	Comparison between no lower back pain and lower back pain group effect size
	Median (IQR)	Lower CI	Upper CI	Median (IQR)	Lower CI	Upper CI		
Single leg squat non-dominant score	2 (1)	1.58	2.01	2 (1)	1.33	1.85	0.22	0.41
Single leg squat dominant score	2 (1)	1.58	2.01	2 (1)	1.34	1.96	0.36	0.43
Deep overhead squat score	2 (1)	2.1	2.49	2 (1)	2.15	2.67	0.43	0.44
Hurdle step non-dominant score	2 (1)	2.10	2.49	2 (1)	2.05	2.54	0.99	0.5
Hurdle step dominant score	2 (1)	2.10	2.49	2 (1)	2.1	2.61	0.68	0.47
In line lunge non-dominant score	2.5 (1)	2.28	2.72	3 (1)	2.26	2.79	0.86	0.49
In line lunge dominant score	3.5 (1)	2.28	2.72	3 (1)	2.26	2.79	0.86	0.49
Trunk stability push up score	3 (0)	2.67	2.99	3 (0)	2.82	3.07	0.3	0.45
Rotational stability test non-dominant score	2 (0)	1.87	2.3	3 (1)	2.27	2.91	0.01	0.27
Rotational stability test dominant score	2 (0)	1.96	2.39	3 (1)	2.27	2.91	0.03	0.29
Plank score	3 (4)	2.41	4.72	7 (4)	4.38	7.22	0.03	0.24

Table notes: n= number of participants. Data presented as medians and interquartile range (IQR). p value calculated using Mann Whitney U test.

Table 5.4: Table comparing continuous screening test results for those with lower back pain compared to those without lower back pain

Test	No lower back pain				Lower back pain				Comparison between lower back pain and no lower back pain group p value	Comparison between lower back pain and no lower back pain group effect size
	n	Mean (SD) or median (IQR)	95% CI lower	95% CI Upper	n	Mean (SD) or median (IQR)	95% CI lower	95% CI upper		
Straight leg raise non-dominant (range of motion)[n]	24	68.58 (12.10)	63.48	73.69	16	74.19 (18.56)	64.30	84.07	0.25	0.37
Straight leg raise dominant (range of motion) [n]	24	69.83 (11.37)	65.03	74.64	16	71.38 (16.94)	62.35	80.40	0.73	0.11
Sit ups (no.)[n]	24	37.13 (11.26)	32.37	41.88	17	37.76 (8.05)	33.63	41.90	0.84	0.06
Push ups (no.)	24	38.50 (16.00)	31.27	43.56	17	32.50 (15.00)	30.04	41.09	0.38	0.51
Oblique sit up to non-dominant shoulder (no.) [n]	24	29.46 (9.37)	25.50	33.42	16	31.44 (7.10)	27.65	35.22	0.48	0.23
Oblique sit up to dominant shoulder (no.)[n]	24	28.04 (10.75)	23.50	32.58	16	28.94 (5.95)	25.77	32.11	0.74	0.1

Table notes: [n] indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations. Data not normally distributed presented as medians and interquartile range (IQR). p values calculated using independent T tests (normally distributed data) and Mann Whitney U tests (data not normally distributed). n=number of participants

5.3.3 Incomplete screening tests

The following tests had one participant decline to perform them: pelvic tilt at set up posture (amount of motion), torso rotation test (quality of movement), toe touch test, 90 90 non-dominant test in standing, lower quarter rotation dominant external rotation test, seated trunk rotation dominant side, straight leg raise test, oblique sit up. This was due to time constraints. The screening took a long time to complete and some of the participants needed to leave due to transport arrangements.

5.3.4 Confounding variables: Performance and training

The lower back pain group (median (IQR)= 3.33 (4.45) hr/week) engaged in less range practice than the uninjured group (median (IQR)= 7.5 (8.5) hr/week; p=0.04; effect size=0.31). The lower back pain group engaged in less cardiovascular training (Median(IQR)= 1.25(0.96) hr/week) than the uninjured group (Median(IQR)= 1.67 (2.43) hr/week; p=0.04; effect size=0.31). There were no other performance or training differences between the group that developed lower back pain and the group that did not develop back pain (Table 5.5). It should be noted that one participant's Flightscope data did not record. It should also be noted that one participant completed only 17 weeks of his training log and declined to complete any further training logs. He was, however, prepared to continue to be contacted for his injury monitoring. An average training for this participant was provided for week 18- week 26.

Table 5.5: Comparison of baseline performance and training variables for the lower back pain group versus the no lower back pain group

Variable	No lower back pain group				Lower back pain group				Comparison between lower back groups p value	Comparison between lower back groups Effect size
	n	Mean (SD) or median (IQR)	95% CI lower	95% CI upper	n	Mean (SD) or median (IQR)	95% CI lower	95% CI upper		
Order of merit (rank)	17	24.00 (28)	16.87	32.54	11	20 (31)	14.28	36.45	0.96	0.49
Handicap (no)	24	1.5 (3)	1.34	3.99	17	2 (2.5)	1	3	0.76	0.47
Carry distance (m) ^{n}	24	214.51 (21.10)	205.61	223.41	16	218.07 (22.69)	205.98	230.16	0.62	0.16
Roll distance (m)	24	12.33 (11.58)	10.95	21.57	16	18.55 (22.20)	13.02	30.20	0.30	0.40
Total distance (m) ^{n}	24	230.73 (16.98)	223.56	237.9	16	239.81 (20.32)	228.81	250.46	0.14	0.49
Ball speed (km/h)	24	238.76 (13.71)	219.22	243.82	16	240.26 (22.07)	217.98	247.43	0.86	0.48
Club head speed (km/h)	24	177.30 (10.54)	164.61	182.51	16	175.32 (13.8)	160.77	181.23	0.33	0.41
Putting practice per week (hours per week)	24	3.98 (6.74)	2.48	11.32	17	3.08 (3.12)	2.26	3.97	0.27	0.4
Short game practice per week (hours per week)	24	4.1 (7.68)	2.56	11.83	17	2.94 (3.44)	2.12	4.07	0.13	0.36
Range practice per week (hours per week)	24	7.5 (8.5)	5	14.2	17	3.33 (4.54)	2.5	7.37	0.04	0.31
Flexibility training per week (hours per week)	24	1.17 (2.75)	0.49	4.98	17	0.92 (1.13)	0.69	1.44	0.37	0.42
Postural training per week (hours per week)	24	0.7 (1.41)	-0.27	4.09	17	0.2 (0.42)	-0.22	1.69	0.32	0.41
Weight training per week (hours per week)	24	2 (3.02)	1.37	6.43	17	1.81 (2.69)	0.93	2.4	0.34	0.41
Cardio per week (hours per week)	24	1.67 (2.43)	1.28	4.57	17	1.25 (0.96)	0.78	1.59	0.04	0.31
Rounds of golf played per week	24	2.88 (2)	-11.83	45.09	17	3.04 (1.64)	1.82	3.32	0.58	0.45

Table notes: : n indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations (SD). Data not normally distributed presented as medians and interquartile range (IQR). p values calculated using independent T tests (normally distributed data) and Mann Whitney U tests (data not normally distributed). n=number of participants CI were displayed as calculated, but please be advised that it is not possible to obtain a negative value for the confidence interval for these values, except for handicap. The lowest possible value is zero

5.3.5 Confounding variables back groups, lower limb groups and upper limb groups: Baseline anthropometrics, medical history and injury history

The lower back pain group had a lower BMI and body mass than the uninjured group (BMI p=0.04, body mass p=0.02; BMI effect size=0.68 body mass effect size =0.78) (Table 5.14). There were no other differences between back pain groups in terms of baseline anthropometrics, medical history or injury history (Table 5.6 and Table 5.7).

Although all the golfers were injury free at the time of inclusion, 17 golfers had a history of lower back pain in the six months prior to inclusion. In terms of prior lower back pain:

- Thirteen golfers had experienced lower back pain in the month prior to inclusion.
- Five golfers had missed golf training days as a result of lower back pain.
- Twenty-one training days were missed in total as a result of prior lumbar spine pain
- Three golfers missed training as a result of prior lower back pain
- Reasons given included: poor posture, overtraining, S-shaped spine, swing faults, bending, insidious onset, carrying of golf bag, playing rugby, muscle spasm, tiredness and high swing speed.
- Numerical pain score out of 10 mean (SD)= 5(2)
- Seven golfers saw a physiotherapist for their lower back pain
- One golfer saw his general practitioner (GP) for his lower back pain.
- The golfers who saw a physiotherapist or GP reported being diagnosed with: muscle spasm and poor vertebral alignment (physiotherapy diagnosis for one golfer).
- The golfers had undergone the following treatments: strapping, RICE (rest, ice, compression, elevation techniques), massage, physiotherapy, strapping, anti-inflammatory topical plasters and foam rolling

It should be noted that five participants declined to complete the medical history and injury history questionnaire. Some participants part completed the medical history and injury history questionnaire (number of completed answers to each question is displayed in Table 5.7).

Table 5.6: Comparison of baseline anthropometric data back pain group versus no back pain group

Variable	No back pain group				Back pain group				Comparison between back group p value	Comparison between back group effect size
	n	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper	n	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper		
Age (years) ^{n}	24	19.54 (2.41)	18.53	20.56	17	20.18 (2.67)	18.8	21.55	0.43	0.25
Height (m) ^{n}	24	1.8 (0.08)	1.76	1.83	17	1.78 (0.05)	1.75	1.8	0.32	0.32
Body mass (kg) ^{n}	24	81.98 (12.54)	76.69	87.28	17	72.42 (11.79)	66.36	78.49	0.02	0.78
BMI (kg/m^2)^{n}	24	25.34 (3.53)	23.85	26.83	17	22.94 (3.52)	21.13	24.76	0.04	0.68

Table notes: : ^{n} indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations (SD). Data not normally distributed presented as medians and interquartile range (IQR). p values calculated using independent T tests (normally distributed data) and Mann Whitney U tests (data not normally distributed). n=number of participants.

Table 5.7: Comparison of baseline medical history and history of injury: No lower back pain group versus the lower back pain group

Variable	No lower back pain group n	No lower back pain group n(%) with condition or health risk factor	Lower back pain group n	Lower back pain group n(%) with condition or health risk factor	p value for between group difference at baseline	Between group Odds ratio	Risk ratio in lower back pain group	Between group comparison effect size
Smoked at least 100 cigarettes in his lifetime	19	9 (47.37%)	17	7 (41.18%)	0.75	1.29	0.88	0.06
History vascular problems	19	1 (5.26%)	16	1 (6.25%)	1	0.83	1.1	0.02
History respiratory problems	17	5 (29.41%)	16	3 (18.75%)	0.69	1.81	0.72	0.12
History of cancer	19	1 (5.26%)	16	1 (6.25%)	1	0.83	1.19	0.02
History of musculoskeletal conditions	19	1 (5.26%)	16	3 (18.75%)	0.31	0.24	1.79	0.21
History of neurological conditions	19	3 (15.79%)	16	2 (12.5%)	1	1.31	0.86	0.05
History of gastrointestinal conditions	19	3 (15.79%)	16	2 (12.5%)	1	1.31	0.86	0.05
History of injury in past six months	18	7 (38.89%)	16	12 (75%)	0.05	0.21	2.37	0.36
History of back pain in the past six months	19	8 (42.11%)	16	9 (46.25%)	0.51	1.77	0.75	0.14

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared test and Fishers exact test. n= number of participants

5.4 Discussion

5.4.1 Discussion of results

Both the lower back pain group scored better than their uninjured counterparts for rotational stability on the dominant and non-dominant sides. The lower back pain group also scored better in the plank test than their uninjured counterparts. The primary muscles used during these tests are the trunk muscles (Oliva-Lozano and Muyor, 2020). These results would therefore imply that increased strength, endurance and control of the trunk musculature were a precursor to sustaining both lower back pain and lower limb pain. Strong spinal or core-muscle may result in increased spinal muscle bracing via co-contraction movement strategies, which may result in increased spinal compression (Granata and Marras, 1995) thereby increasing the risk of spinal injury. It should, however, be noted that the effect sizes for this change are small.

It should also be noted that a large number of screening tests were conducted which increases the risk of false positive conclusions (Weisstein, 2021). The concept of this being a false positive is further supported by the lack of injury correlation in the other tests that assess spinal muscle strength and control, such as the trunk stability push up, single leg bridge, pelvic tilt tests, pelvic rotation test, sit ups and oblique sit ups. Despite the possibility of a spurious result, improved spinal muscle strength and control may be a precursor to lower back pain and lower limb pain in elite golfers and therefore future studies should attempt to confirm or negate this finding.

In terms of confounding variables related to training there were some differences between the back pain group and the no back pain group (cardio and range practice). It should be noted that the effect sizes for these differences were weak and that the golfers all practiced at the same academy and therefore the clinical relevance of these differences is unlikely to influence the study findings.

5.4.2 Clinical Implications

The golfers underwent 30 screening tests. Out of 30 screening tests only three tests were associated with developing lower back pain and all of these tests had a small effect size. These tests are time consuming and generally require that coaches require additional training in order to perform them. This paper brings into question their usefulness in identifying players at risk of sustaining injury. This is consistent with findings in other sports. In a systematic review by Moran et al. (2017) they concluded that there was moderate evidence against the use of the Functional Movement Screen in football to predict injury and that there was limited or conflicting evidence regarding its useful ness in predicting injury in college athletes, American football, ice hockey, basketball, running, fire fighters and police. A systematic review by Moore et al. (2019)provided more nuanced findings. They found mixed results regarding the functional movement composite score (FMS) and subsequent injury risk. A FMS below 15 and FMS asymmetry was associated with a small increase in injury in senior athletes, but not in junior athletes. A FMS below 15 was more likely to be associated with injury risk in rugby, American football and ice hockey than other sports. A FMS score below 15 was associated with a small increase in injury in males, but not females. They also noted that the effect sizes for between the uninjured and injured group's FMS scores were small and therefore may not be clinically meaningful. They concluded that FMS scores are not effective at identifying athletes at high risk of injury and should not be considered a complete injury-risk estimation tool (Moore et al., 2019). In slight contradiction to these reviews, a systematic review by Trinidad-Fernandez et al. (2019) concluded that due to the poor heterogenicity and varied definitions of injury that it is not possible to synthesise findings and draw any firm conclusion regarding FMS score and injury risk.

The questionable efficacy of screening tests to predict subsequent injury is a clear finding emerging from the data. Further, it could be questioned whether we should continue to conduct screening tests in elite golfers? Although many of these tests do not seem to have positive association with injury risk, they are useful in that they give a comprehensive assessment of the golfers' strength and range of motion (Bahr, 2016). These tests have also been shown to be positively associated with golf performance and swing characteristics (Speariett and Armstrong, 2019, Gulgin et al., 2014). According to the International Olympic Committee elite athletes should undergo comprehensive health evaluations (Bahr, 2016, Ljungqvist et al., 2009). Comprehensive health evaluations are important for establishing rapport with the medical team, reviewing medication and preventing doping, establishing a performance baseline and to satisfy medico legal requirements in some countries (Bahr, 2016). Based on the findings of this study, we would recommend continuing to perform functional movement screening to establish baseline muscle function and length, but not as a screening tool to identify golfers at risk of injury.

5.4.3 Future research

Future research should confirm if the rotational stability, plank tests are useful predictors of injury in young elite male golfers. These tests should ideally be conducted in isolation so as to exclude Bonferroni type error.

5.4.4 Limitations

Flightscope assessment was performed outdoors where environmental conditions such as wind speed may have influenced measurements (even though many of the measurements are only made within the first few feet of ball flight). An outdoor setting was chosen above a laboratory setting as it was felt that this would reduce the anxiety that golfers (especially younger) may experience during the assessment, while also being a more realistic representation of actual golfing performance.

There was a difference in BMI and mass between the lower back pain group and the no lower back pain group. This difference had a moderate effect size and should be taken into account when interpreting the results of this study. There is conflicting evidence surrounding the effect of body mass on developing back pain in golf (Smith et al., 2018). A systematic review by Smith et al. (2018) showed that golfers with lower back pain were heavier than golfers without lower back pain. In contrast a longitudinal study by Evans et al. (2005) on trainee professional golfers showed that golfers who with a BMI below 25.7kg/m2 were at greater risk of developing lower back pain. The impacts of this variable (BMI) on the study results is therefore unknown.

5.5 Conclusion

Out of 30 screening tests only three tests were associated with injury. According to this study functional movement screening should not be used to screen young elite male golfers at risk of injury. Functional movement screening, does, however, provide valuable information regarding elite athletes muscle function and length and as per the International Olympic Committee recommendations it should continue to be used for this purpose.

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LINKING PARAGRAPHS CHAPTER 5 AND CHAPTER 6

The first two studies demonstrated that both swing kinematics and muscle recruitment strategies during the swing are associated with developing lower back pain in elite golfers. These studies utilised complex, expensive analysis techniques that may not be readily available to the average golfer or coach. The next step was to see if relatively inexpensive functional movement screens were able to identify players at risk of injury. The third study (chapter 5) of this thesis showed that unfortunately functional movement screening tools appear to have questionable efficacy in identifying golfers at risk of developing injury, therefore I propose that young elite male golfers should be identified as being at risk of lower back pain based on their golf swing kinematics and muscle activity (based on the findings from chapter 3 and chapter 4).

Once a golfer has been identified as being at risk of lower back pain (based on his swing kinematics or muscle activation patterns) he should ideally undergo musculoskeletal interventions aimed at addressing the risk factor identified. Interventions for this group of golfers may include exercises and drills focused on modifying their swing to a less injury provoking movement patterns. These changes in movement pattern may be associated with worsening golf performance; therefore before embarking on altering an elite golfer's swing, it is important to know the performance impact of each swing characteristic.

An elite golfer's primary goal is to play excellent golf. Any lower back pain prevention strategy needs to keep this goal in mind. This is in line with person-centred health rehabilitation and it is essential to improving uptake of any lower back pain intervention strategy proposed. Any swing modification that results in a swing style (based on swing characteristics) that is associated with worse performance will not be tolerated by elite golfers.

The next section, Chapter 6- ‘Golf drive distance better predicts overall golfing performance than conventional measurements of swing characteristics, a cross-sectional study in young elite golfers’ aims to identify the swing factors (including swing characteristics, club and ball performance and X-factor) associated with performance in young elite male golfers so that the golfer, coach and physiotherapist can carefully weight up the performance risk versus the possible reduced injury risk.

CHAPTER 6: GOLF DRIVE DISTANCE BETTER PREDICTS OVERALL GOLFING PERFORMANCE THAN CONVENTIONAL MEASUREMENTS OF SWING CHARACTERISTICS, A CROSS-SECTIONAL STUDY IN YOUNG ELITE GOLFERS.

Abstract

Background: Interventions aimed at altering swing mechanics to less injury provoking styles, may improve a golfers playing longevity and reduce back pain incidence in elite golfers. In contrast, the performance impact of different swing characteristics are not always clear. The drive is the single activity in golf, which contributes the greatest proportion to golfing performance, if such performance can be considered as delivering a golf ball into its target hole. The properties of and individual's drive to predict overall golfing performance (assessed through an order of merit score) is therefore a reasonable proposition. Variables which derive from an individual's driving action and that are often assessed during golfing instruction for the purpose of performance optimisation and injury prevention include the assessment of swing characteristics, club speed and ball flight properties.

Purpose/ hypothesis: The aim of this study was to assess the relationship between competition performance and swing characteristics, X-factor, club and ball performance.

Study design: Cross-sectional study.

Methods: Twenty-eight young elite male golfers were included in this study. At baseline, swing characteristics, X-factor, club and ball performance were measured for 10 drives. The golfers also competed in a yearly order of merit competition. Analysis was conducted to establish if there was a relationship between competition performance (measured by order of merit score) and the baseline variables namely swing characteristics, X-factor, club and ball performance (roll, ball speed and club head speed).

Results: There was an association between competition performance and carry distance ($p=0.01$; $rho=-0.47$) and total drive distance ($p<0.00$; $rho=-0.53$). There was no association between competition performance and swing characteristics, roll, ball speed, club head speed or X-factor.

