An investigation of potential kinematic factors associated with patellofemoral pain syndrome during running

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Declaration

I, Christopher Allan, hereby declare that the work on which this dissertation is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is submitted for another degree in this or any other university.

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cm	Centimetres
EMG	Electromyography
GM	Gluteus Medius muscle
GMax	Gluteus Maximus muscle
GRF	Ground reaction force
Hz	Hertz
ITB	Iliotibial band
Kg	Kilograms
m	Metres
PF	Patellofemoral
PFPS	Patellofemoral pain syndrome
Q angle	Quadriceps angle
VAS	Visual analogue scale
VL	Vastus Lateralis muscle
VMO	Vastus Medialis Oblique muscle
PFJRF	Patellofemoral joint reaction force

Glossary of terms

Patellofemoral pain syndrome	Pain of the knee involving the patella and retinaculum and excluding intra-articular and peri-articular pathology[1].
Kinematics	*"The branch of classical mechanics that describes the motion of points, bodies (objects) and systems of bodies (groups of objects) without consideration of the causes of motion"*[2].
Patellofemoral joint	Articulation between the patella and the femoral trochlea in the lower limb[1].
Pain	A physical and emotional response to a noxious stimulus that is specific to each individual; influenced by an individual's social environment, behaviour, attitudes and beliefs[3].
Range of motion (ROM)	Magnitude of movement occurring at a joint, expressed as degrees (˚)[4].
Visual analogue scale (VAS)	A 100 mm line anchored by *"no pain"* and *"worst imaginable pain"*, which can be used to determine pain intensity when marked by a participant[5].

Abstract

Background

Patellofemoral pain syndrome (PFPS) is a common clinical condition affecting physically active individuals. It is characterised by pain behind or around the patella during loading of the lower limb. It is recognised that there are multiple factors that contribute to PFPS; however these factors are not well understood. There is equivocal evidence for differences in lower limb kinematics in participants with PFPS, particularly during the running gait cycle.

Aim

The aim of this study was to investigate lower extremity kinematics during running in individuals with a history of PFPS compared to those without symptoms.

Specific objectives

(a) To describe lower extremity kinematics during running for individuals with PFPS. (b) To determine whether there are differences in pelvis, hip, knee and ankle kinematics during running in participants with and without PFPS. (c) To determine whether there were any kinematic variables at the pelvis, hip and knee joint during stance phase of running that may be associated with an increased risk of developing PFPS.

Methods

This study had a descriptive cross-sectional study design. Thirty one physically active individuals, who participated in at least two hours of physical activity per week for at least three months prior to testing, were recruited for the study. Fifteen participants presented with PFPS, and 16 participants without PFPS formed the control group. Participants were also required to have a Q-angle within the normal range for males (8.2º-14.2º) and females (11.4º-20.3º) respectively. Participants in the PFPS group were required to have a history of unilateral anterior or retro-patellar pain of non-traumatic origin that did not exceed a six-month period prior to testing. The participants' PFPS also needed to be elicited during one or more symptom provocation tests, namely: resisted terminal knee extension, stair descent, or a unilateral partial squat. The PFPS participants had to be able to run without pain for a minimum period of 10 minutes, which allowed the running test to be completed without reproducing symptoms of PFPS.

All participants gave written informed consent before taking part in the study. Participants were familiarised with all testing procedures. Participants completed medical and training questionnaires, and body composition measurements were performed. Sixteen retro-reflective markers were placed on anatomical landmarks of the lower limbs according to the modified Helen Hayes marker set. Participants were then required to perform a running test, which consisted of 10 sets of running at a self-selected speed on a 10 m pathway. Kinematic data of the pelvis, hip, knee and ankle were recorded by an eight-camera motion analysis system during each repetition of the test. The specific data extracted included range of motion at heel strike and toe off, peak range of motion during swing phase and stance phase. In addition, the range of motion travelled during stance and swing phases and the percentage of stance phase a participant took to reach the peak range of motion during stance phase were calculated.

Results

At foot strike the PFPS group had a decreased knee adduction angle, compared to the control group (p = 0.01). During stance phase the PFPS group had a significantly decreased peak knee flexion angle (p = 0.02); increased knee abduction/adduction range of motion (p = 0.03); increased hip rotation range of motion (p = 0.046); and less percentage of time taken to reach peak hip flexion during the stance phase (p = 0.04), compared to the control group. There were no significant differences between groups at toe off. During swing phase, the PFPS group had a significantly decreased peak hip flexion angle (p = 0.03); increased peak hip rotation angle (p = 0.046); and decreased knee flexion/extension range of motion (p = 0.03), compared to the control group.

A logistic regression analysis identified that the independent factors associated with the development of PFPS were 1) decreased knee flexion angle at foot strike; 2) decreased knee adduction angle at foot strike 3) increased peak hip internal rotation angle during the stance phase, and 4) decreased percentage of stance phase taken to reach peak hip flexion.

Discussion and conclusion

This study identified several lower limb kinematic factors associated with PFPS. These alterations in lower limb kinematics may contribute to the development of PFPS. They may also be compensatory adjustments in the running gait secondary to pain and potential muscle imbalances associated with PFPS.

Due to the fact that the injured group of subjects had pre-existing PFPS, it is not possible to state which findings were potentially compensatory or causative in nature. The findings unique to this study included decreased knee adduction at foot strike, increased hip rotation range of motion (ROM) during stance phase, and increased knee abduction/adduction ROM during stance phase. It was hypothesised that these findings could be causative due a potential associated increase in the Q-angle. The possible compensatory changes in the gait cycle included decreased peak knee flexion angle during stance phase; decreased percentage of stance phase to peak hip flexion; decreased peak hip flexion angle during swing phase; and decreased knee flexion ROM during swing phase. These changes may decrease the posterior directed patellofemoral joint reaction force and the patellofemoral joint reaction force, and may be associated with reduced knee pain. Further studies are required to properly differentiate between causative and compensatory changes in lower limb kinematics in participants with PFPS. It is recommended that treatment of PFPS should include gait re-education and other strategies to address changes in lower limb kinematics.

Chapter 1: Introduction and scope of thesis

1.1 Introduction

Patellofemoral pain syndrome (PFPS) is defined as pain around or under the patella, related to a physical activity that loads the lower limb[1]. It is one of the most common musculoskeletal conditions affecting the knee[1;6;7]. Patellofemoral pain syndrome is one of the most common running related injuries with incidence rates of 16% to 25%[6]. Occupational health statistics show an incidence of 11% of all musculoskeletal complaints within the workplace being due to be PFPS[6]. Notably, up to 25% of individuals with PFPS still have symptoms 20 years after the first onset of symptoms[6].

Although PFPS is a common condition, there is much debate regarding the underlying pathology and pathomechanics[8], as well as the most appropriate management of the condition[9]. Indeed, Crossley[9] et al concluded that current management of PFPS is not based on evidence from well controlled clinical trials. Factors associated with PFPS can be extrinsic or intrinsic in nature. Extrinsic factors relate to training amounts and methods, equipment used and surfaces trained on[10]. Intrinsic factors relate to inherent characteristics of an individual and include mechanical changes that occur within an individual, such as muscle strength and flexibility, as well as to gender and body weight. With PFPS, there is a complex interaction between the extrinsic and intrinsic factors – therefore the factors associated with this condition are multifactorial.

Until recently, it was suggested that most of the intrinsic factors associated with PFPS were caused by biomechanical factors occurring around the patellofemoral joint[11;12]. However, more recent kinematic studies have observed a relationship between PFPS and changes in both femoral and tibial kinematics as well[13;14].

Intrinsic factors now include among others (age, gender, BMI) anatomical anomalies, muscle strength deficits, alterations in flexibility and altered neuromuscular firing patterns at the pelvis, hip, knee, ankle and/or the foot. These factors can cause a multifactorial change in the kinematics seen in individuals with PFPS.

To better understand PFPS it is therefore vital to study the lower limb kinematic variables in individuals with PFPS. These variables may be directly causing the biomechanical factors that result in the patellofemoral joint overload. There may also be compensatory gait patterns that are utilised by the individual with PFPS to avoid pain or to enhance function. Once again, it must be noted that these kinematic changes can occur proximally at the pelvis and hip, locally around the knee or distally at the ankle and foot.