Conclusion: Carry distance and total drive distance predicted competition performance. While precautionary swing modification may reduce injury risk or pain in young elite golfers, golf drive distance is a better indicatory of overall performance.

6.1 Introduction

The golf drive is a unique action responsible for both golfing proficiency and concomitant injury (Smith et al., 2018). In their pursuit for a successful golf swing young elite male golfers are at risk of developing lower back pain and have been shown to have a lower back pain incidence of 57.1% over a one year monitoring period (Smith et al., 2018). A possible contributing factor to lower back pain in elite golfers is the characteristics of the golf swing, therefore interventions aimed at altering swing mechanics to less injury provoking styles (Smith et al., 2018, Cole and Grimshaw, 2016), may improve golfing longevity and reduce lower back pain incidence. A potential risk to golf drive modification in young elite male golfers, however, is reduced competition performance due to the adoption of a potentially less favourable swing characteristic from a performance perspective. The golf drive has often been cited as a strong determinant of overall golf performance in professional golfers (Alexander and Kern, 2005).

Swing characteristics and club and ball performance have been shown to influence competition performance. In a study conducted by Myers et al. (2008) maximum upper torso rotation velocity, maximum torso-pelvic separation, increased X-factor (torso-pelvic separation at the top of the back swing), upper torso rotational velocity at lead arm parallel and at the last 40ms before impact, were shown to increase ball velocity. Similarly, a study conducted by Chu et al. (2010) showed that delayed release of the arms and wrist, X-factor, trunk forward and lateral tilting and weight shifting during the swing were associated with improved ball velocity.

It is likely the more proficient (elite) golfers, have greater magnitudes of these successful swing characteristics, and that younger elite players have a particular ability to acquire the desired, performance related techniques for a successful swing (having less habitual motor memory of imperfect swing characteristics). That said, the ability to acquire performance related, but injury producing movement patterns is also a particular concern in young elite golfers, who have the ability to generate forces linked to both enhanced performance and tissue damage. The formerly cited studies were, however, performed on recreational middle aged golfers with relatively high handicaps, therefore their findings might not be applicable to elite or younger golfers (Myers et al. (2008) participant mean age was 45 years, mean handicap was eight; Chu et al. (2010) participants mean age was 43 years and mean handicap was eight).

Lynn et al. (2013) showed that skilled golfers had greater pelvic acceleration and increased time between peak pelvic velocity and ball contact when compared to less skilled golfers. Pelvic acceleration was also shown to correlate with drive distance and smash ratio (Lynn et al., 2013) (Smash ratio is a measure of the amount of energy transferred from the club head to the ball). The greater the smash ratio, the better the energy transfer (ideal smash ratio=1.5) (Trackman, 2020). Crews and Lutz (2007) showed that professional golfers have greater linear club head speed at impact compared to amateur golfers. These studies give valuable insight into the swing differences between recreational and elite golfers, but they do not give information as to which variables are important to maximise performance amongst elite golfers. In the study by Lynn et al. (2013) correlations were performed on the pooled results from the amateur and professional golfers and therefore the findings should not be generalised to elite golfers. That is, increased pelvic acceleration, for example, in a group that contains both elite and recreational golfers may improve distance and smash ratio, but the same may not be true when the group is narrowed to include only elite golfers.

In order to address the question of whether the above parameters predict performance in young elite golfers the relationship between competition performance (measured by a yearly order of merit score) and swing parameters needs to be assessed. In particular, competition performance should be compared to swing characteristics (C-posture, S-posture, sway, slide, restricted hip turn, reverse spine angle, early extension), X-factor (In this study the X-factor was determined by measuring the rotation of the thorax relative to the pelvis at the top of the backswing (Brown et al., 2013)), club and ball performance (carry distance, roll distance, total distance, ball speed and club head speed) in young elite golfers.

6.2 Materials and Methods

6.2.1 Study design and setting

The study took place at an elite golf academy in Johannesburg, South Africa. This was a cross-sectional study. An overview of this study is shown in Figure 6.1.

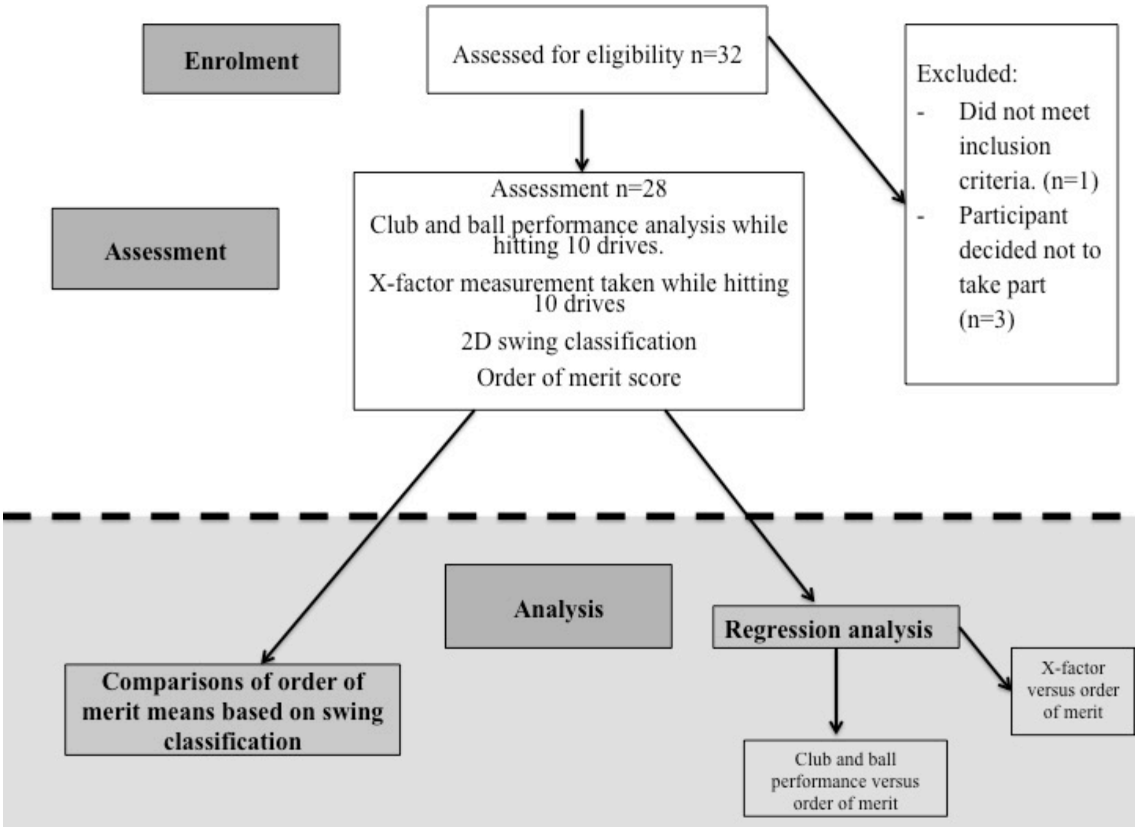

Figure 6.1: Overview of the enrolment, assessment and analysis

6.2.2 Participants

All the golfers enrolled at The Golf School of Excellence, who were classified as elite golfers and taking part in the yearly order of merit competition were invited to take part in the study. As some of the golfers were junior golfers (under 18 years old), a handicap cut off was not applied in the definition of an elite golfer. Further inclusion criteria included being injury free at the time, male and between the ages of 16 years to 30 years old. Due to limited accessibility to female golfers, we were unable to focus the study on female golfers. It was also not possible to combine female golfers performance with male golfers measurements as women and men perform differently. Data were collected from Feb 2017 to Jan 2018.

6.2.3 Ethics

Ethical clearance was granted from the Ethics Committee of the University of the Witwatersrand. The study was undertaken according to the ethical standards and guidelines of the Declaration of Helsinki. Identifiable information was removed from the dataset and study numbers were allocated to each participant. The link between the study numbers and identifiable information was kept in an access controlled file to which only the researcher had access.

Written informed consent was granted by participants over the age of 18 years old. Written informed assent was granted by participants under the age of 18 years old and informed written consent was granted by their legal guardians for them to take part in the study. An information sheet was provided to the parents/guardians by the Golf School of Excellence. Minors (participants under 18 years old) were only approach for inclusion in the study if their parents/guardian had given permission for them to take part in the study

Minors were included in this study as a high volume of sports participation has been shown to increase the risk of developing lower back pain in childhood and adolescence (Sato et al., 2011). Research involving minors is permitted if it is therapeutic research in the best interests of the minor (Strode and Slack, 2015). The inclusion of minors in this study was approved by the University of the Witwatersrand’s Human Research Ethics Committee

6.2.4 Procedure

Each golfer attended an assessment. There were various aspects to this cross-sectional assessment, which are detailed below. At this assessment the golfers were asked to complete a demographic questionnaire. Anthropometric measurements were also taken. The golfers provided the following personal and demographic details: full name, address, contact numbers, email address, handicap.

The golfers also underwent a swing performance assessment. Participants were asked to hit the golf ball with their driver 10 times as they would during a competition where both distance and accuracy were important. Club and ball performance was assessed using the Flightscope X2 Elite system (Flightscope X2 Elite, Flightscope (Pty) Ltd, Stellenbosch, South Africa). This is a three dimensional Doppler tracking radar designed for golf specific application (https://flightscope.com/company/technology/ accessed: 21 Aug 2019) as per Leach et al. (2017). The flight scope data was recorded as an average of 10 swing cycles. The flight scope data recorded carry distance (meters), roll (meters), total distance (meters), ball speed (km/h) and club head speed (km/h). During this swing assessment golfers also underwent kinematic swing analysis, to determine X-factor. X-factor angles were obtained using the Xsens inertial sensor motion analysis system (MVN Link Biomech system; Xsens Technologies B.V., Enschede, The Netherlands). X-factor was determined as trunk angular separation between the torso and pelvis at the top of the backswing (Titleist Performance Institute, 2013). The Xsens system consisted of 17 wireless motion trackers, which captured movement at 240 Hz. This system has been shown to be yield equivalent data to of other kinematic systems (Poitras et al., 2019). Unlike laboratory movement analysis systems, the Xsens system allows for data capture in a natural golf environment.

Golfer swing classification was assessed by the golfer’s coaches using two-dimensional video analysis. All the coaches were certified PGA (Professional Golf Association) professional golf coaches or PGA apprentices at the Golf school of Excellence. Golfer’s swings were characterised as having an: s-posture at address, c-posture at address, sway, slide, restricted hip rotation, early extension or reverse spine angle. S-posture was defined as excessive curvature in the lower back (lumbar lordosis) at set up

(Figure 6.2) (Titleist Performance Institute, 2021h). Swing characteristics were chosen in consultation with Michael Balderstone, the Managing Director and Performance Director at the Golf School of Excellence.

Figure 6.2: S posture at address

C-posture was defined as a set up position in which the golfers shoulders are slumped forward and their thoracic spine appears rounded (thoracic kyphosis) (Figure 6.3) (Titleist Performance Institute, 2021h).

Figure 6.3: C posture at address

A sway was defined as excessive lower body lateral shift away from the target during the backswing (Figure 6.4) (Titleist Performance Institute, 2021h).

Figure 6.4: Sway swing characteristic

A slide was defined as excessive lower body lateral shift towards the target during the downswing (Figure 6.5) (Titleist Performance Institute, 2021h).

Figure 6.5: Slide swing characteristic

Early extension was defined as a 'forward thrust of the lower body towards the ball during the downswing' (Figure 6.6) (Titleist Performance Institute, 2021h).

Figure 6.6: Early extension swing characteristic

Restricted hip rotation was defined as reduced hip rotation during the swing (Figure 6.7).

Figure 6.7: Restricted hip swing characteristic

A reverse spine angle was defined as excessive trunk lateral flexion towards the target during the backswing (Figure 6.8) (Titleist Performance Institute, 2021h).

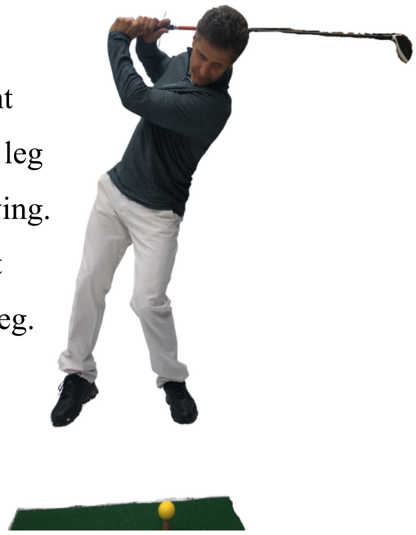

Figure 6.8: Reverse spine angle

2-Dimensional (2D) swing analysis was chosen over 3-Dimensional (3D) swing analysis, due to the restricted accessibility and technical difficulty associated with of 3D analysis. Appropriate 3D analysis can only be performed at selected tertiary institutions and is not readily available to the majority of elite golfers in South Africa. I wanted my findings to be useful to golf coaches who train elite golfers without access to 3D golf swing analysis. X-factor was the only 3D kinematic variable assessed. The reason this was included was that many training facilities in South Africa do have simple kinematic tools that are able to assess X-factor. Flightscope analysis was similarly chosen as many South African coaches have access to this means of measurement.

Golfer performance was ranked using the academy 2017 yearly order of merit score. The golfers played in a weekly order of merit tournament, which resulted in a yearly order of merit score (the sum of the weekly order of merit scores results in a yearly order of merit score). The yearly order of merit score was determined using the following measures: true score, stroke average, fairways hit, greens hit, number of putts, scrambling (this is a score that measures how often a golfer scores par or better after missing a green in regulation) and ups and downs (a golfer is said to have achieved an 'up and down' if he takes only two strokes to get his ball into the hole when his ball is resting around the green or in a greenside bunker). The reason the yearly order of merit was selected over handicap is that it standardised the courses that the golfers played on (it is easier to obtain a lower handicap if the golfer selects easier courses to play on). The reason competition performance was chosen over club performance parameters (such as club head speed, driver distance or smash ration) was that the more meaningful measurement for golfers is how they perform in a competition and not how well they hit the ball e.g. Swing characteristics may not affected ball parameters (club head speed, driver distance or smash ration), but they may impact upon competition performance.

6.2.5 Statistical analysis

Data was analysed using Statistica® Version 12 (StatSoft Inc, Tulsa, USA). Data was established using the z score. In order for the data to be normally distributed ($\frac{Kurtosis\ score}{standard\ error\ kurtosis\ score}$ and $\frac{Skew\ score}{standard\ error\ Skew\ score}$)= -1.96 to 1.96 (Kim, 2013). Normally distributed data was marked with a ‘n’ in the tables. The order of merit data was not normally distributed therefore a Spearman Rho test (simple regression) was performed to determine strength and direction of relationship between continuous data and order of merit (Schober et al., 2018). Rho=0 indicates no relationship; Rho=-1 or +1 indicates perfect monotonic relationship (Schober et al., 2018).

Mann Whitney U test were performed to determine differences between order of merit for those with the swing characteristic and those without the swing characteristic (Table 6.2). Normally distributed data was presented as means and standard deviations. Ordinal data and data that was not normally distributed was presented as medians and interquartile ranges. Effect sizes and confidence intervals were calculated. Effect sizes for non-parametric data was presented as probability of superiority ($P_{a,b}$) $P_{a.b} = \frac{U}{n_a n_b}$ (Pautz et al., 2018a). Effect sizes were interpreted as 0.2 (small), 0.5 (moderate) and 0.8 (large) (Cohen, 1992). Although $p<0.05$ is shown in the tables for comparative reporting, a dichotomised cut-off of $p<0.05$ was not applied to this study. The term ‘statistically significant’ was not used. p values were reported as continuous quantities (Wasserstein et al., 2019). Effect sizes, p values and confidence intervals were taken into account when showing a difference between groups in this paper (Pautz et al., 2018a, Pautz et al., 2018b). The details of comparisons made are shown in Figure 6.9.

Biomechanical studies typically involve small sample sizes. This is largely due to the amount of time it takes to collect data for one participant. Instead of conducting a formal sample size to power the study, p values, confidence intervals and effect sizes were reported. This approach has been used in other biomechanical studies e.g. single leg squat study published by Olivier et al. (2019).

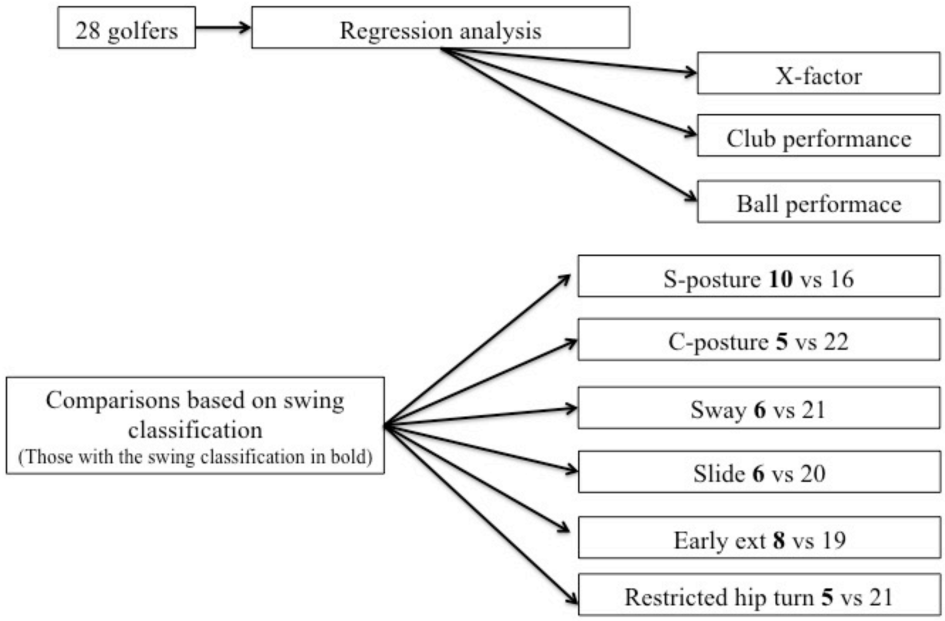

Figure 6.9: Details of between group comparisons made

6.3 Results

The participants age (years) mean (SD) = 19.93(1.44); body mass index (kg/m^2) mean (SD) = 24.60 (3.90); handicap mean (SD)= 2.39(2.77)); order of merit (position) median (IQR) = 24.96 (29). The participant's X-factor ranged from 24.70 degrees to 56.39 degrees.

In terms of swing characteristics 10 golfers had a S-posture at address, five had a C-posture at address, six had a sway, six had a slide, five had restricted hip turn, eight had early extension and two had a reverse spine angle (Table 6.1). As only two golfers had a reverse spine angle, it was not possible to include this swing characteristic in the analysis.

Table 6.1: Swing characteristics frequency table

Variable	Total number	Number No	Percentage No	Number Yes	Percentage Yes
S posture	26	16	61.54	10	38.46
C posture	27	22	81.48	5	18.52
Sway	27	21	77.78	6	22.22
Slide	26	20	76.92	6	23.08
Restricted hip rot	26	21	80.77	5	19.23
Early extension	27	19	70.37	8	29.63

There was no difference in order of merit between those with the S-posture at address (p=0.42, effect size=0.4), or a C-posture at address (p=0.93, effect size=0.49), or a sway (p=0.63, effect size=0.43), or a slide (p=0.98, effect size=0.49), or restricted hip turn (p=0.90, effect size=0.48), or early extension (p=0.59, effect size=0.43) and those without the corresponding swing classification (Table 6.2).

Table 6.2: Categorical data comparison of order of merit differences between elite golfers with the swing characteristic (S posture, C posture, sway, slide, restricted hip rotation and early extension) versus those without the swing characteristic.