Most kinematic studies have focused on studying variables in response to tasks such as stair climbing and drop jumps[15-17]. Very few studies have investigated kinematic variables of patients with PFPS during running, yet PFPS is one of the most common running-related injuries. Furthermore, studies[6;9;11] that were done on running only analysed changes at the peak range of motion during the stance phase of gait. The full gait cycle was not analysed at the different phases (foot strike, toe off and swing phase). Considering that PFPS is the most common overuse injury in runners a better understanding is needed of the kinematics of PFPS in runners at various stages of the gait cycle. Proximal and distal kinematics as well as knee kinematics should be studied. This will increase our understanding and lead to more appropriate treatment strategies for the management of patellofemoral pain syndrome.

1.2 Aims and objectives

1.2.1 Aim

The aim of this study was to investigate lower extremity kinematics during running in individuals with a history of PFPS compared to those without symptoms.

1.2.2 Specific objectives

(a) To describe lower extremity kinematics during running for individuals with PFPS.

(b) To determine whether there are differences in pelvis, hip, knee and ankle kinematics during running in participants with and without PFPS.

(c) To determine whether there were any kinematic variables at the pelvis, hip and knee joint during stance phase of running that may be associated with an increased risk of developing PFPS.

1.2.3 Significance of the dissertation

Although PFPS is a common lower limb condition, current literature focuses on evidence for the relationship between local factors and PFPS, while proximal biomechanics of PFPS are not fully understood. The results of this research may therefore improve the understanding of the proximal and distal kinematic factors that may be associated with PFPS. In addition, it is hoped that the results of this research may facilitate the development of holistic physiotherapy treatment and rehabilitation protocols for PFPS.

1.3 Plan of development

In preparation for the experimental phase of this dissertation, a comprehensive review of the literature on patellofemoral pain will be presented (Chapter 2). This will be followed by a descriptive cross-sectional study that was designed to investigate potential differences in lower limb running kinematics in participants with and without PFPS (Chapter 3). A summary and conclusion section, including recommendations for future research (Chapter 4) will complete this dissertation.

Chapter 2: A review of the potential biomechanical factors associated with patellofemoral pain syndrome in active individuals

2.1 Introduction

Patellofemoral pain syndrome (PFPS) is one of the most common overuse, musculoskeletal conditions of the knee[1;6;7]. It is extremely common in active individuals with studies showing that it is the most widespread single diagnosis among runners, with an incidence of between 16% and 25%[1]. Within the work place, PFPS comprises 11% of all musculoskeletal complaints[1]. It has been shown to have both short and long term effects, with one in four sufferers demonstrating symptoms up to 20 years after the first presentation[1].

Patellofemoral pain syndrome is often described as "*runners knee*" or "*anterior knee pain*", but may be more specifically defined as "*pain of the knee involving the patella and retinaculum and excluding intra-articular and peri-patellar pathology*"[1]. The other term often used in conjunction with PFPS is chondromalacia patellae. However, this is a diagnosis that is made in a subset of individuals with anterior knee pain. It is a condition that is characterised by softening of the patellar articular cartilage, and can only be diagnosed with confidence at the time of knee surgery[1].

Patellofemoral pain syndrome is thought to be caused by a maltracking of the patella within the trochlea of the femur during repetitive flexion and extension of the knee joint[1]. A variety of extrinsic and intrinsic biomechanical factors may contribute to the maltracking of the patella[1]. These include factors related to range of motion, strength, neuromuscular control, local knee joint mechanics and mechanics of the lower limb and pelvis[14]. In this chapter, the literature pertaining to the prevalence, pathology, etiology, and potential biomechanical factors that may be associated with PFPS will be reviewed.

2.2 Review methodology

An online search was conducted using Pubmed, Science Direct, Medline and Google scholar databases. The following keywords were used: "*patellofemoral pain*", "*running*", "*cycling*", "*anterior knee pain*", "*pathology*", "*prevalence*", "*extrinsic causes*", "*intrinsic causes*", "*kinematics*", "*kinetics*", "*EMG*", "*lower limb*" and "*biomechanics*". All articles thought to be relevant to the topic where reviewed. The articles were also assessed for their level of evidence (LOE)[18].

2.3 Prevalence of patellofemoral pain syndrome

Patellofemoral pain syndrome is one of the most common conditions found in the lower limb. It is the most common diagnosis amongst active individuals, especially amongst runners[19]. Approximately 2.5 million runners are diagnosed with PFPS in one year[19]. The incidence rate of PFPS amongst runners varies from 10% to 25%[1;11;19]. Devereaux and Lachman[20] found that 25% of individuals that presented to a sports injury clinic with knee pain had PFPS[20]. Eleven percent of musculoskeletal complaints within the work place are caused by anterior knee pain, of which the most common cause is PFPS[1]. In addition, the incidence of PFPS in army recruits during combat training is reported to be as high as 37%[19].

Numerous studies have reported that PFPS occurs between two to 10 times more frequently in females compared to males[21]. Recently, Boling et al[22] examined the incidence of PFPS in individuals from the United States Naval academy over a period of 2.5 years[22]. Females were 2.23 times more likely to develop PFPS than males. It was concluded that although there is a higher incidence of PFPS amongst females, further research is needed to establish the underlying reasons for the gender differences in the incidence of PFPS.

Patellofemoral pain also has a high rate of recurrence and may develop into a chronic condition. The incidence of recurrent or chronic PFPS has been found to be between 70% and 90%[19].

In a recent study, 80% of participants with PFPS who had received a rehabilitation programme still had symptoms, and 74% had reduced activity levels five years later[19]. In addition, there is some evidence to suggest that having PFPS at a young age may be a predisposing factor to patellofemoral osteoarthritis in later years[19].

It is therefore evident that PFPS is a prevalent condition that may be associated with longstanding pain and loss of function[1;11;19;20;22]. The next sections will review the current definitions of PFPS, and the anatomy and pathophysiology of PFPS.

2.4 Definition of patellofemoral pain syndrome

"*Patellofemoral*" refers to both the patella and femur and the joint between them. This is an appropriate term as there is no specific structure that has been defined as the source of the pain in PFPS[23]. "*Pain*" is the major symptom experienced by patients, although they can also present with instability, clicking, and other less prominent symptoms[23]. Because of this variety of symptoms that can occur in combination, the appropriate term for this grouping of symptoms is a "*syndrome*"[23].

Patellofemoral pain syndrome is a condition that presents as retropatellar and/or peripatellar pain[24]. The pain is associated with activities that load the lower limb, such as walking, running, stair climbing or descending and prolonged sitting[24]. Patellofemoral pain syndrome is one of the conditions categorised as anterior knee pain. Some other causes of anterior knee pain include: articular cartilage injury, patellofemoral arthritis, quadriceps tendinopathy, Sinding-Larsen-Johansson syndrome, prepatellar bursitis and pain referred from the lower back and/or hip[24].

2.5 Anatomy and biomechanics of patellofemoral pain syndrome

The patellofemoral joint is the articulation between the patella and the femoral trochlea[1]. The main function of the patella is to provide a mechanical advantage to the muscles used to extend the knee joint. As the knee flexes, the position of the patella results in a relative lengthening of the lever arm, and therefore a mechanical advantage is offered to the extensors[1].

The patella is a sesamoid bone within the quadriceps muscle tendon. The quadriceps muscle (consisting of the vastus medialis, vastus intermedius, vastus lateralis and rectus femoris) inserts into its superior margin. The patella then attaches to the tibial tubercle via the patellar tendon. It is held in place by a variety of static and dynamic structures. The static structures include the retinaculum, ligaments, cartilage and bone, while the dynamic structures are the muscles involved in moving and positioning the patella[10;25].

The lateral retinaculum, which is fibrous tissue, extends from the surrounding musculature, in particular the vastus lateralis muscle, and attaches to the border of the patella[1]. It also has attachments to the lateral epicondyle of the femur and to Gerdy's tubercle of the tibia[1]. The medial patellofemoral ligament is a thin ligament that pulls the patella into the trochlea in early flexion. It originates from the medial epicondyle of the femur, with attachments to the adductor muscle group. It then runs deep to the vastus medialis oblique muscle, and attaches to the superomedial aspect of the patella. In addition to these static structures, the bony ridges of the trochlea provide a bony, mechanical block to the medial and lateral movement of the patella. The lateral condylar ridge is more prominent and mechanically stops the patella from dislocating laterally[25].

The vastus medialis oblique muscle (VMO) acts as the main dynamic stabiliser of the patella. It attaches to the patella and the distal femur and has a more oblique orientation of its fibres than the rest of the quadriceps muscles.

This orientation of fibres creates a medially directed muscle contraction that assists in maintaining the position of the patella in the intercondylar notch. The posterior surface of the patella has facets that articulate with the femoral condyles. These include a medial and lateral facet, which are separated by a vertical ridge. The medial and lateral facets may be further divided into superior, middle and inferior facets, with an odd facet located on the far medial aspect. The medial and lateral facets articulate with the medial and lateral condyles of the femur[1]. Different areas of these facets articulate with the condyles at different ranges of knee flexion and extension. These areas of contact are termed the patellofemoral contact areas and the force of the contracting quadriceps is transmitted through them[1].

The area of contact varies at varying degrees of knee flexion. In general, the contact area moves, as a band, up the patella from the inferior pole to the superior pole, broadening with more flexion (Figure 2.1). By 90° of flexion the area of contact has reached the superior pole and from here until 135° of flexion the area of contact moves to the medial and lateral surfaces of the patella[26].

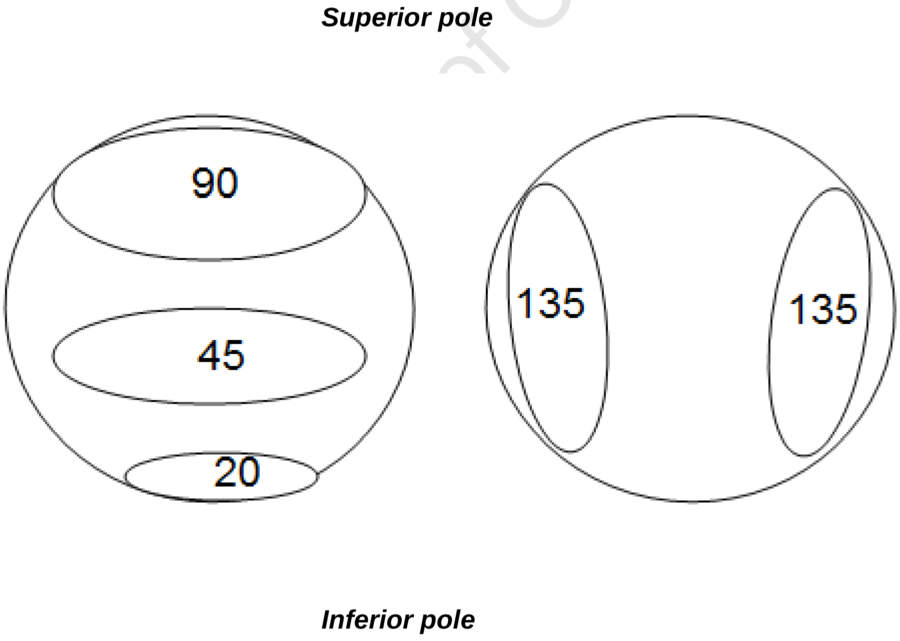

Figure 2.1: Distribution of forces on the posterior surface of the patella during varying ranges of knee flexion and extension[26].

During knee flexion and extension, the patella is forcefully directed posteriorly against the femur. This is due to the quadriceps muscles transmitting forces superiorly and posteriorly towards the hip joint; and the patella tendon transmitting forces inferiorly and posteriorly towards the tibial tuberosity. These two forces create a resultant posteriorly directed force[14]. This posteriorly directed force is termed the patellofemoral joint reaction force and is the force of the ventral surface of the patella against the femoral condyles. The specific areas of contact are shown in Figure 2.1. The patellofemoral joint reaction force increases as both knee flexion, and the strength of the quadriceps contraction increase. Stresses applied on the patellofemoral joint during level walking have been calculated at 2 – 4 MNm^{-2} [7], while stair climbing generates approximately three times a person's body weight through the joint on both stair ascent and descent[1] and running forwards generates 4.5 times a person's body weight. These forces are directed onto the patellofemoral contact areas, not the entire posterior surface.

In addition, due to the normal structural alignment of the lower limb being slightly valgus, the quadriceps force and the patellar tendon force also act on the patella in the frontal plane. The quadriceps force is directed from the insertion of the quadriceps tendon on the superior margin of the patella, back along the femur. The patellar tendon force is the force directed by the patella tendon down towards its insertion onto the tibial tuberosity. The combination of these two forces, in the frontal plane, create a resultant lateral force[14]. This lateral force, if not balanced out by a medially directed force, can result in the patella tracking more laterally in the intercondylar groove. This abnormal tracking or "*maltracking*" of the patella is often thought to be the cause of PFPS[6] by causing an increase in compressive forces on the PF joint. Maltracking has been shown to be present in some studies[27] but not all[14;28].

This lateral force is related to the quadriceps (Q) angle. The Q angle is measured as the angle formed at the intersection between the line drawn from the anterior superior iliac spine to the midpoint of the patella; and the line from the tibial tubercle to the midpoint of the patella[14].

As the Q angle increases, the laterally directed force vector increases[14]. The normal values for the Q-angle are 15.8° ± 4.5° for females and 11.2° ± 3.0° for males[1;14;26;29].

With the amount of stress being directed posteriorly onto small areas of the articular cartilage, disturbances in the amount of pressure, or in the size and shape of the contact areas, may cause imbalances that can lead to possible pathology. With lateral forces influencing the patella's position in the intercondylar groove, and its tracking through the groove during flexion and extension, an imbalance in these forces will predispose to maltracking of the patella. This maltracking changes the contact areas and causes a disruption of the pressure and it is these disturbances that result in the pathology found in PFPS[6].

2.6 Pathophysiology of patellofemoral pain syndrome

The pathophysiology of PFPS is generally poorly understood, due to the multifactorial nature of this condition[10]. There are numerous tissue structures around and within the patellofemoral joint that are susceptible to stress[30;31]. These structures include cartilage, synovium, retinaculum, bone and ligaments. When this stress exceeds the mechanical strength of the tissue microdamage, inflammation and pain will result[10]. However, only those structures that have neuroreceptors are able to generate signals that may be perceived as pain by an individual[30;31].

In PFPS, the cartilage lining the under-surface of the patella is thought to be most overloaded when maltracking occurs. However, cartilage does not have any pain neuroreceptors. It is thought that, when damaged, cartilage causes a mechanical irritation of the synovium in the area, which may lead to the development of PFPS. The continual irritation of the synovium and altered forces on the cartilage could also transfer excessive forces to the subchondral bone, thus exciting nociceptors and causing pain[10;30;31].

Another structure that needs to be considered as a potential cause of PFPS is the lateral retinaculum. The continual lateral movement and positioning of the patella could cause a shortening of the lateral retinaculum, which in turn could cause nerve damage, similar to a Morton's neuroma[10]. The fat pad of Hoffa and medial patellofemoral ligament have also been thought of as potential sources of the pain[10;31]. These structures are exposed to frequent loading during the gait cycle. If this loading exceeds the amount of strain these structures are able to withstand, irritation of the neuroreceptors, and therefore pain, may result[30].

Patellofemoral pain results from an increase in the stress placed on the various structures around the patella. These stresses may be acute or chronic in nature[19]. Acute onset PFPS may be due to a fall, a direct blow to the patella, or a dislocation of the patella. During the acute incident, the patella is forced against the femur. The resulting joint reaction forces could be high enough to cause immediate damage to the cartilage and possibly the subchondral bone of the patella, resulting in pain. Although acute onset patellofemoral pain syndrome, may not have had a biomechanical cause, the resulting pain and damage to the patella, may cause the same biomechanical changes as seen in chronic onset PFPS.

The chronic onset of PFPS occurs when the patellofemoral joint reaction forces are unevenly distributed over an extended period of time. This uneven distribution of forces and continual overload on a specific area eventually results in micro tissue damage, inflammation and pain as mentioned earlier. The chronic onset of PFPS may be influenced by either extrinsic or intrinsic factors[19]. Potential predisposing factors to PFPS will be reviewed in the next section.