Variable	Group without swing characteristic			Group with swing characteristic			Comparison between groups	
	Order of merit median (IQR)	Order of merit 95% CI lower	Order of merit 95% CI upper	Order of merit median (IQR)	Order of merit 95% CI lower	Order of merit 95% CI upper	p-value	Effect size
S posture	18.50 (28.75)	13.13	30.37	26.00 (25.50)	16.90	35.10	0.42	0.4
C posture	19.50 (25.25)	17.52	30.39	27.00 (36.50)	1.44	47.36	0.93	0.49
Sway	20.00 (28.50)	16.65	30.11	22.50 (32.75)	8.93	43.74	0.63	0.43
Slide	23.00 (32.50)	16.86	32.44	22.00 (14.00)	14.13	33.21	0.98	0.49
Restricted hip rot	19.00 (31.50)	16.55	31.16	27.00 (20.00)	8.96	35.44	0.90	0.48
Early extension	20.00 (31.00)	15.82	30.50	21.00 (27.75)	13.63	38.63	0.59	0.43

Table notes:. Data was presented as medians and interquartile range (IQR). p values were calculated using Mann Whitney U tests. n=number of participants. Spearman Rho test (simple regression) was performed to determine strength and direction of relationship.

In terms of ball and club performance there was a negative association between carry distance ($p=0.01$; $rho=-0.47$) and order of merit (Table 6.3) i.e. the longer the drive the lower the order of merit (the better the competition performance). There was also a negative association between total distance ($p<0.00$; $rho=-0.53$) and order of merit (Table 7.3). i.e. the further the ball was hit the lower the order of merit (the better the competition performance). There was no association was seen between competition performance (measured by order of merit) and roll ($p=0.91$), ball speed ($p=0.43$) and club head speed ($p=0.69$) (Table 6.3). There was no relationship between competition performance (measured by order of merit) and X-factor rotation ($p=0.25$) (Table 6.3).

Table 6.3: Relationship between order of merit and X-factor; order of merit and carry distance; order of merit and roll; order of merit and total distance; order of merit and ball distance; order of merit and club head speed; and order of merit

Variable	Mean (SD) or Median (IQR) n=27	95% CI lower	95% CI upper	Spearman coefficient	p value
X factor (degrees)^{n}	-38.70 (7.64)	-41.72	-35.68	0.23	0.25
Carry dist. (meters)^{n}	213.43 (21.73)	205.01	221.86	-0.47	0.01
Roll distance (meters)	19.19 (20.04)	16.07	28.03	0.02	0.91
Total distance (meters)^{n}	235.43 (18.60)	228.22	242.65	-0.53	0.00
Ball speed (km/hour)	238.66 (17.15)	221.40	241.37	-0.16	0.43
Club head speed (km/hour)	176.83 (13.98)	164.43	179.18	-0.08	0.69

Table notes: ^{n} Indicates that the variable was normally distributed. CI= confidence interval. n= number of participants. SD= standard deviation. IQR= interquartile range.

6.4. Discussion

6.4.1 Discussion of results

The results of this study suggest that the longer the drive (measured using total distance or carry distance), the better the overall golfing performance in young elite male golfers. To the author's knowledge, this is the first study that demonstrates that the range practice drive distance of young elite male golfers is positively associated with golfer competition performance (i.e. the longer the average drive on the driving range, the better the overall golfer competition performance). This is consistent with recent evidence on professional golfers claiming that the golf drive distance is the preeminent skill in professional golf (Alexander and Kern, 2005). The importance of the golf drive can be seen in the PGA (Professional Golfer's Association) Tour results with the top ten drivers in the PGA earning 30% more money than the top ten putters (Alexander and Kern, 2005). Increasing golf drive distance has been a focus of golfers and coaches as golf courses have been built longer and old courses have been lengthened thereby favouring golfers with long drives (Alexander and Kern, 2005). Improvements in club design has also seen an improvement in accuracy relative to drive distance (Wiseman and Chatterjee, 2006). This means that a golfer is able to hit the ball further with greater accuracy.

In this study differences in X-factor and swing characteristics amongst elite male golfers did not influence performance. These findings on X-factor are consistent with a study conducted by Cheetham et al. (2001) that showed no difference in X-factor between highly skilled golfers (handicap below 1) and less skilled golfers (handicap above or 14). They did, however, observe a difference in X-factor stretch between the highly skilled golfers and the less skilled golfers (Cheetham et al., 2001). |These findings are not, however, consistent with the studies conducted by Myers et al. (2008) and Chu et al. (2010) on middle aged recreational golfers, which indicates that the findings from performance studies on recreational golfers might not be generalisable to elite golfers. Similarity the results of this study should only be applied to young elite male golfers.

Another possible explanation for the difference in findings between this study and those conducted by Myers et al. (2008) and Chu et al. (2010) was that this study used 2D analysis, whereas the studies performed by Myers et al. (2008) and Chu et al. (2010) used 3D analysis which is a more sensitive tool. The reason 2D analysis was selected over 3D analysis was that I wanted the findings to be useful to coaches in South Africa, who by and large do not have access to 3D dimensional analysis. A second reason that 2D analysis was used was that it might be more reflective of clinically meaningful change. A difference in swing characteristics that cannot be observed by a coach using 2D analysis may be statistically relevant, but it may not reflect a clinically meaningful difference. More research is required into what constitutes a clinically meaningful change in golf swing characteristics in elite golfers.

The variable that impacted overall golfing performance was how far the ball was hit. This would imply that swing characteristics modifications and changes to X-factor can be made without impacting performance, provided the total ball distance does not reduce. This is particularly important in young elite golfers who have high rates of low back pain (Smith et al., 2018) and chronic back pain has been shown to be a major contributor to years lived with disability (Vos et al., 2012). This means that coaches and physiotherapists wanting to alter swing characteristics due to injury risk or pain should do so, provided they continue to monitor drive distance while implementing the changes so as not to hamper performance. These changes to swing biomechanics may reduce back pain and improve golf longevity.

6.4.2 Future research

Three-dimensional studies on the effect of swing kinematics on performance in young elite golfers should be conducted.

6.4.3 Limitations

The Flightscope assessment was performed outdoors on the range, therefore weather conditions such as wind speed may have affected the Flightscope measurements. This setting was selected above a laboratory setting as it was felt that this would reduce the anxiety that golfers (especially juniors) may experience during the assessment.

Only a small number of golfers had a reverse spine angle, therefore the results of this study cannot be generalised to this swing characteristic.

It is possible that other factors related to performance such as injury incidence and emotional well-being may have affected the 2017 yearly order of merit score and these should be considered possible confounding variables.

It should also be noted that some of the golfers played with more than one swing characteristic and that the performance effects of the one characteristic (e.g. sway) may have influenced by another swing characteristic (e.g. slide). Due to the small sample size it was not possible to perform multiple regression analysis.

6.5 Conclusion

Drive distance was associated with improved competition performance in young elite male golfers. X-factor and swing characteristics (c-posture, s-posture, sway, slide, restricted hip rotation, early extension) of elite male golfers did not influence performance. Coaches and physiotherapists wanting to alter swing characteristics due to injury risk or pain should do so, provided they continue to monitor drive distance while implementing the changes so as not to hamper performance. These changes to swing biomechanics may reduce back pain and improve golf longevity.

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LINKING PARAGRAPH BETWEEN CHAPTER 6 AND THE CONCLUSION

Chapter 6 highlighted the importance of person centred care. It is essential to view prevention of lower back pain within the golfer's primary aim, which is to play excellent golf. Undergoing an intervention to reduce lower back pain, such as swing modification, may reduce injury, but it is essential that the golfer is still able to play at golf at his highest possible level, which is his primary objective.

This previous chapter (chapter 6) provided an important final piece of the jigsaw. It enabled athletes, coaches and physiotherapists to perform a risk benefit analysis of the findings presented in chapter 3 and chapter 4. If coaches or physiotherapists identify that young elite male golfers are at risk of lower back pain based on their swing kinematics (reduced non-dominant leg squat at set up and at the top of the backswing, increased dominant knee flexion during impact and decreased dominant hip abduction during the backswing and during follow through) or their muscle activity throughout the swing (increased dominant *latissimus dorsi* and dominant *rectus abdominis* muscle activity during their golf swing) then they can consider recommending swing modifications aimed at addressing the risk factor identified, provided the golfer's golf drive distance is monitored and is not negatively affected throughout the process.

Now all that is left, is to view the whole puzzle from a distance. The final chapter, chapter 7, provides an overview of the material presented and summarises how I achieved the objective of research, which was, 'to identify the musculoskeletal factors associated with lower back pain' so as to fulfil the second step of the TRIPP model of injury prevention.

CHAPTER 7: CONCLUSION

7.1 Conclusion

Lower back pain is a considerable problem for young elite male golfers (Smith et al., 2018). It is essential to address this problem. Lower back pain prevalence amongst young elite male golfers is concerning as it increases the risk of chronicity, and lower back pain is one of the biggest causes of disability in the world and the burden of the disease is set to increase, especially in the developing world (Hoy et al., 2015). Until now the focus of much of the literature has been on older recreational golfers. Young, elite male golfers are an under-researched population. This research broadens the knowledge base about this population of sportsmen and provides the second step to injury prevention as outlined in the TRIPP model of injury prevention, namely, identifying the factors associated with injury.

In order to prevent lower back pain, it is essential to identify the risk factors associated with injury development (Emery and Pasanen, 2019). Up until this point, research focused on the differences between kinematic and muscle recruitment strategies between golfers with back pain versus those without back pain (Smith et al., 2018). Although these cross-sectional studies provided valuable insight into the differences between golfers with lower back pain and those without lower back pain, they did not inform if these changes were as a result of lower back pain at the time or as a precursor to developing lower back pain (chicken or egg situation). In order to determine if these factors were associated with developing lower back pain, baseline measurements should be taken before the golfer develops lower back pain. He should then be monitored to see if he develops lower back pain and then post assessment group allocation can occur so that comparisons can be made of the baseline data. The first two studies presented in this thesis are novel longitudinal cohort studies that take on such an approach.

Both swing kinematics and muscle recruitment strategies during the swing were identified as associated with developing lower back pain in young elite male golfers. The following musculoskeletal variables were associated with developing lower back pain in elite golfers: reduced non-dominant leg squat at set up and at the top of the backswing; increased dominant knee flexion during impact; decreased dominant hip abduction during the backswing and during follow through; increased dominant *latissimus dorsi* activation throughout the swing; and increased dominant *rectus abdominis* activation throughout the swing.

Identifying golfers at risk of lower back pain is an important step in jury prevention so that targeted interventions aimed at reducing the risk of injury can be implemented. The kinematic and EMG swing assessment techniques described in chapter 3 and chapter 4, may be means of identifying golfers at risk of developing lower back pain, but these assessment tools are expensive and require extensive additional training to perform. Functional movement screening has been proposed as a possible cost effective way to identifying golfers at risk of injury. Therefore the third study of this thesis (chapter 5) assessed the ability of functional movement tests to identify golfers at risk of injury. This study showed that functional movement screening tools appear to have questionable efficacy in identifying golfers at risk of developing injury.

Interventions for this group of golfers (those identified as being at high risk of developing lower back pain due to their swing kinematics or muscle activation) may include exercises and drills focused on modifying their swing to a less injury provoking movement pattern. Before embarking on altering an elite golfer's swing, it is important to know the performance impact of any such intervention so that the golfer, coach and physiotherapist can carefully weight up the performance risk versus the possible reduced risk of lower back pain. A young elite male golfer's primary objective is to play good golf. An intervention that may compromise this goal, no matter how good it is at preventing lower back pain, will not be tolerated by elite golfers. The last study showed that drive distance was an important performance variable and was associated with improved competition performance in young elite male golfers. X-factor and swing characteristics (c-posture, s-posture, sway, slide, restricted hip rotation, early extension) did not influence performance.

By identifying the musculoskeletal associations with developing lower back pain in young elite male golfers, an excellent foundation has been set for future researchers to build upon as they embark on the next steps in the TRIPP model of injury prevention, namely, randomised controlled trails aimed at addressing the identified risk factors.

7.2 Clinical Implications

Despite the popularity of functional movement screening in elite golf, study three (chap 5) showed this to have questionable efficacy and accordingly should not be used to screen young elite male golfers at risk of injury. Functional movement screening, does, however, provide valuable information regarding elite athletes muscle function and length, and as per the International Olympic committee recommendations it should continue to be used for this purpose (Ljungqvist et al., 2009).

Based on the findings of chapter 3, chapter 4 and chapter 5, I would like to highlight the usefulness of 3D kinematic analysis in order to identify golfers at risk of developing lower back pain. I cautiously recommend that elite golfers should have: i) at set up: at least four degrees of non-dominant ankle dorsiflexion; ii) at the top of the backswing: at most six degrees of dominant hip adduction; at least 43 degrees of non-dominant knee flexion; at least 22 degrees of non-dominant ankle flexion; iii) at impact dominant knee flexion should be at most 29 degrees; iv) at follow through: at least 2 degrees of dominant hip abduction; v) at most 15 %MVC muscle activity of dominant *rectus abdominis* throughout the swing; vi) at most 10 %MVC muscle activity of dominant *latissimus dorsi* activation throughout the swing . These values should be noted with caution, as this is a small sample size, but in the absence of other guiding data, such benchmark data may be of value to practitioners. These values could initially be used as a guide and then refined or confirmed by future studies.

Coaches and physiotherapists wanting to alter swing characteristics due to lower back pain risk should do so, provided they continue to monitor drive distance while implementing the changes so as not to hamper performance. These changes to swing biomechanics may have the potential to reduce lower back pain and improve longevity in elite golf.

7.3 Future research

According to step two of the screening model proposed by Bahr (2016), future research should use the pre-determined cut offs mentioned above for: reduced non-dominant leg squat at set up and at the top of the backswing; increased dominant knee flexion during impact; decreased dominant hip abduction during the backswing and during follow through; increased dominant *latissimus dorsi* activation throughout the swing; increased dominant *rectus abdominis* activation throughout the swing. Injury free golfers should be screened using these cut offs and then monitored for lower back pain incidence to determine if these variables were able to identify golfers at risk of developing lower back pain.

According to the TRIPP model of injury prevention, now that the following injury associations have been identified: i) reduced non-dominant leg squat at set up and at the top of the backswing; ii) increased dominant knee flexion during impact; iii) decreased dominant hip abduction during the backswing and during follow through; iv) increased dominant *latissimus dorsi* activation throughout the swing; v) and increased dominant *rectus abdominis* activation throughout the swing; intervention studies should be designed that aim to address these deficiencies in golfers who present with them. These studies should ideally be randomised controlled trials. Examples of such interventions could include strengthening of primary hip abductors on the dominant side if a golfer has increased dominant leg adduction during the backswing or the follow through. A second example could include swing modification interventions using teaching aids, visual feedback (such as video analysis) or EMG biofeedback to facilitate: i) A reduction in dominant *latissimus dorsi* activation or dominant *rectus abdominis* activation throughout the swing; ii) An increase in non-dominant leg squat at set up and at the top of the backswing; iii) A reduction in dominant knee flexion at the point of impact; iv) An increase in dominant hip abduction during the backswing and during follow through.

7.4 Limitations

The assessment was performed outdoors. Weather conditions such as wind speed may have influenced Flightscope measurements, despite these having been taken within the first few centimetres of ball contact. This setting was chosen above a laboratory setting as it was felt that this would reduce the anxiety that junior golfers may experience during the assessment.

There were some differences in between groups at baseline. In study one (chapter 3), study two (chapter 4) and study three (chapter 5) there were differences in baseline body mass and body mass index between the lower back pain group and the group that did not develop lower back pain. Whilst I acknowledge the possible impact that these variables may have had on the results, I would like to argue that as in real life scenarios, it is not possible to account for every confounding variable.

It could be argued that the kinematic and EMG differences may be as a result of previous lower back pain episodes. It is true that spinal movement may change as a result of previous lower back pain episodes (van Dieën et al., 2017), but this does not detract from the observation that these particular movements were associated with developing lower back pain and were not as a result of pain at the time (they were not an antalgic movement pattern).

A second kinematic or EMG assessment was not performed after developing lower back pain and it is therefore not possible to tell if the identified swing kinematics and EMG differences between the lower back pain group and the uninjured group persisted after developing lower back pain. This was outside the scope of this thesis. The purpose of this thesis was to identify baseline characteristics associated with the development of lower back pain.

These studies were conducted on young male elite golfers and therefore cannot be generalised to young female elite golfers. In study four (chapter 6) only a small number of golfers had a reverse spine angle, therefore the results of this study cannot be generalised to this swing characteristic.

7.5 Concluding thoughts and personal statement

In this research I have explored the role of muscle activation (EMG) and the resultant movement (kinematics) in potentially predisposing young elite male golfers to lower back pain. Other factors may be important considerations into the future such as differing biophysical attributes linked to developing lower back pain, which include alterations in muscle size, composition and co-ordination (Hartvigsen et al., 2018); psychological factors such as depression, catastrophising, anxiety, fear avoidance and reduced self-efficacy (Hartvigsen et al., 2018); and socio-economic status (Hartvigsen et al., 2018).

Screening, another focus of this research, is one of a limited set of tools available to identify golfers at risk of developing lower back pain. It was hoped that functional movement screening would provide an inexpensive means of identifying golfers at risk of developing lower back. Unfortunately this was not the case. Hopefully, in the future, as the ever growing set of wearable devices increases, these devices may become more cost effective and accessible to all golfers, especially those in low to middle income countries.

The field of injury prevention in golf must move to incorporate considerations of performance maintenance if it is hoped to be adopted widely so as to fulfil the dreams of young golfers both in terms of injury prevention and performance.

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APPRENDICES

Appendix 1: Plagiarism declaration

PLAGIARISM DECLARATION TO BE SIGNED BY ALL HIGHER DEGREE STUDENTS

SENATE PLAGIARISM POLICY: APPENDIX ONE

I Samantha-lynn Quinn (Student number: 0100495K) am a student registered for the degree of Doctor of Philosophy in the academic year 2020.

I hereby declare the following:

- I am aware that plagiarism (the use of someone else's work without their permission and/or without acknowledging the original source) is wrong.
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- I have included as an appendix a report from "Turnitin" (or other approved plagiarism detection) software indicating the level of plagiarism in my research document.

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Appendix 2: Turnitin report

MOVEMENT, THE MUSCULOSKELETAL SYSTEM AND LOWER BACK PAIN IN YOUNG ELITE MALE GOLFERS

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Appendix 3: Ethical clearance certificate

R14/49 Mrs Samantha-lynn Quinn

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)

CLEARANCE CERTIFICATE NO. M1611122

NAME: (Principal Investigator)	Mrs Samantha-lynn Quinn
DEPARTMENT:	Physiotherapy The Golf School of Excellence, Huddle Park Golf Club, Linksfield, Johannesburg
PROJECT TITLE:	The Predictors of Back Injury in Elite Golfers
DATE CONSIDERED:	25/11/2016
DECISION:	Approved unconditionally
CONDITIONS:	
SUPERVISOR:	Benita Olivier
APPROVED BY:	Professor P Cleaton-Jones, Chairperson, HREC (Medical)
DATE OF APPROVAL:	15/02/2017

This clearance certificate is valid for 5 years from date of approval. Extension may be applied for.

DECLARATION OF INVESTIGATORS

To be completed in duplicate and **ONE COPY** returned to the Research Office Secretary in Room 301, Third Floor, Faculty of Health Sciences, Phillip Tobias Building, 29 Princess of Wales Terrace, Parktown, 2193, University of the Witwatersrand. I/we fully understand the conditions under which I am/we are authorized to carry out the above-mentioned research and I/we undertake to ensure compliance with these conditions. Should any departure be contemplated, from the research protocol as approved, I/we undertake to resubmit the application to the Committee. **I agree to submit a yearly progress report**. The date for annual re-certification will be one year after the date of convened meeting where the study was initially reviewed. In this case, the study was initially reviewed in November and will therefore be due in the month of November each year. Unreported changes to the application may invalidate the clearance given by the HREC (Medical).

Quinn
Principal Investigator Signature   Date 15.02.2017

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES

Appendix 4: Permission to conduct research at the Golf School of Excellence

TGSE

BSI BALDERSTONE Sports Institute

Balderstone Sports Institute (Pty) Ltd
t/a The Golf School of Excellence
122 Club Street, Huddle Park Golf & Recreation, Linksfield, Gauteng, RSA
Postnet Suite 264, Private Bag X26, Sunninghill 2157, Gauteng, RSA
E-mail: info@tgse-golf.com
Website: www.tgse-golf.com

19 October 2016

Dear University of the Witwatersrand Human Ethics Committee,

I, Michael Balderstone, Managing Director of The Golf School of Excellence, hereby give Samantha-lynn Quinn (ID: 8301310056084) permission to conduct her PhD study entitled;

"The predictors of back injury in elite golfers"

at The Golf School of Excellence situated at Huddle Park Golf and Recreation, Linkfield, Johannesburg.