2.7 Factors associated with the development of patellofemoral pain syndrome

2.7.1 Extrinsic factors

Any extrinsic factor that alters lower limb biomechanics or load may affect the patellofemoral joint. These factors include equipment used, training errors or training surface. Although extrinsic factors are often mentioned anecdotally in relation to PFPS, there is limited evidence to support the potential relationships between extrinsic factors and the onset of PFPS[1;32].

Van Zyl et al[32] commented on extrinsic causes of PFPS in cyclists. They stated that 80% of cyclists presenting with PFPS demonstrated an abnormal mediolateral movement of the knee during the down stroke. In two related studies[33;34], the reduction of mediolateral movement of the knee was associated with a reduction in PFPS symptoms in cyclists. This movement was reduced by changing saddle height and/or placing a wedge into the cyclist's shoe[33;34]. Van Zyl et al[34] also stated that training errors might contribute to the development of PFPS, and that alterations in training to reduce the load on the joint may facilitate healing.

It is suggested that controlling the foot and lower limbs biomechanics with orthotics or specific running shoes may reduce the risk of injuries in runners[35]. In a prospective study of injuries in recreational runners in Vancouver, running shoe age and running frequency (days per week) were associated with injury[36]. However, Cheung et al[37] suggested that, although there is a link between foot and shank movement and PFPS, there is no reported association between PFPS and footwear. In addition, Richards et al[38] reviewed the evidence-based prescription of running shoes, and found that there was no evidence to support the use of specific shoes for injured runners.

Another extrinsic factor that has been suggested to cause PFPS is over-training[39;40]. A training programme that includes excessive speed, hill, distance or strength training, or uses incorrect gear ratios on a bicycle may cause excessive loading of the patellofemoral joint over time, leading to the development of PFPS[39]. Other training factors that may also influence all running overuse injuries include previous running experience, previous injury, competitive running and excessive weekly mileage[39].

Interestingly, three studies have examined the link between physical fitness and the incidence of PFPS[39-41]. In these studies, the individuals at risk of developing PFPS were found to participate in fewer hours of sports per week than the controls. This seems contradictory to the statement that excessive training hours per week is a cause of PFPS.

The studies explain this contradiction, by suggesting that an excess of training hours per week, over and above what an individual is used to, is possibly a cause of PFPS[39;41].

2.7.2 Intrinsic factors

Intrinsic factors that may be associated with the development of PFPS include gender, anatomical variations, flexibility, strength, neuromuscular control and kinematics[19]. These intrinsic factors may alter the biomechanics of the lower limb, which in turn may lead to altered biomechanics of the patellofemoral joint[11;14;19], resulting in maltracking of the patella, an uneven distribution of the forces onto the contact areas and the subsequent development of pathomechanics of the PFJ[14;19]. Intrinsic factors may be classified as being either local, remote proximal or remote distal[19]. This section focuses predominantly on the kinematic factors that may contribute to the development of PFPS as this is the emphasis of this dissertation.

2.7.2.1 Anatomical variations

Minor anatomical variations can affect the alignment of the lower limb. Anteversion, retroversion, coxa vara and coxa varum at the hip; genu varum, genu valgum and patella alta at the knee; and rear foot inversion or eversion are just some of the anatomical variations that can affect the biomechanics of the lower limb.

In a study by Souza et al[42], MRI studies were done to determine the difference in femoral inclination and anteversion between a group of individuals with PFPS and a healthy pain free group. The results of this study showed that there was a significant difference in the femoral inclination angle but not in the femoral anteversion angle between the two groups. The PFPS group had a greater femoral inclination angle. These factors may lead to local biomechanical changes that will increase patellofemoral load, and may also alter the Q angle. As described in Section 2.5 (page 9), the Q angle is the angle created by the patella tendon pulling the patella inferiorly and laterally and the quadriceps tendon pulling the patella superiorly and laterally. This creates a resultant lateral force on the patella and is seen as a contributing factor to PFPS. Some of the anatomical variations mentioned already may affect the Q angle.

However, the relationship between Q angle and the signs and symptoms of PFPS is not always consistent[1;14;26;29;43]. There are two possible explanations for this. It is possible that static measurements of the Q angle do not account for segmental motion of the tibia or femur during movement, which may influence lateral force vectors, and the Q angle[14;29]. For example, if there is tibial internal rotation at toe off, the tibial tuberosity will move medially and the Q angle will decrease. The same limb may have increased tibial external rotation at heel strike, causing an increase in the Q angle. It is therefore evident that, although anatomical variations or deformities, such as femoral anteversion or coxa vara increase the static Q angle, abnormal motions of the lower limb may alter the dynamic Q angle[14;29]. Powers et al[12] observed significant differences between dynamic Q angles in participants with PFPS compared to pain-free participants. This was most evident during stair descent, which is the task most commonly associated with symptoms of PFPS.

The position of the patella in the trochlea groove may also influence Q angle measurements[26]. For example, a patella that is being laterally displaced due to inflexible lateral structures will have a decreased Q angle. Herrington et al[26] found that correcting the position of the patella before taking the Q angle measurement changed the outcome of the measurement.

Participants with a normally laterally displaced patella had an increased Q angle once the position of the patella was corrected. Participants with a medially displaced or neutral patella had a less significant Q angle after repositioning the patella[26].

For these reasons, a simple static measure of an individuals Q angle is not necessarily a good indicator of the lateral force exerted on the patella during gait. The lateral force could still be a contributing factor to maltracking but a more thorough method of measuring it is needed before its significance, as a predisposing factor for PFPS, can be established. The measurement needs to take the dynamic Q angle and the existing position of the patella into account[26].

In terms of the anatomy of the actual VMO, an ultrasound study of the insertion level, fibre angle and volume of the VMO in individuals with PFPS compared to uninjured controls, was reported [44]. This study showed that all three measures were significantly different between the groups. With the more proximal insertion level of the VMO, it was hypothesised that the lever arm would be shortened and would be less effective at stabilising the patella medially. The fiber angle of the VMO would alter the direction of the force the muscle exerts on the patella and with it being lower, as found in this study; the effectiveness of medial stabilisation is once again reduced. There was also a significant difference in volume of the VMO between groups, with the PFPS group being significantly less. This was also seen in a MRI study of the cross sectional area of the VMO by Pattyn et al[45]. This could be an indication of strength of the VMO and once again the effectiveness of the muscle to medially stabilise. The fact that there are such marked differences in the morphology of the VMO in individuals with PFPS indicates its importance in the cause and therefore rehabilitation of PFPS.

2.7.2.2 Flexibility

In a systematic review on risk factors for patellofemoral pain syndrome[46] only one article, by Witvrou et al[47], examined flexibility, amongst other factors, as a risk factor for PFPS. In the two-year prospective study[47;48], 282 students taking part in physical training over a two year period were monitored.

Each participant was assessed every three months for the two year period. They were evaluated for anthropometric variables, physical fitness, general joint laxity, lower leg alignment characteristics, muscle length and strength, static and dynamic patellofemoral characteristics and psychological parameters. Twenty four students developed PFPS during the two year study period. The flexibility of the hamstrings, quadriceps and gastrocnemius muscles were assessed during that period. The hamstring muscles of the participants were assessed in supine. The examiner performed a straight leg raise and measured the amount of hip flexion with a goniometer. Quadriceps muscle flexibility was assessed in prone. The maximum knee flexion angle was measured on the affected leg with the opposite foot on the floor and the hip bent 90°. Gastrocnemius muscles flexibility was measured with the participant in standing. The amount of ankle dorsiflexion was measured while leaning forward from the ankle, keeping the heel on the floor and the knee straight. A significant correlation was found between a shortened quadriceps muscle, altered vastus medialis muscle reflex response time, decreased explosive strength, and a hyper mobile patella and the incidence of PFPS. It would seem that although a decrease in flexibility is implicated in the development of PFPS[48;49], this study only found the quadriceps to be a predisposing factor.