Yours Sincerely,

MJ Balderstone

Michael Balderstone
Managing Director
Tel: +27 (0) 011 485 1067 / 011 485 1068
Cell: +27 (0) 82 448 0753
E-mail: michael@tgse-golf.com
Web: www.tgse-golf.com

Balderstone Sports Institute (Pty) Ltd
Registration Number: 2005 / 004717 / 07
Directors: M Balderstone

Appendix 5: Information sheet for participants

INFORMATION DOCUMENT

I, Samantha-lynn Quinn, a physiotherapist undertaking my PhD at the University of the Witwatersrand, would like to invite you to participate in my research study entitled, **"The Predictors of back injury in elite golfers".**

Dear TGSE (The Golf School of Excellence) golfer

I, Samantha-lynn Quinn, am doing research on back injuries sustained by elite golfers. Research is just the process used to learn the answer to a question. In this study we want to learn which musculoskeletal attributes and swing characteristics are related to injury. We also want to learn if socio-demographic (which includes: family history, body characteristics, nutritional needs, medical needs, medical history) training workload, and/or previous injury, are related to future risk of injury. If we know which factors predict injury, then we are able to identify golfers at risk and suggest rehabilitation protocols to prevention injury. The recording of training workload is beneficial, as the interpretation of findings will account for the role of training workload in injury risk.

Invitation to participate

I would like to invite you to participate in this research project. The project is scheduled to commence in February 2017 and finish in October 2017.

Who is eligible to take part in this study?

Elite golfers at TGSE aged 16-35 years who are injury free at the time of screening.

What is involved in the study?

Testing will be arranged at the Golf School of Excellence on a date and time suitable to your school schedule.

On the first day of testing, you will be asked to:

- <u>Step 1:</u> complete a socio-demographic questionnaire. This includes information such as: body characteristics, family history, medical history, training workload and previous exercise related injuries. This questionnaire involves personal questions. We will ensure anonymity by assigning you a study number. The key to the study numbers will be in the sole and secure possession of the principle investigator. You may omit questions you consider to be sensitive or prefer not to answer. This will take approximately 30-45 minutes to complete.

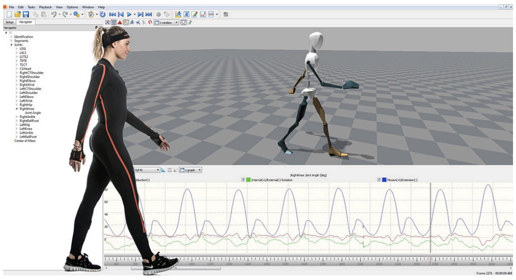
- Step 2: You will then be given time to warm up in your own preferred manner.
- Step 3: A musculoskeletal screening will then be performed. This will help determine your unique body attributes such as flexibility, strength and endurance. This will take approximately half an hour to complete.
- Step 4: Swing analysis: This will take approximately 1 hour to complete. This will include 2D and a 3D analysis of your golf swing. In order to do this biomechanical equipment will be used. This includes: i) a body suit that measures your joint angles (xsens MVN biomech link). This is a tight fitting lycra suit. You are permitted to wear shorts over the suit.
- 

Diagram : xsens body suit
(image taken from: https://www.xsens.com/wp-content/uploads/2014/11/MVN-BIOMECH-Link.jpg)

ii) a force plate that you stand on (Bertec Force Plate).

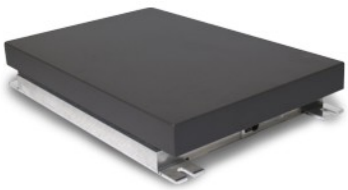


Diagram 2: Bertec force plate

(image taken from: http://www.velamed.com/files/force_plate1.jpg_)

iii) small EMG sensors that are attached to your skin by double sided tape (Trigono wireless EMG). These sensors give us information as to how your muscles are performing during the swing.

Diagram : Trigono wireless EMG http://www.jali.dreamhosters.com/site/wp-content/uploads/2016/01/tws-receivers-digitallab.png

iv) A video camera will be used to capture your swing. The videos will be stored on an external hard drive which will be password protected. The videos will be labelled with the study number only. Video recordings will be stored for a minimum of two years after publication or six years in the absence of publication.
v) Your swing performance will be measured using flightscope.

Diagram: Flight scope (Image taken from: https://static1.squarespace.com/static/56c2f264d210b85521c3f49f/57b7e268f5e231469b36ba93/57b7e458bebafbd8b058a445/1471669358046/FlightscopeGolf2015-069.jpg)

Completion of all the documents and the initial assessments should not take more than 2.5 hours.

For six months after the first assessment: you will be asked to monitor you injury status and training workload. Your training will be monitored by means of an application that you can load onto your cell phone. You will need to manually uploading your training weekly. If you get injured during the six months after the your initial assessment, you will be asked to contact Stacey Rosin (a qualified physiotherapist). She will make an appointment with you at a time that is convenient for you. She will assess your injury and classify your injury. This physiotherapy consult will be free of charge and will only involve assessment, and not treatment of your condition. The physiotherapy assessment (should you get injured) will not take more than half an hour. The weekly training schedule will take approximately 10 minutes per week to fill in.

Participation is voluntary and refusal to participate will not involve any loss of benefits to which you are otherwise entitled. You may withdraw from this study at any time. In order to withdraw from the study, you need to email the principal researcher. You do not need to provide a reason for withdrawal.

Are there any risks of taking part in this study?
You will not be exposed to any additional risks other than the risk that you are normally exposed to when training.

What are the benefits of taking part in this study?
Each golfer will receive a report. This report will include your functional movement screen score and a biomechanical assessment of your swing. Non-identifiable, generalised results will be made available to all participants at the end of the study. No monetary award will be given for taking part in this study.

Will information be handled as confidential?
Every effort will be made to keep personal information confidential. Absolute confidentiality cannot be guaranteed. Personal information may be disclosed if required by law. To ensure confidentiality of participants, each golfer will be assigned a study number, which will be your means of identification during the study. This number will be written on all data collection sheets including the questionnaire. Only the principal researcher will keep the key that links the participant to their number. This information will be stored on a password-protected laptop. Names of participants are to be written on the consent form. These consent forms will be kept separately from the questionnaires. All information will be used only for the purpose intended in this study only.

For further information, you are welcome to contact me on 083 226 6529 or send an email to samantha.l.quinn@gmail.com

For more information on, or for the reporting of ethical concerns, you may contact the chair of the Human Research Ethics Committee of the University of the Witwatersrand:

Prof Cleaton Jones; Tel: 011-717-2301, email peter.cleaton-jones1@wits.ac.za
Or
Ms Zanele Ndlovu/ Mr Rhulani Mkansi/ Mr Lebo Moeng Administrative Officers
011 717 2700/ 2656/ 1234/ 1252
Email: HREC-Medical.ResearchOffice@wits.ac.za

Kind regards

Samantha-Lynn Quinn
Physiotherapy Department
University of the Witwatersrand

Appendix 6: Video recording consent

VIDEO RECORDING CONSENT

Study title: The predictors of back injury in elite golfers

Dear Sir,

Please will you complete the following consent form if you are willing to participate in this study.

I am willing to participate in the study (please circle)

- Yes
- No

I___________________________________ (e-mail address:________________________ and cell phone number:___________________) hereby agree to participate in the study as described to me in the information sheet. I hereby agree to the video recording of my golf swing

I understand that the video recording will be used to analyse my golf swing and will only be accessible to the researcher and Michael Balderstone. The videos will be stored on an external hard drive, which will be password protected and the videos will be labelled with the study number only. Video recordings will be stored for a minimum of two years after publication or six years in the absence of publication

I am aware that I will not be exposed to any additional risks other than that usually experienced during my daily activities and that I can withdraw from the study at any time without suffering any repercussions. I understand that there are no monetary rewards for my participation and that participation is voluntary and I am not in any way obliged to take part.

Signature of Participant ______________________

Date_____________________________

Signature of Researcher _______________________

Appendix 7: Consent form

Consent form

Study title: The Predictors of back injury in elite golfers

Dear Sir,

Please will you complete the following consent form if you are willing to participate in this study.

I am willing to participate in the study (please circle)

- Yes
- No

I__________________________________ (e-mail address:_______________________ and cell phone number:___________________) hereby agree to participate in the study as described to me in the information sheet. I hereby agree to complete the data sheet to the best of my ability and to the extent with which I feel comfortable.

I am aware that I will not be exposed to any additional risks other than that usually experienced during my daily activities and that I can withdraw from the study at any time without suffering any repercussions. I understand that there are no monetary rewards for my participation and that participation is voluntary and I am not in any way obliged to take part.

Signature of Participant _____________________ Date ____________________________

Signature of Researcher ______________________

For office use only: Study number:___________________

Appendix 8: Information sheet for parents

This information sheet was provided to the parents/guardians by the Golf School of Excellence. Children were only approach for inclusion in the study if their parents had given permission to take part.

INFORMATION SHEET FOR PARENTS

INFORMATION DOCUMENT

I, Samantha-lynn Quinn, a physiotherapist undertaking my PhD at the University of the Witwatersrand, would like to invite your son to participate in my research study entitled, **"The Predictors of back injury in elite golfers".**

Dear TGSE (The Golf School of Excellence) parents

I, Samantha-lynn Quinn, am doing research on back injuries sustained by elite golfers. Research is just the process used to learn the answer to a question. In this study we want to learn which musculoskeletal attributes and swing characteristics are related to injury. We also want to learn if socio-demographic (which includes: family history, body characteristics, nutritional needs, medical needs, medical history) training workload, and/or previous injury are related to future risk of injury. If we know which factors predict injury, then we are able to identify golfers at risk and suggest rehabilitation protocols to prevention injury. The recording of training workload is beneficial, as the interpretation of findings will account for the role of training workload in injury risk.

Invitation to participate
I would like to invite your son to participate in this research project. The project is scheduled to commence in February 2017 and finish in October 2017.

Who is eligible to take part in this study?

Elite golfers at TGSE aged 16-35 years who are injury free at the time of screening.

What is involved in the study?

Testing will be arranged at TGSE on a date and time suitable with the school schedule.

On the first day of testing, your son will be asked to:

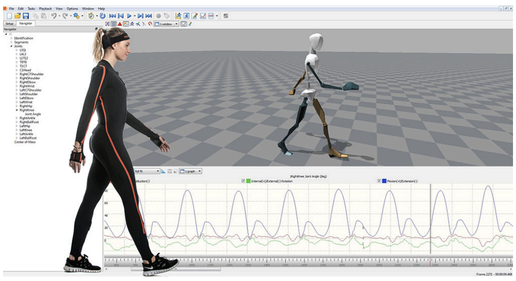
- Step 1: complete a socio-demographic questionnaire. This includes information such as: body characteristics, family history, medical history, training workload and previous exercise related injuries. This questionnaire involves personal questions. We will ensure anonymity by assigning your son a study number. The key to the study numbers will be in the sole and secure possession of the principle investigator. Your son may omit questions he considers to be sensitive or prefers not to answer. This will take approximately 30-45 minutes to complete.
- Step 2: Your son will then be given time to warm up in his own preferred manner.
- Step 3: A musculoskeletal screening will then be performed. This will help determine his unique body attributes such as flexibility, strength and endurance. This will take approximately half an hour to complete.
- Step 4: Swing analysis: This will take approximately 1 hour to complete. This will include 2D and a 3D analysis of your son's golf swing. In order to do this biomechanical equipment will be used. This includes: i) a body suit that measures your joint angles (xsens MVN biomech link). This is a tight fitting lycra suit. Your son is permitted to wear shorts over the suit.
- 

  Diagram : xsens body suit
  (image taken from: https://www.xsens.com/wp-content/uploads/2014/11/MVN-BIOMECH-Link.jpg)

  ii) a force plate that you stand on (Bertec Force Plate).

Diagram 2: Bertec force plate

(image taken from: http://www.velamed.com/files/force_plate1.jpg_)

iii) small EMG sensors that are attached to your son's skin by double sided tape (Trigono wireless EMG). These sensors give us information as to how your muscles are performing during the swing.

Diagram : Trigono wireless EMG http://www.jali.dreamhosters.com/site/wp-content/uploads/2016/01/tws-receivers-digitallab.png

iv) A video camera will be used to capture your son's swing. The videos will be stored on an external hard drive which will be password protected. The videos will be labelled with the study number only. Video recordings will be stored for a minimum of two years after publication or six years in the absence of publication.

v) Your son's swing performance will be measured using flightscope.

Diagram: Flight scope (Image taken from: https://static1.squarespace.com/static/56c2f264d210b85521c3f49f/57b7e268f5e231469b36ba93/57b7e458bebafbd8b058a445/1471669358046/FlightscopeGolf2015-069.jpg)

Completion of all the documents and the initial assessments should not take more than 2.5 hours.

For six months after the first assessment: your son will be asked to monitor his injury status and training workload. His training will be monitored by means of an application that he can load onto his cell phone. He will need to manually upload his training weekly. If he gets injured during the six months after his initial assessment, he will be asked to contact Stacey Rosin (a qualified physiotherapist). She will make an appointment with him at a time that is convenient for him. She will assess your son's injury and classify the injury. This physiotherapy consult will be free of charge and will only involve assessment, and not treatment of your son's condition. The physiotherapy assessment (should your son get injured) will not take more than half an hour. The weekly training schedule will take approximately 10 minutes per week to fill in.

Participation is voluntary and refusal to participate will not involve any loss of benefits to which your son is otherwise entitled. He may withdraw from this study at any time. In order to withdraw from the study, he will need to email the principal researcher. He does not need to provide a reason for withdrawal.

Are there any risks of taking part in this study?
Your son will not be exposed to any additional risks other than the risk that he is normally exposed to when training.

What are the benefits of taking part in this study?
Each golfer will receive a report. This report will include your son's functional movement screen score and a biomechanical assessment of your son's swing. Non-identifiable, generalised results will be made available to all participants at the end of the study. No monetary award will be given for taking part in this study.

Will information be handled as confidential?
Every effort will be made to keep personal information confidential. Absolute confidentiality cannot be guaranteed. Personal information may be disclosed if required by law. To ensure confidentiality of participants, each golfer will be assigned a study number, which will be your son's means of identification during the study. This number will be written on all data collection sheets including the questionnaire. Only

the principal researcher will keep the key that links the participant to their number. This information will be stored on a password-protected laptop. Names of participants are to be written on the consent form. These consent forms will be kept separately from the questionnaires. All information will be used only for the purpose intended in this study only.

For further information, you are welcome to contact me on 083 226 6529 or send an email to samantha.l.quinn@gmail.com

For more information on, or for the reporting of ethical concerns, you may contact the chair of the Human Research Ethics Committee of the University of the Witwatersrand:

Prof Cleaton Jones; Tel: 011-717-2301, email peter.cleaton-jones1@wits.ac.za
Or
Ms Zanele Ndlovu/ Mr Rhulani Mkansi/ Mr Lebo Moeng Administrative Officers
011 717 2700/ 2656/ 1234/ 1252
Email: HREC-Medical.ResearchOffice@wits.ac.za

Kind regards

Samantha-Lynn Quinn
Physiotherapy Department
University of the Witwatersrand

Appendix 9: Informed assent

Informed assent was obtained from all minor participants. The minor participants were not re-consented if they reached the age of majority during the time course of the empirical research work; or during the data storage period as this was not a requirement from the University of the Witwatersrand's Human Research Ethics Committee

INFORMED ASSENT

Study title: The Predictors of back injury in elite golfers

Dear Sir,

Please will you complete the following assent form if you are willing to participate in this study.

I am willing to participate in the study (please circle)

- Yes
- No

I___________________________________ (e-mail address:________________________ and cell phone number:____________________) hereby agree to participate in the study as described to me in the information sheet. I hereby agree to complete the data sheet to the best of my ability and to the extent with which I feel comfortable.

I am aware that I will not be exposed to any additional risks other than that usually experienced during my daily activities and that I can withdraw from the study at any time without suffering any repercussions. I understand that there are no monetary rewards for my participation and that participation is voluntary and I am not in any way obliged to take part.

Signature of Participant (minor) ______________________ Date

____

Signature of Researcher _______________________

For office use only: Study number:____________________

Appendix 10: Video recording assent

VIDEO RECORDING ASSENT

Study title: The predictors of back injury in elite golfers

Dear Sir,

Please will you complete the following consent form if you are willing to participate in this study.

I am willing to participate in the study (please circle)

- Yes
- No

I__________________________________ (e-mail address:_______________________ and cell phone number:___________________) hereby agree to participate in the study as described to me in the information sheet. I hereby agree to the video recording of my golf swing

I understand that the video recording will be used to analyse my golf swing and will only be accessible to the researcher and Michael Balderstone. The videos will be stored on an external hard drive, which will be password protected and the videos will be labelled with the study number only. Video recordings will be stored for a minimum of two years after publication or six years in the absence of publication

I am aware that I will not be exposed to any additional risks other than that usually experienced during my daily activities and that I can withdraw from the study at any time without suffering any repercussions. I understand that there are no monetary rewards for my participation and that participation is voluntary and I am not in any way obliged to take part.

Signature of Participant ____________________

Date___________________________

Signature of Researcher _____________________

Appendix 11: Video recording consent for minor child

VIDEO RECORDING CONSENT FOR MINOR CHILD

Study title: The predictors of back injury in elite golfers

Dear Sir/ Madam,

Please will you complete the following consent form if you are willing to allow your minor child to participate in this study.

I am willing to allow my minor child, ______________________________(Name, ID number) to participate in the study (please circle)

- Yes
- No

I__________________________________ (e-mail address:_______________________ and cell phone number:___________________) hereby agree to allow my child/ child of whom I am the legal guardian _________ __________________(ID: _______________) to participate in the study as described to me in the information sheet. I hereby agree to the video recording of my minor child's golf swing

I understand that the video recording will be used to analyse my golf swing and will only be accessible to the researcher and Michael Balderstone. The videos will be stored on an external hard drive, which will be password protected and the videos will be labelled with the study number only. Video recordings will be stored for a minimum of two years after publication or six years in the absence of publication

I am aware that I will not be exposed to any additional risks other than that usually experienced during my daily activities and that I can withdraw from the study at any time without suffering any repercussions. I understand that there are no monetary rewards for my participation and that participation is voluntary and I am not in any way obliged to take part.

Signature of Participant ______________________

Date_____________________________

Signature of Researcher _______________________

Appendix 12: Consent for minor child

CONSENT FOR MINOR CHILD

Study title: The Predictors of back injury in elite golfers

Dear Sir/Madam,

Please will you complete the following consent form if you are willing that your child participates in this study.

I am willing for my child to participate in the study (please circle)

- Yes
- No

I___________________________________ (e-mail address:________________________ and cell phone number:____________________) hereby agree to allow my child/ child of whom I am the legal guardian _________ __________________(ID:_______________) to participate in the study as described to me in the information sheet. He agrees to complete the data sheet to the best of his ability and to the extent with which I and he feels comfortable.

I am aware that my child will not be exposed to any additional risks other than that usually experienced during his daily activities and that I and he can withdraw from the study at any time without suffering any repercussions. I understand that there are no monetary rewards for his participation and his participation is voluntary and he is not in any way obliged to take part.

Signature of Parent ______________________ Date _____________________________

Signature of Researcher _______________________

For office use only: Study number:____________________

Appendix 13:Muscles requiring special focus: erector spinae, latissimus dorsi, rectus abdominis and external oblique.

The *erector spinae, latissimus dorsi, rectus abdominis and external oblique* muscles were selected for monitoring during this research as they are superficial trunk muscles that have been shown to be very active during the golf swing (Cole and Grimshaw, 2016).

The *erector spinae, latissimus dorsi, rectus abdominis and external oblique* are used extensively as prime movers or stabilisers of the lumbar spine (see individual muscle function discussion in A13.1-A13.4) therefore fatigue in these muscles as a result of repetitive movement may result in increased risk of developing pain in the lower back (Edwards et al., 2020). The concept of these muscles fatiguing with golf practice is supported by a study conducted on 14 golfers by Sorbie et al. (2017) which showed that the lumbar region of the *erector spinae* muscle experienced significant fatigue after a golf practice session.

In order to better understand the role that the *erector spinae, latissimus dorsi, rectus abdominis and external oblique* may play in the development of lower back pain in young elite male golfers, a closer look at the anatomy and function of these muscles is needed.

A13.1 Erector spinae muscle

The *erector spinae* muscle is the largest of the intrinsic back muscles. This muscle is found posterolateral to the vertebral column between the *spinous processes* medially and the angle of the rib laterally. They are covered by the *thoracolumbar* facia, *serratus posterior inferior*, *rhomboid* and *splenius* muscles. The *erector spinae* originates from a thick board tendon attached to the sacrum, the spinous processes of the lumbar and thoracic vertebrae and the *iliac crest*. It inserts depending on its division. The *erector spinae* muscle is divided into three muscles: *iliocostalis* (laterally), *longissimus* (centrally) and *spinalis* (medially). The *iliocostalis* is further divided regionally into *iliocostalis lumborum, iliocostalis thoracis* and the *iliocostalis cervicis*. The *iliocostalis* inserts into the angles of the ribs and the *transverse processes* of the lower cervical vertebrae. The *longissimus* is similarly divided regionally into the *longissimus thoracis, longissimus cervicis* and *longissimus capitis*. The *longissimus* muscle inserts into the base of the skull. The *spinalis* is also divided regionally into the *spinalis thoracis*, *spinalis cervicis* and *spinalis capitis*. The *spinalis* interconnects the spinous processes of adjacent vertebrae and attaches into the skull (Drake et al., 2009)

The *erector spinae* is the primary extensor of the vertebral column and the head. It acts bilaterally to extend the back and neck as well as eccentrically controlling trunk flexion. When acting unilaterally, the *erector spinae* contributes to lateral flexion of the trunk (Drake et al., 2009)

A13.2 Latissimus dorsi muscle

The *latissimus dorsi* muscle is a flat, large triangular muscle (Drake et al., 2009). It originates at the lower portion of the lumbar spine and tapers as it ascends into the narrow tendon that attaches into the *humerus* anteriorly (Drake et al., 2009). The primary movements caused by this muscle are adduction, extension and medial rotation of the upper limb. *Latissimus dorsi* can also act to prevent upward movement of the shoulder (Drake et al., 2009). Although this muscle is not a primary mover of the lumbar spine, it has broad attachments to the spinous processes and therefore there is ongoing research into the possible role this muscle has on trunk movement (including a possible role in spine extension, lateral flexion and rotation) (Jeno and Varacallo, 2020). A study by Ugale and Kothari (2020) on 100 participants showed a relationship between *latissimus dorsi* tightness and lower back pain.

A13.3 Rectus abdominis muscle

The *rectus abdominis* is a flat, long muscle. It extends the length of the anterior abdominal wall. It is a paired muscle that is separated by the linea alba. The *rectus abdominis* muscle's origin is from the *pubic crest*, *pubic tubercle* and *pubic symphysis* and it inserts into the costal cartilages of the ribs five to seven and the *xiphoid process*. The *rectus abdominis* acts to compress the abdominal content, flex the vertebral column and tense the abdominal wall (Drake et al., 2009).

A13.4 External oblique muscle

The *external oblique* is the most superficial of the three flat muscles of the anterolateral group of abdominal wall muscles. It is found immediately deep to the superficial abdominal fascia. The *external oblique* muscle originates from the outer surfaces of the lower eight ribs. The lateral muscle fibres of the external oblique muscle are placed in an inferomedial direction. When approaching the midline, the aponeuroses entwine and form the *linea alba*. The *linea alba* extends from the *pubic symphysis* to the *xiphoid process*. The *external oblique* acts to compress the abdominal contents and flex the trunk when acting bilaterally. When acting on one side, it laterally flexes the trunk towards the same side and turns the anterior part of the abdomen towards the opposite side (Drake et al., 2009).

Appendix 14: Additional Screening performed

Additional screening for lower limb injury, neck/thoracic and upper limb injury was performed on the same cohort of participants found in chapter 5. The methodology was identical to that described in chapter 5 and occurred concurrently.

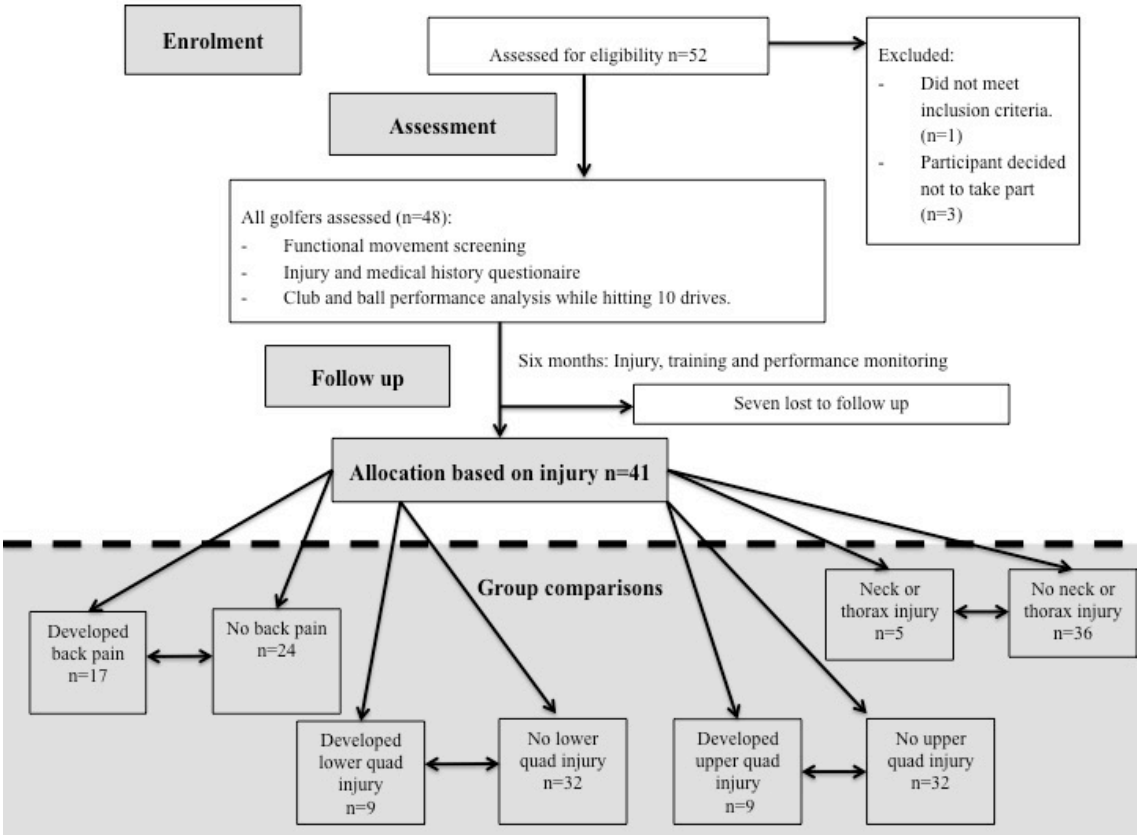


Figure A14.1: Cohort diagram for additional screening

A14.1: Participants and injury incidence

Nine participants developed pain in their upper limb (upper limb injury incidence of 22%). Three of which were to the shoulder region and six in the elbow, wrist and hand region. Nine participants developed pain in their lower limb, three of which occurred in the hip region, two in the knee and four in the ankle (lower limb injury incidence of 22%). Five participants developed pain in the neck and thoracic region (neck and thoracic spine injury incidence of 12%). It should be noted that some participants sustained injury in more than one area.

A14.2: Screening tests for lower limb pain

There was a difference between the group that developed lower limb injury and those who did not in the following screening tests: rotational stability test non dominant side ($p=0.01$; effect size=0.23), rotational stability test dominant side ($p=0.02$, effect size=0.24) (Table A14.2). The group that developed lower limb pain scored better in rotational stability tests than the group that did not go on to develop lower limb pain. There were no differences observed in any other screening tests for lower limb pain (Table A14.1- A14..3).

The group that developed lower limb pain had a lower handicap (mean (SD)= 0.43 (1.27)) versus the group that did not develop lower limb injury (mean (SD)= 3.05 (2.84); $p=0.01$; effect size=0.99). There were no other performance or training differences between the group that developed lower limb pain and the group that did not develop lower limb pain in terms of performance and training (Table A14.4).

There were no differences between lower limb groups in terms of baseline anthropometrics, medical history or injury history (Table A14.5-Table 15.6).

Table A14.1: Page 1 of 4: Table comparing categorical screening test results for those with lower limb pain compared to those who did not develop lower limb pain

Variable	Score	No lower limb pain group n	No lower limb pain per category n(%)	Lower limb pain group n	Lower limb pain per category n (%)	Comparison between no limb pain and lower limb pain group p value	Comparison between no lower limb pain and lower limb pain group effect size
Spinal extension clearing test	Pain	32	2 (6.25%)	9	1 (11.11%)	0.54	0.08
	No pain		30 (93.75%)		8 (88.88%)		
Spinal flexion clearing test	Pain	32	1 (3.13%)	9	0 (0%)	1	0.08
	No pain		31 (96.88%)		9 (100%)		
Pelvic tilt test posture at set up score	Neutral	32	28 (87.5%)	9	9 (100%)	0.54	0.17
	Excessive anterior		2 (6.25%)		0 (0%)		
	Excessive posterior		2 (6.25%)		0 (0%)		
Pelvic tilt at set up posture amount of motion score	Normal motion	32	19 (59.38%)	8	7 (87.5%)	0.48	0.25
	Limited posteriorly		4 (12.5%)		0 (0%)		
	Limited anteriorly		1 (3.13%)		0 (0%)		
	Both limited		8 (25%)		1 (12.5%)		
Pelvic tilt at set up posture quality of movement	Smooth movement	32	6 (18.75%)	9	3 (33.33%)	0.38	0.15
	Shaking movement		26 (81.25%)		6 (66.67%)		
Pelvic rotation stability score	Good stability	32	28 (87.5%)	9	8 (88.89%)	0.85	0.14
	Limited stability left side		2 (6.25%)		1 (11.11%)		
	Limited stability right side		1 (3.13%)		0 (0%)		
	Both sides stability limited		1 (3.13%)		0 (0%)		
Pelvic rotation test mobility score	Full mobility	32	19 (59.38%)	9	9 (100%)	0.15	0.36
	Limited mobility to the left side		1 (3.13%)		0 (0%)		
	Limited mobility to the right side		2 (6.25%)		0 (0%)		
	Both sides showed limited mobility		10 (31.25%)		0 (0%)		
Pelvic rotation quality of movement score	Good rotatory movement	32	19 (59.38%)	9	8 (88.89%)	0.13	0.26
	Lateral movement more than rotatory movement		13 (40.63%)		1 (11.11%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared test and Fishers exact test. n= number of participants

Table A14.1: Continued Page 2 of 4: Table comparing categorical screening test results for those with lower limb pain compared to those who did not develop lower limb pain

Variable	Score	No lower limb pain group n	No lower limb pain per category n(%)	Lower limb pain group n	Lower limb pain per category n(%)	Comparison between no limb pain and lower limb pain groups' p value	Comparison between no lower limb pain and lower limb pain group effect size
Torso rotation test stability score	Good stability	32	32 (100%)	9	8 (88.89%)	0.22	0.3
	Limited to the left side		0 (0%)		1 (11.11%)		
Torso rotation test mobility score	Full mobility	32	27 (84.38%)	9	9 (100%)	0.66	0.2
	Limited mobility to the left side		1 (3.13%)		0 (0%)		
	Limited mobility to the right side		1 (3.13%)		0 (0%)		
	Both sides showed limited mobility		3 (9.38%)		0 (0%)		
Torso rotation test quality of movement	Good rotatory movement	31	29 (93.55%)	9	8 (88.89%)	0.55	0.07
	More lateral movement		2 (6.45%)		1 (1.11%)		
Toe touch test score	Full range of motion	31	17 (54.84%)	9	8 (88.89%)	0.21	0.34
	Decreased toe touch on the left		3 (9.68%)		1 (11.11%)		
	Decreased toe touch on the right		1 (3.23%)		0 (0%)		
	Decreased toe touch bilaterally		10 (32.26%)		0 (0%)		
90 90 non dominant in standing	Standing <spine	31	4 (12.9%)	9	1 (11.11%)	0.55	0.17
	standing=spine		16 (51.61%)		3 (33.33%)		
	Standing>spine		11 (35.48%)		5 (55.56%)		
90 90 dominant in standing	Standing <spine	32	3 (9.38%)	9	2 (22.22%)	0.33	0.23
	standing=spine		19 (59.38%)		3 (33.33%)		
	Standing>spine		10 (31.25%)		4 (44.44%)		
90 90 shoulder in set up posture dominant score	Standing <spine	32	3 (9.38%)	9	2 (22.22%)	0.5	0.18
	standing=spine		16 (50%)		3 (33.33%)		
	Standing>spine		13 (40.63%)		4 (44.44%)		
90 90 shoulder in set up posture non-dominant score	Standing <spine	32	5 (15.63%)	9	2 (22.22%)	0.28	0.25
	standing=spine		20 (62.5%)		3 (33.33%)		
	Standing>spine		7 (21.88%)		4 (44.44%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared test and Fishers exact test. n= number of participants

Table A14.1: Continued Page 3 of 4: Table comparing categorical screening test results for those with lower limb pain compared to those who did not develop lower limb pain

Variable	Score	No lower limb pain group n	No lower limb pain per category n(%)	Lower limb pain group n	Lower limb pain per category n(%)	Comparison between no limb pain and lower limb pain group p value	Comparison between no lower limb pain and lower limb pain group effect size
Single leg bridge non-dominant score	Glutes normal	32	23 (71.88%)	9	9 (100%)	0.2	0.28
	Glutes weak		8 (25%)		0 (0%)		
	Glutes inhibited		1 (3.13%)		0 (0%)		
Single leg bridge dominant score	Glutes normal	32	26 (81.25%)	9	9 (100%)	0.37	0.22
	Glutes weak		5 (15.63%)		0 (0%)		
	Glutes inhibited		1 (3.13%)		0 (0%)		
Lower quarter rotation non-dominant: internal rot score	>60 degrees	32	14 (43.75%)	9	5 (55.56%)	0.47	0.19
	=60 degrees		14 (43.75%)		2 (22.22%)		
	<60 degrees		4 (12.5%)		2 (22.22%)		
Lower quarter rotation non-dominant: external rot score	>60 degrees	32	26 (81.25%)	9	8 (88.89%)	0.63	0.15
	=60 degrees		3 (9.38%)		1 (11.11%)		
	<60 degrees		3 (9.38%)		0 (0%)		
Lower quarter rotation dominant: internal rot score	>60 degrees	32	17 (53.13%)	9	5 (55.56%)	0.99	0.02
	=60 degrees		11 (34.38%)		3 (33.33%)		
	<60 degrees		4 (12.5%)		1 (11.11%)		
Lower quarter rotation dominant: external rot score	>60 degrees	31	21 (67.74%)	9	8 (88.89%)	0.43	0.2
	=60 degrees		8 (25.81%)		1 (11.11%)		
	<60 degrees		2 (6.45%)		0 (0%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared test and Fishers exact test. n= number of participants

Table A14.1 Continued Page 4 of 4: Table comparing categorical screening test results for those with lower limb pain compared to those who did not develop lower limb pain

Variable	Score	No lower limb pain group n	No lower limb pain per category n (%)	Lower limb pain group n	Lower limb pain per category n (%)	Comparison between no limb pain and lower limb pain group p value	Comparison between no lower limb pain and lower limb pain group effect size
seated trunk rot non-dominant score	>45 degrees	32	29 (90.63%)	9	9 (100%)	0.63	0.15
	=45 degrees		1 (3.13%)		0 (0%)		
	<45 degrees		2 (6.25%)		0 (0%)		
Seated trunk rot dominant score	>45 degrees	32	29 (90.63%)	8	8 (88.89%)	0.67	0.14
	=45 degrees		1 (3.13%)		0 (0%)		
	<45 degrees		2 (6.25%)		0 (0%)		
Seated trunk rot non-dominant: club behind back score	>45 degrees	32	29 (90.63%)	9	9 (100%)	0.63	0.15
	=45 degrees		1 (3.13%)		0 (0%)		
	<45 degrees		2 (6.25%)		0 (0%)		
Seated trunk rot dominant: club behind back score	>45 degrees	32	30 (93.75%)	9	9 (100%)	0.74	0.12
	=45 degrees		1 (3.13%)		0 (0%)		
	<45 degrees		1 (3.13%)		0 (0%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared test and Fishers exact test. n= number of participants

Table A14.2: Table comparing ranking screening test results for: those with lower limb pain compared to those without lower limb pain

Variable	No lower limb pain percentage per category n=32			Lower limb pain percentage per category n=9			Comparison between no lower limb pain and lower limb pain group p value	Comparison between no lower limb pain and lower limb pain group effect size
	Median (IQR)	Lower CI	Upper CI	Median (IQR)	Lower CI	Upper CI		
Single leg squat non-dominant score	2 (0.75)	1.60	1.96	1 (1)	1.04	1.85	0.15	0.34
Single leg squat dominant score	2 (0.75)	1.62	2.01	1 (1)	1.04	1.85	0.14	0.33
Deep overhead squat score	2 (1)	2.17	2.52	2 (1)	1.95	2.72	0.96	0.49
Hurdle step non-dominant score	2 (1)	2.14	2.48	2 (1)	1.88	2.56	0.7	0.45
Hurdle step dominant score	2 (1)	2.17	2.52	2 (0.5)	1.88	2.56	0.59	0.44
In line lunge non-dominant score	3 (1)	2.38	2.74	2 (1)	1.95	2.72	0.31	0.39
In line lunge dominant score	3 (1)	2.38	2.74	2 (1)	1.95	2.72	0.31	0.39
Trunk stability push up score	3 (0)	2.71	2.98	3 (0)	3	3	0.49	0.42
Rotational stability test non-dominant score	2 (0.75)	1.95	2.36	3 (0.5)	2.44	3.12	0.01	0.23
Rotational stability test dominant score	2 (1)	2.02	2.43	3 (0.5)	2.44	3.12	0.02	0.24
Plank score	5 (6)	3.48	5.49	3 (7)	1.62	7.05	0.97	0.45

Table notes: n= number of participants. Data presented as medians and interquartile range (IQR). p value calculated using Mann Whitney U test.