2.7.2.3 Strength

Several studies have examined the strength of specific muscles, namely the knee extensors, hip abductors, hip extensors and hip external rotators, in participants with PFPS, compared to controls[6;8;11;17;48;50-54]. The main hypothesis of these studies is that decreased muscle strength may be associated with an inability of the muscles to dynamically control the position of the lower limb during specific tasks, thus predisposing to PFPS. Table 2.1 provides a summary of relevant experimental studies that assessed muscle strength in participants with and without PFPS[6;8;11;17;48;50-54]. Some of these articles investigated other factors other than strength[6;8;11;17;48;50;53], and will be discussed in later sections of this review. The level of evidence (LOE) according to evidence based medicine criteria is noted[18].

Table 2.1 Summary of experimental studies that assessed muscle strength in participants with and without PFPS.

Article	Study design	Participants	Strength testing performed	Results
Ireland et al[52]	Cross sectional laboratory study (Level of evidence (LOE): III)	15 females with PFPS and 15 matched controls with no history of knee pain	Isometric strength of hip abduction and external rotation with a handheld dynamometer	PFPS group was weaker in hip abduction ($p < 0.001$) and external rotation ($p < 0.001$)
Robinson et al[54]	Cross sectional laboratory study (LOE: III)	10 females with PFPS and 10 matched controls with no history of knee pain	Isometric strength of hip abduction, extension and external rotation with a handheld dynamometer	PFPS group was weaker in hip extension ($p < 0.001$), hip abduction ($p = 0.007$) and then hip external rotation ($p = 0.004$) when compared to controls and to the uninvolved leg
Boling et al[51]	Case control design (LOE: III)	20 participants (13 female and 7 male) with PFPS and 20 matched controls with no history of knee pain	Isokinetic strength of hip extension, abduction and external rotation with an isokinetic dynamometer	Weaker peak hip eccentric abduction torque ($p = 0.014$); and weaker average concentric ($p = 0.048$) and eccentric ($p = 0.032$) hip external rotation torque in the PFPS group.
Piva et al[53]	Case control design (LOE: III)	30 participants (17 female and 13 males) with PFPS and 30 age and gender matched control participants with no history of knee pain	Isometric strength of hip abduction and external rotation with a handheld dynamometer	PFPS group had decreased strength in hip abduction ($p = 0.016$)
Bolgla et al[6]	Cross sectional laboratory study (LOE: III)	18 females with PFPS and 18 matched controls with no history of knee pain	Isometric strength of hip abduction and external rotation with a handheld dynamometer	PFPS group was weaker in hip abduction ($p = 0.006$) and external rotation ($p = 0.002$)

Table 2.1 Summary of experimental studies that assessed muscle strength in participants with and without PFPS (continued).

Article	Study design	Participants	Strength testing performed	Results
Dierks et al[11]	Cross-sectional experimental laboratory study (LOE: III)	20 recreational runners with PFPS and 20 matched uninjured runners with no history of knee pain	Isometric strength of hip abduction and external rotation with a handheld dynamometer	Both groups had decreased strength in hip external rotation ($p < 0.01$) and abduction ($p < 0.01$) with fatiguing run; PFPS group was significantly lower before and after run.
Willson et al[55]	Controlled laboratory study (LOE: III)	20 females with PFPS and 20 matched controls with no history of knee pain	Isometric strength of hip abduction and external rotation and lateral trunk flexion with a handheld dynamometer	Decreased strength of lateral trunk flexion (24% lower; $p = 0.06$), hip abduction (13% lower; $p = 0.09$) and hip external rotation (14% lower; $p = 0.03$) in PFPS group
Souza et al[8]	Controlled laboratory study using cross-sectional design (LOE: III)	21 females with PFPS and 20 matched female controls with no history of knee pain	Isometric strength of hip abduction and hip extension with an isokinetic dynamometer capable of testing isometric strength	Decreased hip extension torque ($p = 0.005$) and hip abduction torque ($p = 0.02$) in PFPS group
Souza et al[42]	Cross sectional study (LOE: III)	19 females with PFPS and 19 matched uninjured females in the control group	Isometric, isotonic and isokinetic testing of hip extension and pelvis drop; Isometric testing of hip external rotation and abduction	Decreased strength in isometric pelvic drop ($p = 0.001$), hip external rotation ($p = 0.002$), hip extension ($p = 0.01$), Hip Abduction ($p = 0.04$); isokinetic eccentric pelvis drop ($p = 0.02$), isokinetic concentric hip extension ($p = 0.03$); isotonic pelvis drop ($p = 0.008$), isotonic hip extension ($p < 0.001$)
Boling et al[50]	Cohort study design (prognosis) (LOE: I)	40 PFPS participants out of 1597 US naval recruits	Isometric strength of knee flexion and extension; hip abduction, extension, external and external rotation	Decreased knee extension torque ($p = 0.01$) and flexion torque ($p = 0.02$); increased hip external rotation torque ($p = 0.04$) in PFPS group

(A) Trunk lateral flexion strength

Willson et al[55] assessed lateral trunk flexion strength in a side lying plank position. The handheld dynamometer was placed directly inferior to the lateral iliac crest and the individual exerted a maximal voluntary contraction upwards against it. It was found that the PFPS group had a decrease in lateral trunk flexion strength compared to healthy controls. The hip abductors would also be used in this test position, so the strength deficit may be a combination of hip abduction and trunk lateral flexion. Hip abduction strength was also shown to be decreased, so it is not possible to state how much of this strength deficit is due to weak hip abduction and how much is due to weak trunk lateral flexion.

(B) Standing pelvic drop

One study[42] investigated the isometric, isokinetic and isotonic strength of standing pelvic drop. Pelvic drop strength was assessed with a multimodal dynamometer, with the individual standing on their affected leg and their uninjured leg attached to the torque arm of the dynamometer. Isometric strength was assessed with the participant trying to elevate their uninjured limb with maximal voluntary effort by abducting their weight bearing leg (hip hike). Concentric and eccentric isokinetic testing was performed in the same manner between 10° abduction and 10°adduction of the weight bearing leg at $10°.s^{-1}$. Isotonic endurance testing was performed until there was a drop of ≤ 75% power output. All pelvic drop strengths were decreased except for concentric isokinetic strength. This decrease in strength could result in excessive pelvis tilting during running which would result in a tightening of lateral structures and more lateral force on the patella.

(C) Hip abduction strength

The ten studies reviewed[6;8;11;42;50-55] that assessed hip abduction strength, found weakness in the hip abductors in PFPS participants compared to controls. Seven of these studies[6;11;50;52-55] used isometric testing with a handheld dynamometer, two studies used an multimodal dynamometer to test isometric torques[4;42] and one study used an isokinetic dynamometer to determine both concentric and eccentric torques[44]. The test was always performed with the participant in side lying with the affected leg on the top.

For the isometric test the participant exerted a maximal voluntary isometric contraction upwards against a handheld dynamometer placed just proximal to the lateral femoral epicondyle[6;11;50;52;54;55] or just proximal to the lateral malleolus[53]. When the multimodal dynamometer was used, the axis of the dynamometer was aligned with their hip joint centre and the resistance pad was placed over the lateral femoral epicondyle of the knee. The isometric test performed on the multimodal dynamometer[4;42] involved exerting a maximal voluntary contraction against the resistance pad, which didn't move but was able to measure torque exerted. For the isokinetic test, both concentric and eccentric strength tests were performed through the available range of motion at $60°/s^{-1}$.

Willson et al[49] and Dierks et al[17] tested abduction strength before and after specific fatiguing activities and found that the weakness was more pronounced after exertion or a prolonged run respectively. The isokinetic strength testing protocol used by Boling et al[51], comparing 20 PFPS participants to 20 control participants, showed that the PFPS group was weaker than the control group for peak eccentric hip abduction. In all the case controlled studies comparing already injured participants to healthy controls, a weakness in hip abduction strength was found, but in the prospective study by Boling et al[50], decreased hip abduction strength was not found as a possible risk factor for PFPS. This could imply that the weakness develops as a result of the PFPS.

(D) Hip external rotation strength

Nine[6;11;42;50-55] of the studies reviewed assessed hip external rotation strength. Eight studies[6;11;42;50;52-55] assessed isometric strength using a handheld dynamometer with participants either in prone[46] with the hip at 0° and knee at 90°; or in sitting[6;11;50;52-55] with their hip and knee both flexed 90°. The isometric dynamometer was placed proximal to the medial malleolus and attached to a fixed object. Although the external rotators were tested at two different angles, both positions showed a decrease in strength. Boling et al[50;51] used an isokinetic dynamometer, and external rotation strength was measured in sitting with the hip and knee flexed 90°. Concentric and eccentric external rotation strength was assessed through full available range of motion at $60°/s^{-1}$.