Table A14.3: Table comparing continuous screening test results for: those with lower limb pain compared to those without lower limb pain

Test	No lower limb pain				lower limb pain				Comparison between no lower limb pain and lower limb pain groups p value	Comparison between no lower limb pain and lower limb pain group effect size
	n	Mean (SD) or median (IQR)	95% CI lower	95% CI Upper	n	Mean (SD) or median (IQR)	95% CI lower	95% CI upper		
Straight leg raise non-dominant (range of motion)[n]	31	69.13 (15.86)	63.31	74.95	9	76.67 (10.55)	68.56	84.77	0.19	0.51
Straight leg raise dominant (range of motion)[n]	31	69.95 (13.93)	64.54	74.75	9	73.22 (13.19)	63.08	83.36	0.50	0.26
Sit ups (no.)[n]	32	38.25 (10.60)	34.42	42.07	9	34.33 (6.76)	29.13	39.53	0.30	0.39
Push ups (no.)[n]	32	37.34 (13.88)	32.34	42.35	9	35.22 (8.54)	28.66	41.79	0.67	0.16
Oblique sit up to non-dominant shoulder (no.)	31	30.39 (8.61)	27.23	33.54	9	29.78 (8.57)	23.19	36.37	0.85	0.47
Oblique sit up to dominant shoulder (no.)	31	28 (13)	25.15	32.40	9	28 (10.5)	22.89	31.33	0.63	0.47

Table notes: [n] indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations. Data not normally distributed presented as medians and interquartile range (IQR). p values calculated using independent T tests (normally distributed data) and Mann Whitney U tests (data not normally distributed). n=number of participants

Table A14.4: Comparison of baseline performance and training variables for the lower limb pain group versus the no lower limb pain group

Variable	No lower limb pain				Lower limb pain				Comparison between lower limb pain groups P value	Comparison between lower limb pain groups Effect size
	n	Mean (SD) or median (IQR)	95% CI lower	95% CI upper	n	Mean (SD) or median (IQR)	95% CI lower	95% CI upper		
Order of merit (rank)	21	27 (24)	20.95	33.43	7	9 (40)	0.32	36.25	0.16	0.32
Handicap (no) ^{n}	32	3.05 (2.84)	1.76	4.34	9	0.43 (1.27)	-0.75	1.61	0.01	0.99
Carry distance (m)^{n}	31	214.39 (20.78)	206.77	222.01	9	221.27 (24.45)	202.47	240.06	0.41	0.32
Roll distance(m)	31	12.7 (13.57)	12.3	21.46	9	11.09 (28.65)	6.76	38.67	0.95	0.49
Total distance (m) ^{n}	31	231.50 (18.69)	224.65	238.35	9	243.91 (15.95)	231.65	256.17	0.08	0.68
Ball speed(m)	31	238.72 (18.81)	221.63	242.61	9	241.30 (19.88)	209.79	253.32	0.92	0.49
Club head speed (km/h)	31	177.12 (17.57)	165.22	179.96	9	178.67 (13.69)	155.59	189.11	0.95	0.49
Putting practice per week (hours per week)	32	3.25 (3.8)	2.23	8.85	9	4.12 (6.12)	2.04	7.18	0.61	0.44
Short game practice per week (hours per week)	32	3.26 (4.68)	2.36	9.33	9	4.58 (5.42)	1.81	6.73	0.96	0.49
Range practice per week(hours per week)	32	6.06 (7.38)	4.94	12.08	9	2.7 (5.51)	1.24	8.08	0.14	0.33
Flexibility training per week (hours per week)	32	1.14 (1.67)	0.54	3.87	9	0.58 (2.37)	0.15	2.77	0.65	0.45
Postural training per week (hours per week)	32	0.42 (1.18)	0.01	3.36	9	0.15 (0.98)	-0.12	1.11	0.47	0.42
Weight training per week (hours per week)	32	2 (2.75)	1.47	5.26	9	0.75 (2.8)	0.05	3.12	0.20	0.35
Cardio per week (hours per week)	32	1.5 (1.61)	1.03	3.53	9	1.6 (1.88)	0.89	2.95	0.61	0.44
Rounds of golf played per week	32	2.96 (1.77)	-8.03	34.08	9	2.96 (2.4)	1.56	4.03	0.99	0.5

Table notes: : ^{n} indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations (SD). Data not normally distributed presented as medians and interquartile range (IQR). p values calculated using independent T tests (normally distributed data) and Mann Whitney U tests (data not normally distributed). n=number of participants. CI were displayed as calculated, but please be advised that it is not possible to obtain a negative value for the confidence interval for these values, except for handicap. The lowest possible value is zero

Table A14.5: Comparison of baseline anthropometric data lower limb injury group versus no lower limb injury group

Variable	No lower limb injury				Lower limb injury				Comparison between lower limb group p value	Comparison between lower limb group effect size
	n	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper	n	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper		
Age (years)^n	32	19.56 (2.7)	18.59	20.54	9	20.67 (2)	19.51	21.82	0.25	0.44
Height (m)	32	1.8 (0.13)	1.76	1.81	9	1.78 (0.06)	1.74	1.84	0.89	0.48
Body mass (kg)^n	32	76.68 (12.62)	72.13	81.23	9	82.78 (13.94)	72.06	93.5	0.22	0.47
BMI (kg/m^2)^n	32	23.95 (3.41)	22.72	25.18	9	25.78 (4.44)	22.36	29.2	0.19	0.5

Table notes: : ^{n} indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations (SD). Data not normally distributed presented as medians and interquartile range (IQR). p values calculated using independent T tests (normally distributed data) and Mann Whitney U tests (data not normally distributed). n=number of participants.

Table A14.6: Comparison of baseline medical history and history of injury: No lower limb injury group versus the lower limb injury group

Variable	No lower limb pain group n	No lower limb pain group n(%) with condition or health risk factor	Lower limb pain group n	Lower limb pain group n(%) with condition or health risk factor	p value for between group difference at baseline	Between lower limb group comparison Odds ratio	Risk ratio in lower limb pain group	Between group comparison effect size
Smoked at least 100 cigarettes in his lifetime	28	12 (42.86%)	8	4 (50%)	1	0.75	1.25	0.06
History vascular problems	27	1 (3.7%)	8	1 (12.5%)	0.41	0.27	2.36	0.16
History respiratory problems	25	5 (20%)	8	3 (37.5%)	0.37	0.42	1.88	0.18
History of cancer	27	1 (3.7%)	8	1 (12.5%)	0.41	0.27	2.36	0.16
History of musculoskeletal conditions	27	2 (7.41%)	8	2 (25%)	0.22	0.24	2.58	0.23
History of neurological conditions	27	3 (11.11%)	8	3 (25%)	0.57	0.38	2	0.17
History of gastrointestinal conditions	27	4 (14.81%)	8	1 (12.5%)	1	1.22	0.86	0.03
History of injury in past six months	26	14 (53.85%)	8	5 (62.5%)	1	0.7	1.32	0.07
History of lower limb injury in past six months	26	5 (19.23%)	8	1 (12.5%)	1	1.67	0.67	0.07

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared test and Fishers exact test. n= number of participants

A14.3: Screening tests for cervical and thoracic spine pain

There were no differences observed between the group that developed cervical or thoracic spine pain versus the group that did not develop cervical or thoracic spine pain (Table A14.7-Table A14.9). There were no differences between the cervical/thoracic spine injured group and the uninjured group in terms of training and performance (Table A14.10) or baseline anthropometrics, medical history or injury history (Table A14.11-Table A14.12)

Table A14.7: Page 1 of 4: Comparison between group with neck/ thorax pain versus no neck/ thorax pain for categorical tests

Test	Score	No neck or thoracic pain group n	No neck or thoracic pain n per category (%)	Neck or thoracic pain group n	Neck or thoracic pain n per category (%)	Comparison between no neck/thorax pain and neck/thorax pain groups. P value	Comparison between no neck/thorax pain and neck/thorax pain groups. Effect size
Spinal extension clearing test score	Pain	36	3 (8.33%)	5	0 (0%)	1	0.1
	No pain		33 (91.67%)		5 (100%)		
Spinal flexion clearing test	Pain	36	1 (2.78%)	5	0 (0%)	1	0.06
	No pain		35 (97.22%)		5 (100%)		
Pelvic tilt test posture at set up posture score	Neutral	36	33 (91.67%)	5	4 (80%)	0.22	0.27
	Excessive anterior		1 (2.78%)		1 (20%)		
	Excessive posterior		2 (5.56%)		0 (0%)		
Pelvic tilt at set up position test amount of motion score	Normal motion	35	22 (62.86%)	5	4 (80%)	0.82	0.15
	Limited posteriorly		4 (11.43%)		0 (0%)		
	Limited anteriorly		1 (2.86%)		0 (0%)		
	Both limited		8 (22.86%)		1 (20%)		
Pelvic tilt at set up position test quality of movement score	Smooth movement	36	8 (22.22%)	5	1 (20%)	1	0.02
	Shaking movement		28 (77.78%)		4 (80%)		
Pelvic rotation test stability score	Good stability	36	31 (86.11%)	5	5 (100%)	0.85	0.14
	Limited stability left side		3 (8.33%)		0 (0%)		
	Limited stability right side		1 (2.78%)		0 (0%)		
	Both sides stability limited		1 (2.78%)		0 (0%)		
Pelvic rotation test mobility score	Full mobility	36	24 (66.67%)	5	4 (80%)	0.9	0.12
	Limited mobility to the left side		1 (2.78%)		0 (0%)		
	Limited mobility to the right side		2 (5.56%)		0 (0%)		
	Both sides showed limited mobility		9 (25%)		1 (20%)		
Pelvic rotation test quality of movement score	Good rotatory movement	36	23 (63.89%)	5	4 (80%)	0.65	0.11
	Lateral movement more than rotatory movement		13 (36.11%)		1 (20%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared Test and Fishers Exact Test.

Table A14.7: Continued Page 2 of 4: Comparison between group with neck/ thorax pain versus no neck/ thorax pain for categorical tests

Test	Score	No neck or thoracic pain group n	No neck or thoracic pain n per category (%)	Neck or thoracic pain group n	Neck or thoracic pain n per category (%)	Comparison between no neck/thorax pain and neck/thorax pain groups. P value	Comparison between no neck/thorax pain and neck/thorax pain groups. Effect size
Torso rotation test stability score	Good stability	36	35 (97.22%)	5	5 (100%)	1	0.06
	Limited to the left side		1 (2.78%)		0 (0%)		
Torso rotation test mobility score	Full mobility	36	31 (86.11%)	5	5 (100%)	0.85	0.14
	Limited mobility to the left side		1 (2.78%)		0 (0%)		
	Limited mobility to the right side		1 (2.78%)		0 (0%)		
	Both sides showed limited mobility		3 (8.33%)		0 (0%)		
Torso rotation test quality of movement	Good rotatory movement	35	32 (91.43%)	5	5 (100%)	1	0.11
	More lateral movement		3 (8.57%)		0 (0%)		
Toe touch test score	Full range of motion	35	22 (62.86%)	5	3 (60%)	0.75	0.18
	Decreased toe touch on the left		4 (11.43%)		0 (0%)		
	Decreased toe touch on the right		1 (2.86%)		0 (0%)		
	Decreased toe touch bilaterally		8 (22.86%)		2 (40%)		
90 90 shoulder non-dominant score	Standing <spine	35	5 (14.29%)	5	0 (0%)	0.28	0.25
	standing=spine		15 (42.86%)		4 (80%)		
	Standing>spine		15 (42.86%)		1 (20%)		
90 90 shoulder dominant score	Standing <spine	36	5 (13.89%)	5	0 (0%)	0.41	0.21
	standing=spine		18 (50%)		4 (80%)		
	Standing>spine		13 (36.11%)		1 (20%)		
90 90 shoulder golf: posture dominant score	Standing <spine	36	5 (13.89%)	5	0 (0%)	0.26	0.26
	standing=spine		15 (41.67%)		4 (80%)		
	Standing>spine		16 (44.44%)		1 (20%)		
90 90 shoulder: golf posture non-dominant score	Standing <spine	36	7 (19.44%)	5	0 (0%)	0.44	0.2
	standing=spine		19 (52.78%)		4 (80%)		
	Standing>spine		10 (27.78%)		1 (20%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared Test and Fishers Exact Test.

Table A14.7: Continued Page 3 of 4: Comparison between group with neck/ thorax pain versus no neck/ thorax pain for categorical tests

Test	Score	No neck or thoracic pain group n	No neck or thoracic pain n per category (%)	Neck or thoracic pain group n	Neck or thoracic pain n per category (%)	Comparison between no neck/thorax pain and neck/thorax pain groups. P value	Comparison between no neck/thorax pain and neck/thorax pain groups. Effect size
Single leg bridge non-dominant score	Glutes normal	36	28 (77.78%)	5	4 (80%)	0.93	0.06
	Glutes weak		7 (19.44%)		1 (20%)		
	Glutes inhibited		1 (2.78%)		0 (0%)		
Single leg bridge dominant score	Glutes normal	36	30 (83.33%)	5	5 (100%)	0.61	0.15
	Glutes weak		5 (13.89%)		0 (0%)		
	Glutes inhibited		1 (2.78%)		0 (0%)		
Lower quarter rotation non-dominant: internal rot score	>60 degrees	36	15 (41.67%)	5	4 (80%)	0.16	0.3
	=60 degrees		16 (44.44%)		0 (0%)		
	<60 degrees		5 (13.89%)		1 (20%)		
Lower quarter rotation non-dominant: external rot score	>60 degrees	36	29 (80.56%)	5	5 (100%)	0.56	0.17
	=60 degrees		4 (11.11%)		0 (0%)		
	<60 degrees		3 (8.33%)		0 (0%)		
Lower quarter rotation dominant: internal rot score	>60 degrees	36	18 (50%)	5	4 (80%)	0.41	0.21
	=60 degrees		13 (36.11%)		1 (20%)		
	<60 degrees		5 (13.89%)		0 (0%)		
Lower quarter rotation dominant: external rot score	>60 degrees	36	25 (69.44%)	4	4 (100%)	0.43	0.21
	=60 degrees		9 (25%)		0 (0%)		
	<60 degrees		2 (5.56%)		0 (0%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Chi Squared Test and Fishers exact test.

Table A14.7: Continued Page 4 of 4: Comparison between group with neck/ thorax pain versus no neck/ thorax pain for categorical tests

Test	Score	No neck or thoracic pain group n	No neck or thoracic pain n per category (%)	Neck or thoracic pain group n	Neck or thoracic pain n per category (%)	Comparison between no neck/thorax pain and neck/thorax pain groups. p value	Comparison between no neck/thorax pain and neck/thorax pain groups. Effect size
seated trunk rot non-dominant score	>45 degrees	36	33 (91.67%)	5	5 (100%)	0.8	0.1
	=45 degrees		1 (2.78%)		0 (0%)		
	<45 degrees		2 (5.56%)		0 (0%)		
Seated trunk rot dominant score	>45 degrees	35	32 (91.43%)	5	5 (100%)	0.79	0.11
	=45 degrees		1 (2.86%)		0 (0%)		
	<45 degrees		2 (5.71%)		0 (0%)		
Seated trunk rot non-dominant: club behind back score	>45 degrees	36	33 (91.67%)	5	5 (100%)	0.8	0.1
	=45 degrees		1 (2.78%)		0 (0%)		
	<45 degrees		2 (5.56%)		0 (0%)		
Seated trunk rot dominant: club behind back score	>45 degrees	36	34 (94.44%)	5	5 (100%)	0.86	0.08
	=45 degrees		1 (2.78%)		0 (0%)		
	<45 degrees		1 (2.78%)		0 (0%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared Test and Fishers Exact Test.

Table A14.8: Table comparing ranking screening test results for: those with neck/thorax pain compared to those without neck/thorax limb pain

Variable	No neck or thoracic pain percentage per category n=36			Pain in neck or thorax percentage per category n=5			Comparison between no back pain and back pain groups. P value	Comparison between no back pain and back pain groups. Effect size
	Median (IQR)	Lower 95% CI	Upper 95% CI	Median (IQR)	Lower 95% CI	Upper 95% CI		
Single leg squat non-dominant score	2 (1)	1.58	1.92	1 (1)	0.72	2.08	0.25	0.33
Single leg squat dominant score	2 (1)	1.55	1.90	2 (2)	0.76	2.84	0.92	0.49
Deep overhead squat score	2 (1)	2.20	2.53	2 (0.5)	1.64	2.76	0.57	0.42
Hurdle step non-dominant score	2 (1)	2.17	2.5	2 (0)	2	2	0.25	0.33
Hurdle step dominant score	2 (1)	2.2	2.53	2 (0)	2	2	0.2	0.32
In line lunge non-dominant score	3 (1)	2.39	2.73	2 (0.5)	1.64	2.76	0.22	0.32
In line lunge dominant score	3 (1)	2.39	2.73	2 (0.5)	1.64	2.76	0.22	0.32
Trunk stability push up score	3 (0)	2.74	2.98	3 (0)	3	3	0.63	0.43
Rotational stability test non-dominant score	2 (1)	2.05	2.45	3 (1)	1.92	3.28	0.3	0.35
Rotational stability test dominant score	2 (1)	2.11	2.51	3 (1)	1.92	3.28	0.38	0.36
Plank score	5 (7)	3.54	5.55	4 (5.5)	0.14	7.46	0.59	0.38

Table notes: n= number of participants. Data presented as medians and interquartile range (IQR). p value calculated using Mann Whitney U test.

Table A14.9: Table comparing continuous screening test results for: those with neck or thorax pain compared to those without neck or thorax pain

Test	No neck or thorax pain				Has neck or thorax pain				Comparison between no lower quad injury and lower quad injury groups p value	Comparison between no back pain and back pain groups effect size
	n	Mean (SD) or median (IQR)	95% CI lower	95% CI Upper	n	Mean (SD) or median (IQR)	95% CI Lower	95% CI Upper		
Straight leg raise non-dominant (range of motion)	35	70 (25)	65.11	75.92	5	70 (12.5)	60.90	85.10	1.00	0.5
Straight leg raise dominant (range of motion) ^{n}	35	74 (20)	65.38	74.96	5	72 (25)	56.27	88.53	0.74	0.16
Sit ups (no.) ^{n}	36	37.25 (10.04)	33.85	40.65	5	38.40 (10.26)	25.66	51.14	0.81	0.11
Push ups (no.) ^{n}	36	36.28 (13.21)	31.81	40.75	5	41.20 (9.60)	29.28	53.12	0.43	0.38
Oblique sit up to non-dominant shoulder (no.) ^{n}	35	30.17 (8.47)	27.26	33.08	5	30.80 (9.63)	18.85	42.75	0.88	0.07
Oblique sit up to dominant shoulder (no.) ^{n}	35	28.06 (9.48)	24.80	31.31	5	30.80 (5.12)	24.44	37.16	0.53	0.3

Table notes: ^{n} indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations. Data not normally distributed presented as medians and interquartile range (IQR). p values calculated using independent T tests (normally distributed data) and Mann Whitney U tests (data not normally distributed). n=number of participants

Table A14.10: Comparison of baseline performance and training variables for the neck/thorax pain group versus the no neck/thorax pain group

Variable	No neck /thorax pain				Neck/thorax pain				Comparison between neck/thorax groups p value	Comparison between neck/thorax groups effect size
	n	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper	n	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper		
Order of merit (rank)	24	21.5 (29.75)	17.87	30.96	4	27 (32.75)	1.23	55.27	0.65	0.42
Handicap (no)	36	1.5 (3.75)	1.4	3.33	5	3 (2)	1.18	4.02	0.4	0.38
Carry distance (m)^{n}	35	217.51 (21.78)	210.03	224.99	5	204.91 (17.62)	183.03	226.79	0.23	0.59
Roll (m)	35	12.05 (16.06)	12.71	21.66	5	18.5 (34.36)	1.78	52.06	0.16	0.3
Total distance (m)^{n}	35	234.65 (19.38)	228	241.31	5	231.76 (14.01)	214.37	249.16	0.75	0.15
Ball speed (km/h)	35	239.81 (19.27)	221.51	242.07	5	234.3 (17.91)	219.36	247.53	0.33	0.36
Club head speed (km/h)	35	177.10 (16.16)	164.45	179.05	5	180.8 (15.99)	165.75	190.34	0.06	0.41
Putting practice per week (hours per week)	36	3.62 (3.6)	2.44	8.24	5	3.42 (9.17)	-1.86	12.41	0.96	0.49
Short game practice per week (hours per week)	36	3.65 (4.5)	2.58	8.74	5	2.39 (7.3)	-1.33	9.99	0.74	0.45
Range practice per week(hours per week)	36	5.85 (7.52)	4.91	11.37	5	3.28 (5.5)	-0.03	8.48	0.3	0.35
Flexibility training per week (hours per week)	36	1.14 (1.71)	0.65	3.63	5	0.77 (2.15)	-0.05	2.75	0.98	0.49
Postural training per week (hours per week)	36	0.27 (1.26)	0.09	3.06	5	0.26 (0.6)	-0.02	0.77	0.83	0.47
Weight training per week (hours per week)	36	2 (3.14)	1.38	4.8	5	1.5 (3.01)	0.2	4.14	0.92	0.48
Cardio per week (hours per week)	36	1.59 (1.75)	1.26	3.48	5	0.85 (1.58)	-0.14	2.15	0.17	0.31
Rounds of golf played per week	36	2.94 (1.77)	-6.75	30.51	5	3.35 (2.88)	0.91	4.83	0.9	0.48

Table notes: : ^{n} indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations (SD). Data not normally distributed presented as medians and interquartile range (IQR). p values calculated using independent T tests (normally distributed data) and Mann Whitney U tests (data not normally distributed). n=number of participants CI were displayed as calculated, but please be advised that it is not possible to obtain a negative value for the confidence interval for these values, except for handicap. The lowest possible value is zero

Table A14.11: Comparison of baseline anthropometric data neck/thoracic injury versus no neck/thoracic limb injury

Variable	Neck/thoracic injury				Neck thoracic injury				Comparison between lower limb groups	Comparison between lower limb groups
	n	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper	n	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper	P value	Effect size
Age (years)	36	19 (3)	18.78	20.55	5	21 (2.5)	18.96	22.64	0.16	0.3
Height (m)^n	36	1.79 (0.07)	1.76	1.81	5	1.83 (0.09)	1.7	1.93	0.42	0.39
Body mass (kg)^n	36	78 (12.2)	73.87	82.13	5	78.16 (19.63)	53.78	102.54	0.98	0.01
BMI (kg/m^2)^n	36	24.47 (3.7)	23.22	25.72	5	23.47 (3.88)	18.65	28.29	0.58	0.27

Table notes: ^{n} indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations. Data not normally distributed presented as medians and interquartile range (IQR). p values calculated using independent T tests (normally distributed data) and Mann Whitney U tests (data not normally distributed). n=number of participants

Table A14.12: Comparison of baseline medical history and history of injury: Neck/thoracic pain group versus the no neck/thoracic pain group

Variable	No neck/ thorax pain group n	No neck/ thorax pain group n(%) with condition or health risk factor	Neck/ thorax pain group n	neck/ thorax pain group n(%) with condition or health risk factor	p value for between group difference at baseline	Between group odds ratio	Risk ratio in lower neck/ thorax group	Between group comparison effect size
Smoked at least 100 cigarettes in his lifetime	31	13 (41.94%)	5	3 (60%)	0.64	0.48	1.88	0.13
History of vascular problems	30	1 (3.33%)	5	1 (20%)	0.27	0.14	4.13	0.25
History of respiratory problems	29	7 (24.14%)	4	1 (25%)	1	0.95	1.04	0.01
History of cancer	30	2 (6.67%)	5	0 (0%)	1	NA	NA	0.1
History of musculoskeletal conditions	30	3 (10%)	5	1 (20%)	0.48	0.44	1.94	0.11
History of neurological conditions	30	4 (13.33%))	5	1 (20%)	0.56	0.62	1.5	0.07
History of gastrointestinal conditions	30	5 (16.67%)	5	0 (0%)	1	NA	NA	0.17
History of injury in past six months	29	15 (51.72%)	5	4 (80%)	0.35	0.27	3.16	0.2

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared test and Fishers exact test. n= number of participants. As no participant in either group had neck or thoracic pain in the six months prior to taking part in the study it was not possible to add this variable to the analysis.

A14.4: Screening tests for upper limb pain

There was a difference between the group that developed upper limb pain and the group that did not develop upper limb pain in terms of their pelvic posture at address ($p=0.02$; effect size= 0.44) (Table A14.13). The upper quadrant injured participants (22% in the injured group and 0% in the uninjured group) tended to have a higher percentage of participants with an excessive posterior tilt at set up. There were no differences observed in any other screening tests for upper limb pain (Table A14.13-15).

The group that developed upper limb pain had a slower club head speed (median (IQR)= 173.48 (12.11) km/hr) than the group that did not develop upper limb pain (median (IQR) = 178.67 (11.16) ($p=0.02$; effect size= 0.25). There were no other performance or training differences between the group that developed upper limb pain and the group that did not develop upper limb pain in terms of performance and training (Table A14.16).

There were no differences between upper limb groups in terms of baseline anthropometrics, medical history or injury history (Table A14.17 and Table A14.18).

Table A14.13: Page 1 of 4: Comparison between group with upper limb pain versus no upper limb pain categorical tests

Test	Score	No upper limb pain group n	No upper limb pain n per category (%)	Upper limb pain group n	Upper limb pain n per category (%)	Comparison between no upper limb pain and upper limb pain group p value	Comparison between no upper limb pain and upper limb pain group effect size
Spinal extension clearing test score	Pain	32	3 (9.38%)	9	0 (0%)	1	0.15
	No pain		29 (90.63%)		9 (100%)		
Spinal flexion clearing test	Pain	32	0 (0%)	9	1 (11.11%)	0.22	0.3
	No pain		32 (100%)		8 (88.89%)		
Pelvic tilt test posture at set up posture score	Neutral	32	30 (93.75%)	9	7 (77.78%)	0.02	0.44
	Excessive anterior		2 (6.25%)		0 (0%)		
	Excessive posterior		0 (0%)		2 (22.22%)		
Pelvic tilt at set up position test amount of motion score	Normal motion	32	21 (65.63%)	8	5 (62.5%)	0.4	0.27
	Limited posteriorly		2 (6.25%)		2 (25%)		
	Limited anteriorly		1 (3.13%)		0 (0%)		
	Both limited		8 (25%)		1 (12.5%)		
Pelvic tilt at set up position test quality of movement score	Smooth movement	32	7 (21.88%)	9	2 (22.22%)	1	0
	Shaking movement		25 (78.13%)		7 (77.78%)		
Pelvic rotation test stability score	Good stability	32	29 (90.63%)	9	7 (77.78%)	0.24	0.32
	Limited stability left side		1 (3.13%)		2 (22.22%)		
	Limited stability right side		1 (3.13%)		0 (0%)		
	Both sides stability limited		1 (3.13%)		0 (0%)		
Pelvic rotation test mobility score	Full mobility	32	22 (68.75%)	9	6 (66.67%)	0.75	0.17
	Limited mobility to the left side		1 (3.13%)		0 (0%)		
	Limited mobility to the right side		1 (3.13%)		1 (11.11%)		
	Both sides showed limited mobility		8 (25%)		2 (22.22%)		
Pelvic rotation test quality of movement score	Good rotatory movement	32	21 (65.63%)	8	6 (66.67%)	1	0.01
	Lateral movement more than rotatory movement		11 (34.38%)		3 (33.33%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared test and Fishers exact test. n= number of participants

Table A14.13: Continued Page 2 of 4: Comparison between group with upper limb pain versus no upper limb pain categorical tests

Test	Score	No upper limb pain group n	No upper limb pain n per category (%)	Upper limb pain group n	Upper limb pain n per category (%)	Comparison between no upper limb pain and upper limb pain group p value	Comparison between no upper limb pain and upper limb pain group effect size
Torso rotation test stability score	Good stability	32	32 (100%)	9	8 (88.89%)	0.22	0.3
	Limited to the left side		0 (0%)		1 (11.11%)		
Torso rotation test mobility score	Full mobility	32	28 (87.5%)	9	8 (88.89%)	0.85	0.14
	Limited mobility to the left side		1 (3.13%)		0 (0%)		
	Limited mobility to the right side		1 (3.13%)		0 (0%)		
	Both sides showed limited mobility		2 (6.25%)		1 (11.11%)		
Torso rotation test quality of movement	Good rotatory movement	31	28 (90.32%)	9	9 (100%)	1	0.15
	More lateral movement		3 (9.68%)		0 (0%)		
Toe touch test score	Full range of motion	31	18 (58.06%)	9	7 (77.78%)	0.65	0.2
	Decreased toe touch on the left		3 (9.68%)		1 (11.11%)		
	Decreased toe touch on the right		1 (3.23%)		0 (0%)		
	Decreased toe touch bilaterally		9 (29.03%)		1 (11.11%)		
90 90 shoulder non-dominant score	Standing <spine	32	4 (12.5%)	8	1 (12.5%)	0.31	0.24
	standing=spine		17 (53.13%)		2 (25%)		
	Standing>spine		11 (34.38%)		5 (62.5%)		
90 90 shoulder dominant score	Standing <spine	32	3 (9.38%)	9	2 (22.22%)	0.49	0.19
	standing=spine		17 (46.88%)		5 (44.44%)		
	Standing>spine		12 (43.75%)		2 (33.33%)		
90 90 shoulder golf: posture dominant score	Standing <spine	32	3 (9.38%)	9	2 (22.22%)	0.56	0.17
	standing=spine		15 (46.88%)		4 (44.44%)		
	Standing>spine		14 (43.75%)		3 (33.33%)		
90 90 shoulder: golf posture non-dominant score	Standing <spine	32	5 (15.63%)	9	2 (22.22%)	0.48	0.19
	standing=spine		17 (53.13%)		6 (66.67%)		
	Standing>spine		10 (31.25%)		1 (11.11%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared test and Fishers exact test. n= number of participants

Table A14.13: Continued Page 3 of 4: Comparison between group with upper limb pain versus no upper limb pain categorical tests

Test	Score	No upper limb pain group n	No upper limb pain n per category (%)	Upper limb pain group n	Upper limb pain n per category (%)	Comparison between no upper limb pain and upper limb pain group p value	Comparison between no upper limb pain and upper limb pain group effect size
Single leg bridge non-dominant score	Glutes normal	32	26 (81.25%)	9	6 (66.67%)	0.45	0.2
	Glutes weak		5 (15.63%)		3 (33.33%)		
	Glutes inhibited		1 (3.13%)		0 (0%)		
Single leg bridge dominant score	Glutes normal	32	28 (87.5%)	9	7 (77.78%)	0.52	0.18
	Glutes weak		3 (9.38%)		2 (22.22%)		
	Glutes inhibited		1 (3.13%)		0 (0%)		
Lower quarter rotation non-dominant: internal rot score	>60 degrees	32	15 (46.88%)	9	4 (44.44%)	0.91	0.07
	=60 degrees		12 (37.5%)		4 (44.44%)		
	<60 degrees		5 (15.63%)		1 (11.11%)		
Lower quarter rotation non-dominant: external rot score	>60 degrees	32	26 (81.25%)	9	8 (88.89%)	0.5	0.18
	=60 degrees		4 (12.5%)		0 (0%)		
	<60 degrees		2 (6.25%)		1 (11.11%)		
Lower quarter rotation dominant: internal rot score	>60 degrees	32	18 (56.25%)	9	4 (44.44%)	0.76	0.12
	=60 degrees		10 (31.25%)		4 (44.44%)		
	<60 degrees		4 (12.5%)		1 (11.11%)		
Lower quarter rotation dominant: external rot score	>60 degrees	31	21 (67.74%)	9	8 (88.89%)	0.14	0.31
	=60 degrees		9 (29.03%)		0 (0%)		
	<60 degrees		1 (3.23%)		1 (11.11%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared test and Fishers exact test. n= number of participants

Table A14.13: Continued Page 4 of 4: Comparison between group with upper limb pain versus no upper limb pain categorical tests

Test	Score	No upper limb pain group n	No upper limb pain n per category (%)	Upper limb pain group n	Upper limb pain n per category (%)	Comparison between no upper limb pain and upper limb pain group p value	Comparison between no upper limb pain and upper limb pain group effect size
seated trunk rot non-dominant score	>45 degrees	32	30 (93.75%)	9	8 (88.89%)	0.54	0.17
	=45 degrees		1 (3.13%)		0 (0%)		
	<45 degrees		1 (3.13%)		1 (11.11%)		
Seated trunk rot dominant score	>45 degrees	31	29 (93.55%)	9	8 (88.89%)	0.56	0.17
	=45 degrees		1 (3.23%)		0 (0%)		
	<45 degrees		1 (3.23%)		1 (11.11%)		
Seated trunk rot non-dominant: club behind back score	>45 degrees	32	30 (93.75%)	9	8 (88.89%)	0.54	0.17
	=45 degrees		1 (3.13%)		0 (0%)		
	<45 degrees		1 (3.13%)		1 (11.11%)		
Seated trunk rot dominant: club behind back score	>45 degrees	32	30 (93.75%)	9	9 (100%)	0.74	0.12
	=45 degrees		1 (3.13%)		0 (0%)		
	<45 degrees		1 (3.13%)		0 (0%)		

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared test and Fishers exact test. n= number of participants

Table A14.14: Table comparing ranking screening test results for those with upper limb pain compared to those without upper limb pain.

Variable	No upper limb pain n=32			Upper limb pain n=9			Comparison between no upper limb pain and upper limb pain group p value	Comparison between no upper limb pain and upper limb pain group effect size
	Median (IQR)	Lower CI	Upper CI	Median (IQR)	Lower CI	Upper CI		
Single leg squat non-dominant score	2 (1)	1.57	1.93	2 (1)	1.15	1.96	0.43	0.41
Single leg squat dominant score	2 (1)	1.58	1.98	2 (1)	1.15	1.96	0.38	0.4
Deep overhead squat score	2 (1)	2.20	2.55	2 (0.5)	1.88	2.56	0.51	0.42
Hurdle step non-dominant score	2 (1)	2.12	2.45	2 (1)	1.95	2.72	0.82	0.47
Hurdle step dominant score	2 (1)	2.14	2.48	2 (1)	1.95	2.72	0.94	0.49
In line lunge non-dominant score	3 (1)	2.38	2.74	2 (1)	1.95	2.72	0.31	0.39
In line lunge dominant score	3 (1)	2.38	2.74	2 (1)	1.95	2.72	0.31	0.39
Trunk stability push up score	3 (0)	2.75	3	3 (0)	2.63	3.15	0.96	0.49
Rotational stability test non-dominant score	2 (1)	2.	2.44	3 (1)	2.15	2.96	0.21	0.36
Rotational stability test dominant score	2 (1)	2.07	2.51	3 (1)	2.15	2.96	0.31	0.37
Plank score	5 (6.25)	3.29	5.31	6.5 (7)	2.17	7.83	0.54	0.36

Table notes: n= number of participants. Data presented as medians and interquartile range (IQR). p value calculated using Mann Whitney U test.

Table A14.15: Table comparing continuous screening test results for: those with upper limb pain compared to those without upper limb pain

Test	No upper limb pain				Upper limb pain				Comparison between no upper limb pain and upper limb pain groups p value	Comparison between no upper limb pain and upper limb pain group effect size
	n	Mean (SD) or median (IQR)	95% CI lower	95% CI Upper	n	Mean (SD) or median (IQR)	95% CI lower	95% CI upper		
Straight leg raise non-dominant (range of motion)	31	70.00 (17)	65.58	76.42	9	70.00 (25.5)	57.25	83.20	0.70	0.46
Straight leg raise dominant (range of motion)[n]	31	72.32 (11.77)	68.00	76.64	9	64.00 (18.24)	49.98	78.02	0.22	0.41
Sit ups (no.)[n]	32	37.68 (10.74)	33.99	41.63	9	35.89 (7.51)	30.12	41.66	0.61	0.19
Push ups (no.)[n]	32	37.63 (13.81)	32.65	42.6	9	34.22 (8.61)	27.60	40.84	0.49	0.26
Oblique sit up to non-dominant shoulder (no.)[n]	31	29.90 (9.02)	26.59	33.21	9	31.44 (6.65)	26.33	36.56	0.64	0.18
Oblique sit up to dominant shoulder (no.)[n]	31	27.77 (9.47)	24.30	31.25	9	30.56 (7.50)	24.79	36.32	0.42	0.31

Table notes: [n] indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations. Data not normally distributed presented as medians and interquartile range (IQR). p values calculated using independent T tests (normally distributed data) and Mann Whitney U tests (data not normally distributed). n=number of participants

Table A14.16: Comparison of baseline performance and training variables for the upper limb pain group versus the no upper limb pain group

Variable	No upper limb pain n	No upper limb pain Mean (SD) or median (IQR)	No upper limb pain 95% CI lower	No upper limb pain 95% CI upper	Upper limb pain n	Upper limb pain Mean (SD) or median (IQR)	Upper limb pain 95% CI lower	Upper limb pain 95% CI upper	Comparison between upper limb pain groups P value	Comparison between upper limb pain groups Effect size
Order of merit (rank)	20	18.5 (27)	15.43	29.37	8	38 (25.75)	18.07	44.68	0.24	0.35
Handicap (no)	32	1.5 (3.5)	1.28	3.34	9	2 (4)	1.04	4.3	0.47	0.42
Carry dist. (m) [n]	31	217.58 (21.89)	209.56	225.61	9	210.25 (20.36)	194.6	225.9	0.38	0.34
Roll (m)	31	12.6 (15.02)	12.58	21.46	9	18.1 (25.19)	7.88	38.42	0.57	0.43
Total dist. (m) [n]	31	234.57 (18.73)	227.69	241.44	9	233.35 (19.53)	218.34	248.36	0.87	0.06
Ball (m)	31	241.3 (14.9)	224.3	243.89	9	233.9 (17.38)	198.98	250.53	0.16	0.34
Club km/h	31	178.67 (11.16)	167.56	181.66	9	173.48 (12.11)	147.7	183.09	0.02	0.25
Putting practice per week (hours per week)	32	3.25 (4.13)	2.39	9.05	9	3.79 (3.88)	2.25	5.69	0.75	0.46
Short game practice per week (hours per week)	32	3.26 (4.68)	2.17	9.05	9	4.58 (4.81)	1.55	8.66	0.65	0.45
Range practice per week(hours per week)	32	5.81 (7.77)	4.58	11.65	9	3.38 (6.75)	1.4	10.71	0.51	0.43
Flexibility training per week (hours per week)	32	1.14 (2.05)	0.65	4.01	9	0.75 (1.12)	0.47	1.58	0.68	0.45
Postural training per week (hours per week)	32	0.27 (0.95)	-0.23	3.03	9	0.27 (1.84)	-0.38	3.39	0.24	0.37
Weight training per week (hours per week)	32	2 (3.06)	1.52	5.33	9	1.69 (2.32)	0.49	2.25	0.24	0.37
Cardio per week (hours per week)	32	1.54 (1.68)	1.09	3.59	9	1.48 (1.79)	0.79	2.64	0.99	0.5
Rounds of golf played per week	32	3.02 (2)	-7.84	34.25	9	2.15 (2.1)	1.16	3.13	0.12	0.33

Table notes: : [n] indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations (SD). Data not normally distributed presented as medians and interquartile range (IQR). p values calculated using independent T tests (normally distributed data) and Mann Whitney U tests (data not normally distributed). n=number of participants. CI were displayed as calculated, but please be advised that it is not possible to obtain a negative value for the confidence interval for these values, except for handicap. The lowest possible value is zero

Table A14.17: Comparison of baseline anthropometric data upper limb injury versus no upper limb injury

Variable	No upper limb injury				Upper limb injury				Comparison between upper limb group p value	Comparison between upper limb group effect size
	n	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper	n	Mean (SD) or Median (IQR)	95% CI lower	95% CI upper		
Age (years)	32	19.72 (3)	18.72	20.71	9	21 (2.5)	19.06	21.16	0.30	0.38
Height (m)^n	32	1.78 (0.08)	1.76	1.81	9	1.81 (0.05)	1.77	1.84	0.45	0.29
Body mass (kg)^n	32	78.62 (13.63)	73.7	83.53	9	75.89 (10.83)	67.56	84.22	0.58	0.21
BMI (kg/m^2)^n	32	24.65 (3.84)	23.27	26.04	9	23.26 (3.01)	20.94	25.57	0.32	0.38

Table notes: : ^{n} indicates that data that was normally distributed. All other data was not normally distributed. Normally distributed data presented as means and standard deviations (SD). Data not normally distributed presented as medians and interquartile range (IQR). p values calculated using independent T tests (normally distributed data) and Mann Whitney U tests (data not normally distributed). n=number of participants.