All the case controlled studies showed a decrease in hip external rotation strength in PFPS participants when compared to healthy, matched controls. This included the isokinetic testing, which showed a decrease in the average eccentric and concentric torque of the external rotators. However in a prospective study Boling et al[50] showed that an increase in hip external rotation torque was a potential risk factor for PFPS. They conducted a prospective investigation to determine risk factors for the development of PFPS in Naval recruits over a 2.5-year period. Range of motion and muscle strength of the knee and hip were assessed during jump landing tasks. Three predisposing factors for PFPS were identified, namely decreased quadriceps muscle strength, increased strength of the external rotators of the hip, and increased internal rotation ROM of the hip. It was hypothesized that the individuals that developed PFPS had the increased hip external rotation strength to control the excessive internal rotation on the jump-landing task.

(E) Hip extension strength

Five studies[8;42;50;51;54] examined the strength of hip extension in PFPS individuals compared to healthy control participants. These studies used two different positions to test hip extension strength. The first position was with the individual in prone with their knee bent 90° and their hip in 0° extension and slight external rotation[47]. The participants exerted a maximal voluntary contraction against the handheld dynamometer placed over the distal posterior thigh, in an extension direction. The other position of testing was in prone with the lower limbs off the edge of the testing table. The individuals being tested in this position either exerted a maximal voluntary contraction against a handheld dynamometer[43] or a multimodal dynamometer[4;42] for isometric testing. Isokinetic testing of concentric and eccentric muscle strength was also performed in this position in two studies[42;51]. For the isokinetic testing, the axis of rotation was positioned with the hip joint centre and the resistance pad placed over the posterior thigh, proximal to the popliteal fossa. The tests were performed at $60°/s^{-1}$ and $10°/s^{-1}$. Souza et al[42] also conducted isotonic testing until fatigue of hip extension in the same position. Fatigue was defined when power dropped by ≥ 75%. There was a significant decrease in strength in the PFPS group compared to the healthy individuals.

Boling et al[50;51] found no significant difference in hip extension strength between groups. In contrast studies by Souza et al[4] and Robinson et al[47] both found a decrease in hip extension strength in individuals with PFPS. It was hypothesised that this decrease in gluteus maximus muscle strength (the main hip extensor) was a possible contributing factor to the increase in hip internal rotation range of motion during activities in PFPS participants. This is because the gluteus maximus muscle also functions as an external rotator of the hip. Souza et al[42] conducted a stepwise regression analysis to compare various hip muscle strengths to the degree of average hip internal rotation. The only predictor of average hip internal rotation in PFPS individuals was isotonic hip extension endurance.

(F) Knee extension strength

In the prospective study by Boling et al[43], knee extension strength was tested in all participants at the beginning of the two year study. It was tested in sitting with the hip and knee at 90° using a handheld dynamometer. When a regression analysis was completed comparing the injured and uninjured groups at the end of the two year period, it was found that weak knee extension, and therefore decreased quadriceps muscle strength, was a risk factor for the development of PFPS.

(G) Knee flexion strength

In the same study by Boling et al[43], using the same study design and testing knee flexion, therefore hamstring muscle strength, it was found that a decrease in the hamstring strength was a risk factor for the development of PFPS. They stated that "*the exact relationship between decreased hamstring strength and the development of PFPS is not clearly understood; however, decreased hamstring strength may be due to an overall weakness of the thigh musculature in people who develop PFPS.*"

2.7.2.4 Neuromuscular control

Although strength deficits have been shown to be contributing factors to PFPS, the recruitment patterns of the same muscles may also contribute to the development of PFPS. Table 2.2 provides a summary of relevant experimental studies that assessed neuromuscular control of the lower limb in participants with and without PFPS. The level of evidence (LOE) according to evidence based medicine criteria is noted[18].

Table 2.2: Summary of experimental studies that assessed muscle activity in participants with and without PFPS.

Article	Study design	Participants	Neuromuscular investigation	Results
Brindle et al[15]	Case controlled study (LOE: III)	16 participants with PFPS (12 female; 4 male) and 12 age matched controls (7 female and 5 male) with no history of knee pathology	EMG activity of vastus medialis oblique (VMO); vastus lateralis (VL); gluteus medius (GM) while ascending and descending stairs	Delayed onset of GM muscle contraction before foot strike ($p = 0.004$) in PFPS participants
Crossley et al[16]	Case controlled study (LOE: III)	48 participants with PFPS and 18 matched controls with no history of knee pathology	EMG activity of VMO and VL during a step up and step down task	There was a delay in the onset of the VMO relative to the VL in PFPS participants ($p = 0.02$)
Mellor et al[56]	Case controlled study (LOE: III)	10 participants with PFPS (7 females; 3 males) were compared to 10 matched controls from an earlier study	EMG activity of VMO and VL during isometric knee extension at 30° knee flexion	There was a reduction in the synchronisation between VMO and VL in PFPS ($p < 0.0001$)
McClinton et al[57]	Case controlled study (LOE: III)	20 participants (9 female; 11 males) with PFPS and 20 matched control participants (10 female; 10 male)	EMG activity of VMO and VL during step ups at 5 different heights	VMO:VL ratios were not different between groups but PFPS participants had increased activation duration ratio's ($p = 0.04$)

Table 2.2: Summary of experimental studies that assessed muscle activity in participants with and without PFPS (continued).

Article	Study design	Participants	Neuromuscular investigation	Results
Souza et al[8]	Controlled laboratory, cross sectional design study (LOE: III)	21 female participants with PFPS and 20 matched control participants	EMG activity of gluteus maximus (GMax) and GM during running, a drop jump and a step down task	There was an increase in activation of GMax during step down and running tasks in the PFPS group (p = 0.04)
Willson et al[58]	Cross sectional design study (LOE: III)	20 females with PFPS and 20 matched healthy females in the control group	EMG data for gluteus medius and gluteus maximus. Onset timing, activation duration, mean activation level and peak activation level	Delayed (p = 0.03) and shorter activation (p = 0.01) of gluteus medius in the PFPS group

(A) Vastus medialis oblique muscle and vastus lateralis muscle recruitment

The most researched muscle recruitment pattern in relation to PFPS is the relationship between vastus medialis oblique (VMO) and vastus lateralis (VL). For a long time it has been thought that the muscle recruitment pattern of VMO in relation to VL is important, in order to time and control the movement of the patella into and through the interchondylar groove during knee flexion and extension[12;56;57]. The vastus medialis oblique muscle has been shown to be an important medial stabiliser of the patella[30].

Much research has been focused on the activation patterns and neuromuscular control of these muscles[56;57]. Mellor et al[56] examined the synchronisation of VMO and VL in participants with PFPS compared to control participants, during a resisted knee extension test. There was a difference in the synchronisation of the VMO and VL activation patterns in most PFPS participants. Powers et al[12;59] compared the movement of the patella and femur at the knee in weight-bearing and non-weight-bearing situations, using kinematic magnetic resonance imaging. It was found that in a non-weight bearing position the patella seems to move within the trochlea groove, while in a weight bearing position, the femur moves under the patella. The lack of movement of the patella in a weight bearing situation has also been shown by MacIntyre et al[28;44]. MRI studies of the patella during a closed chain task and were unable to show abnormalities in the tracking of the patella. With this difference in movement patterns between weight bearing and non-weight bearing tasks, it could possibly be expected that there is a difference in activation patterns of the surrounding musculature. So the results found in a non-weight bearing study cannot necessarily be translated to a weight bearing situation like running.

McClinton et al[57] compared the onset timing and activation of the VMO and VL of participants with PFPS and controls, during walking up varying step heights. There were no significant differences between VMO and VL timing and magnitude ratios at various step heights. These findings do not support the theory that VMO activation is delayed or inhibited in participants with PFPS.

Their testing was done in a weight bearing situation, during which Powers et al[12] has shown that the femur moves under the patella. In this situation it would be informative to evaluate muscles affecting the femoral motion, like the muscles controlling hip motion.