Table A14.18: Comparison of baseline medical history and history of injury: Upper limb injury group versus the no upper limb injury group

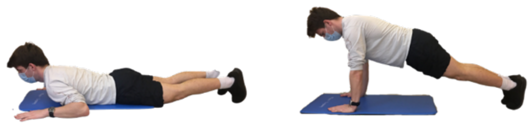
Variable	No upper limb pain group n	No upper limb pain group n(%) with condition or health risk factor	Upper limb pain group n	Upper limb pain group n(%) with condition or health risk factor	p value for between group difference at baseline	Between upper limb group comparison Odds ratio	Risk ratio in upper limb pain group	Between group comparison effect size
Smoked at least 100 cigarettes in his lifetime	27	12 (44.44%)	9	4 (44.44%)	1	1	1	0
History vascular problems	26	1 (3.85%)	9	1 (11.11%)	0.45	0.32	2.06	0.14
History respiratory problems	24	6 (25%)	9	2 (22.22%)	1	1.17	0.89	0.03
History of cancer	26	1 (3.85 %)	9	1 (11.11%)	0.45	0.32	2.06	0.14
History of musculoskeletal conditions	26	2 (7.69%)	9	2 (22.22%)	0.27	0.29	2.21	0.2
History of neurological conditions	26	3 (11.54%)	9	2 (22.22%)	0.59	0.46	1.71	0.13
History of gastrointestinal conditions	26	3 (11.54%)	9	2 (22.22%)	0.59	0.46	1.71	0.13
History of injury in past six months	25	12 (48%)	9	7 (77.78%)	0.24	0.26	2.76	0.26
History of upper limb injury in past six months	25	4 (16%)	9	3 (33.33%)	0.35	0.38	1.93	0.19

Table notes: Data presented as number (n) per category and percentages. p value calculated using Pearson Chi Squared test and Fishers exact test. n= number of participants

Appendix 15: Screening tool

Test Image	Test description
Single leg squat test  	The participant was asked to stand on one leg and perform the single leg squat with the non stance leg in neutral as shown in figure (Olivier et al., 2019). Scoring was based on Crossley et al. (2011). In order for a participant to be scored as good: The participant needed to not loose their balance The movement needed to be smooth. The squat needed to be performed to at least 60 degrees of knee flexion. There was no trunk deviation during the squat. There was no pelvic lateral shift. No hip adduction during the squat. No apparent knee valgus during the squat. The knee needed to remain over the centre of the foot.
Deep over head squat  	The participant was asked to stand with their feet shoulder width apart while holding a dowel overhead. Then the participant was asked to squat while maintaining an upright torso, keeping the heels on the floor and the dowel above their head. Then the participant stood upright again. The scoring was based on Cook et al. (2014a).

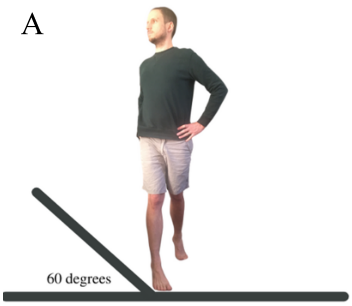
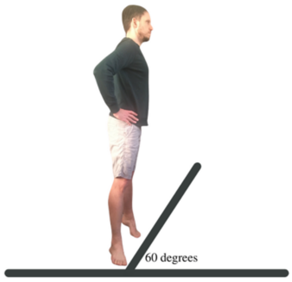
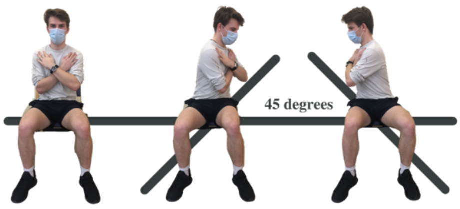
<table>
<tr><td>Test Image</td><td>Test description</td></tr>
<tr><td>Hurdle step test
 </td><td>The participant was asked to stand in front of the hurdle. The hurdle was adjusted to the height of the participant’s tibial tuberosity. The participant was asked to hold the dowel with both hands and place it behind his neck. The participant was then asked to step over the hurdle while maintaining upright posture and maintaining alignment between their foot, knee and hip. The participant placed their foot on the other side of the hurdle, without accepting weight transfer onto that foot. The moving leg was then returned to its starting position. The scoring was based on Cook et al. (2014a).</td></tr>
<tr><td>In line lunge test
 </td><td>A measurement was made from the floor to the participant’s tibial tuberosity. The participant was asked to stand on a line with one foot in front and the other foot behind. The distance from the toes of the back foot to the heel of the front foot was the same as the distance from the floor to the participant’s tibial tuberosity. The participant stood with their toes facing forward while he grasped the dowel behind his back (perpendicular to the floor). The participant was then asked to lunge. The scoring was based on Cook et al. (2014a).</td></tr>
</table>

Test Image	Test description
Straight leg raise 	The participant was asked to lie in supine. A goniometer was then placed with the stationary arm parallel to the edge of the table and the movable arm was placed parallel to the lateral shaft of the thigh. The axis was placed over the superior half of the greater trochanter (Hsieh et al., 1983). The participant was then asked to actively lift their leg while keeping their knee straight. A goniometer measurement was then taken by the physiotherapist.
Trunk stability push up test 	The participant was asked to assume a prone position with their feet together, knees fully extended and their hands placed at the level of their forehead. The participant was then asked to perform a push up in this position. The participant's body should ideally move as a single unit with no lag. The scoring was based on Cook et al. (2014b).
Spinal extension clearing test 	The participant was asked to assume a prone position with his hands at forehead height and then he was asked to perform a push up while keeping his hips on the floor. The scoring was based on Cook et al. (2014b)

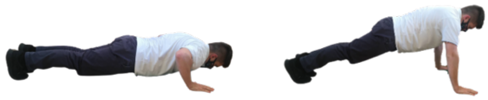
Test Image	Test description
Rotatory stability test 	The participant was asked to assume a four point kneeling position. He was then asked to extend his left hand and left leg. He was then asked to bring his left elbow towards his left knee while maintaining his balance on his right hand and right knee. The same movement pattern is performed on right side. The scoring was based on Cook et al. (2014b).
Spinal flexion clearing test 	The participant was asked to assume a four point kneeling position. He was then asked to rock backward until his buttocks touched his heels and his chest touched his thighs. The scoring was based on Cook et al. (2014b).
Pelvic tilt test 	The participant was asked to assume a golf set up posture. The participants posture was assessed in this golf set up position. He was then asked to anterior tilt his pelvis (arch their lower back) and posterior tilt his pelvis (flatten their back). Both the quality of the movement and the range of motion was assessed. The movement was demostrated to the particpant if he did not understand how to perform the test. The scoring was based on Titleist Performance Institute (2021f).

Test Image	Test description
Pelvic rotation test 	The participant was asked to stand in golf posture with his arms crossed. He was then asked to keep his torso as still as possible while rotating his pelvis left and right (as he would during the golf swing). The scoring was based on Titleist Performance Institute (2021e). Stability during this movement, range of motion and quality of movement was assessed.
Torso rotation test 	The participant was asked to stand in golf posture with his arms crossed. He was then asked to keep his pelvis as still as possible while rotating his torso left and right (as he would during the golf swing). The scoring was based on Titleist Performance Institute (2021j). Stability during this movement, range of motion and quality of movement was assessed.
Toe touch test (Figure 5.14) 	The participant was asked to stand and place his feet together. He was then asked to bend as far forward as possible. The scoring was based on Titleist Performance Institute (2021i).

Test Image	Test description
90 90 shoulder test in standing 	The participant was asked to stand upright and position his shoulder in 90 degrees of abduction and maximum external rotation. The scoring was based on Titleist Performance Institute (2021a). Scoring was based on whether or not the forearm achieved a position parallel to the spine (i.e. perpendicular to the floor).
90 90 shoulder test in golf posture 	The participant was asked to stand in the set up position and then position his shoulder in 90 degrees of abduction and maximum external rotation. The scoring was based on Titleist Performance Institute (2021a). Scoring was based on whether or not the forearm achieved a position parallel to the spine.
Single leg bridge with leg extension test 	The participant was asked to ly supine and assume the crook lying position. The participant was then asked to perform a bridge. Once in this position he was asked to extend one knee while maintaining a good pelvic alignment. The scoring was based on Titleist Performance Institute (2021c). Participants were scored as weak if they were unable to perform the test or they if they found it difficult maintain the position or if they shook while holding this position. Gluteals were considered inhibited if the participant cramped during the exercise.

Test Image	Test description
Lower quarter rotation test  	The participant was asked to stand with their hands on their hips. The participant was then asked to shift all their weight onto the right foot and move their left foot behind the right foot and place it on the floor in a dorsiflexion position. The participant was then asked to rotate as far as he could both clockwise (Figure 5.18A) and anticlockwise (Figure 5.18B) on his right leg. Markers on the floor were used to determine if the participant achieved a 60 degree angle while rotating both clockwise and anticlockwise. The movement was repeated on the left leg. The scoring was based on Titleist Performance Institute (2021d).
Seated trunk rotation test 	The physiotherapist marked out two lines 45 degrees from the horizontal and placed a chair over the line. The participant was asked to sit on the chair and crosses his arms across his chest. He was then asked to rotate his thorax clockwise and anticlockwise. Using markers on the floor the physiotherapist determined if the participant was able to rotate to a 45 degree angle both a clockwise and anti clockwise direction. This test was then repeated with the golf club or dowel placed behind the participants shoulders. The scoring was based on Titleist Performance Institute (2021g).

Test Image	Test description
Seated trunk rotation test with club behind shoulders 	The physiotherapist marked out two lines 45 degrees from the horizontal and placed a chair over the line. The participant was asked to sit on the chair and with the golf club or dowel placed behind his shoulders. He was then asked to rotate his thorax clockwise and anticlockwise. Using markers on the floor the physiotherapist determined if the participant was able to rotate to a 45 degree angle in both a clockwise and anti clockwise direction. The scoring was based on Titleist Performance Institute (2021g).
Plank endurance test 	The participant was asked to assume the plank position, which is a prone bridge supported by the forearms and feet. Then at various times points the participant was asked to lift a leg or an arm or both (see functional screen scoring table). The scoring was based on Tong et al. (2014).
Sit up test  	The participant was asked to perform as many sit ups as he could in one minute. The participant was permitted to stabilise his legs during the sit up if he wished.

Test Image	Test description
Push up test 	The participant was asked to perform as many push ups as he could in one minute.
Oblique sit up test 	The participant was asked to perform as many sit oblique sit ups as he could in one minute. The participant was permitted to stabilise his legs during the sit up if he wished.

Appendix 16: Scoring of functional tests.

Table A16 Continued Page 1 of 3: Table of functional tests scoring and the type of data each that each functional movement test yields.

Test	Type of data	Test scoring	Numerical value assigned to score
Single leg squat	Ordinal (ranking tests)	Good	1
		Fair	2
		Poor	3
Deep overhead squat	Ordinal (ranking tests)	Pain while performing this test	0
		Unable to complete the movement pattern or assume the start position	1
		Able to perform the movement pattern, but requires compensation	2
		Correct movement pattern without compensation	3
Hurdle step	Ordinal (ranking tests)	Pain	0
		Unable to complete the movement pattern or assume the start position	1
		Able to perform the movement pattern, but requires compensation	2
		Correct movement pattern without compensation	3
In line lunge	Ordinal (ranking tests)	Pain while performing this test	0
		Unable to complete the movement pattern or assume the start position	1
		Able to perform the movement pattern, but requires compensation	2
		Correct movement pattern without compensation	3
Straight leg raise	Continuous	Goniometer measurement in degrees	Continuous measurement
Trunk stability push up	Ordinal (ranking tests)	Pain	0
		Unable to complete the movement pattern or assume the start position	1
		Able to perform the movement pattern, but requires compensation	2
		Correct movement pattern without compensation	3
Spinal extension test	Nominal (Categorical tests)	Pain in this position	None
		No pain in this position	None
Rotational stability test	Ordinal (ranking tests)	Pain while performing this test	0
		Unable to complete the movement pattern or assume the start position	1
		Able to perform the movement pattern, but requires compensation	2
		Correct movement pattern without compensation	3
Spinal flexion clearing test	Nominal (Categorical tests)	Pain while performing this test	None
		No pain	None
Pelvic tilt test at address posture at set up score	Nominal (Categorical tests)	Neutral	None
		Excessive anterior	None
		Excessive posterior	None

Table A16 Continued Page 2 of 3: Table of functional tests scoring and the type of data each that each functional movement test yields.

Test	Scoring of test	Test scoring	Numerical value assigned to score
Pelvic tilt test at set up amount of motion score	Nominal (Categorical tests)	Normal motion	None
		Limited posteriorly	None
		Limited anteriorly	None
		Both limited	None
Pelvic tilt test at set up quality of movement score	Nominal (Categorical tests)	Smooth movement	None
		Shaking movement	None
Pelvic rotation test stability score	Nominal (Categorical tests)	Good stability	None
		Limited stability left side	None
		Limited stability right side	None
		Both stability limited	None
Pelvic rotation test mobility score	Nominal (Categorical tests)	Good mobility (full active range of motion)	None
		Limited mobility when moving to the left side	None
		Limited mobility when moving towards the right side	None
		Both sides show limited movement (reduced active range of motion).	None
Pelvic rotation test quality of movement score	Nominal (Categorical tests)	Good rotatory movement	None
		Participant tends to perform a more lateral movement than a rotatory movement.	None
Torso rotation test stability score	Nominal (Categorical tests)	Good stability	None
		Limited stability when moving to the left side	None
		Limited stability right side	None
		Stability limited towards both sides	None
Torso rotation test mobility score	Nominal (Categorical tests)	Good mobility or active range of motion during this test	None
		Limited mobility left side	None
		Limited mobility right side	None
		Both sides mobility limited	None
Torso rotation test quality of movement score	Nominal (Categorical tests)	Good rotatory movement	None
		More lateral movement	None
Toe touch test	Nominal (Categorical tests)	Full range of motion	None
		Decreased toe touch on the left	None
		Decreased toe touch on the right	None
		Decreased toe touch bilaterally	None

Table A16 Continued Page 3 of 3: Table of functional tests scoring and the type of data each that each functional movement test yields.

Test	Scoring of test	Test scoring	Numerical value assigned to score
90 90 shoulder in standing	Nominal (Categorical tests)	Standing<spine	None
		Standing=spine	None
		Standing>spine	None
90 90 shoulder in golf posture	Nominal (Categorical tests)	Standing<spine	None
		Standing=spine	None
		Standing>spine	None
Single leg bridge with leg extension	Nominal (Categorical tests)	Glut normal	None
		Gluts weak	None
		Glut inhibited	None
lower quarter rotation test internal rotation measurement	Nominal (Categorical tests)	>60°	None
		=60°	None
		<60°	None
lower quarter rotation test external rotation measurement	Nominal (Categorical tests)	>60°	None
		=60°	None
		<60°	None
Seated trunk rotation with arms crossed	Nominal (Categorical tests)	>45°	None
		=45°	None
		<45°	None
Seated trunk rotation with club behind back	Nominal (Categorical tests)	>45°	None
		=45°	None
		<45°	None
Plank test	Ordinal (ranking tests)	Hold basic plank for 60 sec	1
		Then lift right arm off the ground for 15 sec	2
		Then return the right arm to the start position and lift the left arm off the ground for 15 sec	3
		Return the left arm to the starting position and then lift right leg off the ground for 15 sec.	4
		Then return the right leg to start position and lift the left leg off the ground for 15 sec	5
		Then lift the left leg and right arm off the ground for 15 sec.	6
		Return the left and right arm to the start position, then lift the right leg and left arm off the ground for 15 sec.	7
		Return to the basic plank position for 30 sec.	8
		Repeat until failure steps 1-8 and record the number of steps achieved.	9
Sit up test	Continuous	Maximum number of sit ups the golfer is able to achieve in 1 minute	Continuous measurement
Push up test	Continuous	Maximum number of push ups the golfer is able to achieve in 1 minute	Continuous measurement
Oblique sit up test	Continuous	Maximum number of oblique sit ups to the golfer is able to achieve in 1 minute	Continuous measurement

Appendix 17: Participant information sheet

PARTICIPANT INFORMATION SHEET

Principal researcher information only: CONFIDENTIAL INFORMATION

Subject information sheet

Date: ___________________

Participant Code	

Personal Information

Name	
Age	
Gender	Male
Date of Birth	
ID	
Telephone number (home)	
Telephone number (work)	
Telephone number (cell)	
Email address	
Golf Handicap	
Amateur or professional golf status	
Full time or social golfer	
Occupation	

Address (Postal)

____

____

____

____

____

Residential Address

Next of kin (If under 18 years please use parent or guardian)	
Relationship of next of kin	
Telephone number next of kin	
Additional contact number of next of kin	
Email of next of kin	

Appendix 18: Demographic, medical history and history of lower back pain questionnaire

Background Information

Study Number

Gender (Please circle)

Male

Female

Age

Current height (without shoes) in cm

Current weight in Kg

Health behaviours

Have you smoked at least 100 cigarettes in your ENTIRE LIFE?

Yes

No

General medical history

Condition	Yes	No
Do you have a history or diagnosis of any of the following vascular conditions such as angina pectoris, coronary artery disease, arrhythmia, congestive cardiac failure, blood pressure problems, clotting abnormalities, TIA or stroke, heart attack etc.?		
Do you have a history or diagnosis of a respiratory or lung condition such as exercise induced asthma, asthma, emphysema, chronic bronchitis / cough etc.?		
Do you have a history or diagnosis of any kind of cancer?		
Do you have a history or diagnosis of a musculoskeletal condition such as osteoporosis, chronic lower back or neck pain, osteoarthritis, rheumatoid arthritis, fibromyalgia etc.?		
Do you have a history or diagnosis of a neurological condition such as anxiety, depression, multiple sclerosis, Parkinson's, epilepsy / seizures, Polio, Alzheimer's etc.?		
Do you have a history or diagnosis of a gastrointestinal condition such as stomach or duodenal ulcer, Irritable Bowel Syndrome, gastric acid reflux, diverticulitis, gall bladder disease or gall stones, liver problems, haemorrhoids etc.?		

INJURY RELATED QUESTIONS

Have you sustained any injuries in the last six months?

□ Yes

□ No

Injury definition: Any pain in the muscle, bone or nerve other than that caused from blisters or minor skin lesions

In which body area did you sustain these injury(ies)?

□ Lower back

□ Hip

□ Knee

□ Ankle/foot

□ Shoulder

□ Elbow

□ Hand

□ Other

In which body area(s) is / are the other injury / injuries?

LOWER BACK PAIN

Although you are currently injury free, have you experienced low back pain in the last month?

□ Yes

□ No

How often have you experienced lower back pain in the last six
months?

□ Monthly

□ Weekly

□ Daily

□ Always

Did you miss GOLF DAYS as a result of the injury / injuries above?

□ Yes

□ No

Number of days ________________________

Did you miss GOLF TOURNAMENTS as a result of the injury / injuries above?

□ Yes

□ No

Number of tournaments ________________________

How did your low back injury / injuries occur?

On average, when you experience lower back, the pain is:

□ Constant (all the time)

□ Intermittent (on and off)

□ Transient (short lived)

On average when you experience lower back pain, what score would you give it out of ten (Ten being the worst and zero being no pain at all)? ______________________

What treatment have you received for your lower back?

□ No Treatment

□ RICE (Rest, Ice, Compression, Elevation)

□ Topical treatments, dressings or strapping.(Please Specify)

□ Medical attention by a paramedic / emergency services / casualty department

□ Medical attention by a General Practitioner

□ Medical attention by a physiotherapist

□ Medical attention by specialist surgeon

□ Other

Topical treatments, dressings or strapping. (Please specify) ___________________

Other (please specify) _______________________________

If you saw a doctor or a physiotherapist for your back pain, what back injury did they diagnose you with? ____________________

Appendix 19: Training log

Week	Date	Injury (yes/no)	Putting (hrs:min)	Short (hrs:min)	Range (hrs:min)	Flexibility (hrs:min)	Postural training (hrs:min)	Weight training (hrs:min)	Cardio (hrs:min)	Rounds of gold (no.)
Week 1										
Week 2										
Week 3										
Week 4										
Week 5										
Week 6										
Week 7										
Week 8										
Week 9										
Week 10										
Week 11										
Week 12										
Week 13										
Week 14										
Week 15										
Week 16										
Week 17										
Week 18										
Week 19										
Week 20										
Week 21										
Week 22										
Week 23										
Week 24										
Week 25										
Week 26										

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