In addition Chester[60], conducted a systematic review of all research studies investigating relative timing of onset of the VMO and VL in participants with PFPS compared to asymptomatic participants. Although there was a trend towards a delay in VMO to VL onset, there was substantial variety in the findings. It was concluded that the clinical and therapeutic significance of this aspect of PFPS is difficult to assess.

(B) Gluteus muscle recruitment

Brindle[15], investigated the EMG activity of the gluteus medius muscle, VMO and VL during closed chain activity of ascending and descending stairs in participants with PFPS and controls,. There were differences in the EMG activity of the VMO and VL muscles between groups. However there was a delayed onset and shorter firing duration of the gluteus medius muscle during the stair ascent and descent in participants with PFPS, compared to pain free control participants.

Further, Souza et al[8] examined lower limb kinematics, strength and average EMG magnitudes of gluteus medius and maximus during running, drop jumps and step downs in participants, with and without PFPS. Participants with PFPS had increased peak internal rotation ROM; decreased hip extension and abduction strength; and increased gluteus maximus recruitment compared to the control group. There was no difference in average gluteus medius EMG activity between groups. It was hypothesized that the increased gluteus maximus recruitment was compensatory for the decreased hip extension and abduction strength. In contrast Willson et al[58] found changes in gluteus medius activation timing and duration but no change in gluteus maximus activation patterns during a running trial in participants with PFPS compared to an uninjured control group. These differences may potentially be related to the different methods of EMG analysis.

Souza et al analysed EMG activity averaged over the entire stance phase; while Willson et al focused EMG activity immediately prior to foot strike and during the first 50% of stance phase. The gluteus medius is far more active in the period before foot strike and during the first 50% of stance phase and is resting in the later part of the stance phase, which may account for the contrasting findings.

Neuromuscular control has been shown to be of importance in the aetiology of sports injuries[52]. Previous studies have investigated neuromuscular control of the lower limb in injuries such as achilles tendinopathy[61] and iliotibial band syndrome[62]. It has been postulated that a loss of neuromuscular control may be associated with altered lower limb kinematics, in the frontal or transverse planes, at specific times of the gait cycle, which may predispose to the development of PFPS[8]. Poor neuromuscular control of the pelvis, hip, knee and ankle joints may contribute to maltracking of the patella, and should be addressed during the management of PFPS[13;14]. Further investigation into the role of neuromuscular control of femoral and tibial movements and their relative contribution to patellar maltracking is required.

2.7.2.5 Kinematics

It is postulated that altered kinematics of the lower limb may be one of the main contributing factors to PFPS. Any alteration in the kinematics of the lower limb, may result in the patella tracking through the intercondylar groove of the femur incorrectly. The changes in the kinematics seen in PFPS patients may be as a result of any of the factors already mentioned; a combination of them or may be due to compensatory mechanisms from the pain itself. Table 2.3 provides a summary of relevant experimental studies that assessed lower limb kinematics in participants with and without PFPS. The level of evidence (LOE) according to evidence based medicine criteria is noted[18].

Table 2.3 Summary of experimental studies that assessed lower limb kinematics in participants with and without PFPS.

Article	**Study Design**	**Kinematics analysed**	**Participants**	**Analysis performed**	**Results**
Salsich et al[63]	Cross sectional study (LOE: III)	Peak hip, knee and ankle flexion angles	10 participants (5 female; 5 male) in PFPS group and 10 (5 female; 5 male) matched control participants	6 cameras and force plate generated 3D kinematics and GRF while ascending and descending stairs	Reduced knee ext moment in PFPS group (p = 0.006)
Brindle et al[15]	Case controlled study (LOE: III)	Knee flexion angle and pelvis orientation in frontal plane at toe contact	16 participants (12 females; 4 males) in the PFPS group and 12 (7 females; 5 males) participants in the control group	6 cameras generated 3D kinematics while ascending and descending stairs	No statistically significant differences in kinematics between groups
Crossley et al[16]	Case controlled study (LOE: III)	Knee flexion at heel strike and peak knee flexion during stance phase	48 participants with PFPS and 18 asymptomatic control participants	Motion analysis of 3 lateral markers while doing a step up and down in time with a metronome	Decreased knee flexion in PFPS participants at heel strike (p = 0.03) and peak (p = 0.02) during stair descent and ascent

Table 2.3 Summary of experimental studies that assessed lower limb kinematics in participants with and without PFPS (continued).

Article	Study Design	Kinematics analysed	Participants	Analysis performed	Results
McClinton et al[57]	Case controlled study (LOE: III)	Knee flexion angle at foot-step contact	20 participants (9 female; 11 males) with PFPS and 20 healthy participants (10 females; 10 males)	8 cameras generated 3D kinematics while doing step ups at 5 different heights	PFPS participants had increased knee flexion at foot strike (p = 0.04)
Willson et al[17]	Controlled laboratory study (LOE: III)	Average hip and knee angles and angles at peak knee extension moment	20 females with PFPS and 20 age matched asymptomatic females	6 cameras and force plate generated kinematics and GRF while doing single leg squats. running and repetitive single leg jumps	No difference between tasks; PFPS had increased knee external rotation (p = 0.06); increased hip adduction (p = 0.01); decreased hip internal rotation (p = 0.01) in all three tasks
Bolgla et al[6]	Cross-sectional study (LOE: III)	Average hip internal rotation, hip adduction and knee varus joint angles during stance phase of stair descent	18 female participants with PFPS and 18 asymptomatic females in the control group	7 camera video analysis while doing step up, and down	Kinematics were not significantly different

Table 2.3 Summary of experimental studies that assessed lower limb kinematics in participants with and without PFPS (continued).

Article	Study Design	Kinematics analysed	Participants	Analysis performed	Results
Souza et al[42]	Cross sectional study (LOE: III)	Average hip internal rotation during the first 50% of stance phase	19 females with PFPS and 19 matched uninjured controls	3D motion analysis during running at a selected speed	Significantly greater average hip internal rotation in PFPS group ($p < 0.001$)
Dierks et al[11]	Cross-sectional study (LOE: III)	Peak knee adduction; peak hip adduction; peak hip internal rotation	20 runners with PFPS (5 male and 15 female) and 20 asymptomatic runners (5 male and 15 female); running defined as more than 15km per week	6 camera 3D kinematic analysis at the beginning and end of a prolonged run.	PFPS had an increased hip adduction angle ($p = 0.045$)
Willson et al[55]	Controlled laboratory study (LOE: III)	Contra-lateral pelvic drop and hip and knee angles at peak knee extension moment	20 females with PFPS and 20 matched healthy female controls	6 camera 3D kinematic analysis and force plate while doing single leg jumps; before and after exertional jumps till 17/20 fatigue	Increased contra-lateral pelvic drop ($p = 0.003$) in PFPS participants after exertion and generally increased hip adduction ($p = 0.02$), flexion ($p = 0.05$) and internal rotation ($p = 0.02$) angles in PFPS

Table 2.3 Summary of experimental studies that assessed lower limb kinematics in participants with and without PFPS (continued).

Article	Study Design	Kinematics analysed	Participants	Analysis performed	Results
Grenholm et al[64]	Controlled laboratory study (LOE: III)	Hip adduction, knee flexion and ankle dorsi- and plantar flexion at foot contact of stance and swing legs	17 females with PFPS (for longer than 1 year) and 17 matched controls	5 camera 3D kinematic analysis during stair descent	Minor differences between the groups including a lower knee angular velocity ($p = 0.01$) and greater plantar flexion in swing phase in PFPS participants ($p = 0.04$)
Souza et al[8]	Cross sectional study (LOE: III)	Peak hip internal rotation and adduction during stance phase	21 females with PFPS and 20 matched females in the control group	3D kinematics while running, doing a drop jump and step down	Increased peak internal rotation ($p < 0.001$)
McKenzie et al[65]	Cross sectional case controlled study (LOE: III)	Knee flexion; hip flexion, adduction and internal rotation at foot strike	10 recreational female athletes with PFPS and 10 uninjured healthy females in control group	3D kinematics while ascending and descending three steps at 2 different speeds	Greater knee flexion ($p < 0.001$); greater hip adduction ($p < 0.001$) and greater hip internal rotation ($p < 0.001$)

Table 2.3 Summary of experimental studies that assessed lower limb kinematics in participants with and without PFPS (continued).

Article	Study Design	Kinematics analysed	Participants	Analysis performed	Results
Boling et al[50]	Cohort study (LOE: I)	Peak hip flexion, adduction and internal rotation angle and peak knee flexion, valgus and internal rotation angle during stance phase	1597 US navy recruited; 40 developed PFPS (24 female and 16 male)	Prospective study using electromagnetic tracking sensors for kinematics during a jump landing	Decreased knee flexion (p = 0.02) and increased hip internal rotation (p = 0.04) during jump landing were identified as possible risk factors for the development of PFPS
Hetsroni et al[66]	Prospective study (LOE: I)	Bilateral peak foot pronation during stance, pronation range of motion, time to peak pronation from heel strike	473 infantry; 61 developed PFPS	Prospective study using 2D kinematics while barefoot walking	No significant association between pronation and PFPS; Significant association between PFPS and pronation velocity (p=0.007)

Table 2.3 Summary of experimental studies that assessed lower limb kinematics in participants with and without PFPS (continued).

Article	Study Design	Kinematics analysed	Participants	Analysis performed	Results
Thijs et al[49]	Prospective cohort study (LOE: I)	Peak pressure data; mediolateral pressure distribution in foot; displacement of centre of pressure	84 military recruits; 36 developed PFPS (25 male and 19 female)	Prospective study using plantar pressure measurements while barefoot walking	More laterally directed pressure at foot strike (p = 0.003); shorter time to maximal pressure on 4th metatarsal (p = 0.01); slower maximal velocity of centre of pressure during forefoot contact (p = 0.002).
Thijs et al[67]	Prospective cohort study (LOE: I)	Peak pressure data; mediolateral pressure distribution in foot; displacement of centre of pressure	102 (89 females; 13 males) novice recreational runners; 17 (16 females; 1 male) developed PFPS	Prospective study using standing foot posture index and foot scan while standing and barefoot running	Higher vertical peak under lateral heel (p = 0.04) and metatarsals 2 (p = 0.02) and 3 (p = 0.03); No association between pronated or supinated feet

Table 2.3 Summary of experimental studies that assessed lower limb kinematics in participants with and without PFPS (continued).

Article	Study Design	Kinematics analysed	Participants	Analysis performed	Results
Levinger et al[68]	Cross sectional comparative study (LOE: III)	Peak rear foot inversion/eversion; dorsiflexion/plantarflexion; abduction/adduction; peak tibial internal/external transverse rotation and the time to all peaks	13 female participants with PFPS and 14 healthy matched female controls	3D kinematics and force plate while barefoot walking	Delayed peak rear foot eversion ($p=0.02$); earlier peak dorsiflexion ($p=0.02$); lower peak medial GRF ($p=0.03$); minimum vertical GRF trough ($p=0.02$) and second vertical peak GRF ($p=0.01$).

Eight of the 11 studies assessed lower limb kinematics during stair ascent and descent[6;8;15;16;57;63-65]; four studies examined lower limb kinematics during a single legged jump[8;17;50;55]; three studies assessed lower limb kinematics during running[8;11;17]; and one study examined lower limb kinematics during a squat[17]. Table 2.4 and 2.5 provide a summary of the changes in lower limb kinematics that have been identified in participants with PFPS. It is evident that there is little agreement between studies in changes in lower limb kinematics in participants with PFPS. This section will examine the different kinematic findings in more detail.

Table 2.4 Summary of main kinematic findings in the lower limb excluding the ankle and foot in reviewed literature.

Main lower limb kinematic findings in participants with PFPS
No differences between PFPS and control groups[15;6]
Increased pelvic contra-lateral drop[55]
Increased hip flexion[55]
Increased hip adduction[11;55;65]
Increased hip internal rotation[50;65]
Decreased hip internal rotation[17;55]
Increased hip external rotation[17]
Decreased knee flexion[16;50]
Increased knee flexion[57;65]
Increased ankle plantar flexion during swing phase[64]

Table 2.5: Summary of main kinematic findings in the ankle and foot in reviewed literature.

Changes in foot and ankle kinematics in participants with PFPS [2;49;66-68]
Increased pronation velocity[58]
Increased laterally directed pressure at foot strike[59]
Decreased time to maximal pressure on 4th metatarsal[60]
Delayed peak rear foot eversion[61]

(A) Increased pelvic contra-lateral drop[55]

Willson et al[55] assessed participants before and after single legged jumps. There was a significant difference in the amount of contra-lateral pelvic drop after the jumps. It was postulated that the hip musculature was unable to prevent excessive contralateral drop when fatigued, potentially leading to increased hip adduction, altered knee biomechanics, an increased Q angle and a decrease in the flexibility of the lateral structures (for example, tensor fascia lata, gluteus maximus and ITB) of the knee.

(B) Increased hip flexion[55]

Willson et al[55] also found an increase in hip flexion at maximal knee flexion in participants with PFPS. This is when the quadriceps would exert their maximal posterior force on the patella. Willson et al explained this as a compensatory mechanism used during landing to move the body weight forwards, thus changing the centre of gravity to over the knee joint centre. This moves the load from the knee extensors to the hip extensors during upward movement, creating less posterior directed force on the patella, and potentially reducing loading of the PFJ. This may be a compensatory kinematic change to minimise the pain associated with PFPS.

(C) Increased hip adduction[11;17;55]

Three studies showed increased hip adduction in participants with PFPS[11;17;55]. Two of the studies observed running kinematics[11;55]. All three studies investigated the effects of either fatigue or greater loaded tasks on kinematics in participants with PFPS. The increase in adduction may be explained by decreased hip abduction strength in participants with PFPS. Increased adduction ROM may contribute to the development of PFPS through associated increased Q angles, and increased tightness of the lateral structures of the knee.

(D) Increased hip internal rotation[8;17;50;55]

Increased hip internal rotation was found in two studies[8;50], that examined kinematics during a jump landing task[50] and running, jump landing and step down tasks[8] respectively. The increased hip internal rotation ROM was theorised to be related to weakness of the hip external rotators. However, increased hip external rotation strength has also been described in participants with PFPS as discussed in section 2.7.2.3 (D), page 20.

(E) Decreased hip internal rotation[17;55]

Two studies[17;55] showed a decrease in hip internal rotation range of motion in participants with PFPS. This was explained as a compensatory action to try decrease the rotational load on the knee which exhibited increase external rotation ROM. There is equivocal evidence for changes in hip rotation kinematics in participants with PFPS, and this needs to be investigated further.

(F) Increased knee external rotation[17]

Willson et al[17] noted an increase in knee external rotation during three progressively demanding tasks of squatting, running and single leg jump landing in participants with PFPS. Increased knee external rotation ROM has been shown to significantly increase retro-patellar stress, particularly on the lateral facet and may also increase the Q angle, which may contribute to the development of PFPS.

(G) Increased and decreased knee flexion[16;50;57]

Once again there was a discrepancy in some of the research results. Crossley et al[16] and Boling et al[50] observed a decrease in knee flexion ROM. Crossley et al[16] found that during stair ascent and descent participants with PFPS had decreased peak knee flexion during stance phase and at foot strike. Their explanation for this finding was that PFPS sufferers would try decrease the amount of patellofemoral joint reaction forces by reducing the amount of knee flexion during activities. Boling et al[50] conducted a prospective study and found that a significant amount of participants that developed PFPS had decreased knee flexion during a jump landing task prior to the onset of symptoms. Their explanation of this was done in conjunction with their finding that another risk factor was a decrease in quadriceps muscle strength. They speculated that due to a decrease in quadriceps strength, participants would land with less knee extension in order to minimise the ground reaction force on landing[50].

In contrast, McClinton et al[57] found that during ascending steps, PFPS sufferers had more increased knee flexion than control participants. The authors hypothesised that this was possibly due to a few factors, including, a loss of knee joint position sense, altered weight distribution to reduce forces on the PFJ, and a compensatory change in the centre of gravity.

(H) Increased ankle plantarflexion during swing phase[64]

Grenholm et al[64] found that the ankle joint had an increase in plantarflexion during the swing phase of stair ascent and descent. They felt that this was another mechanism of decreasing the load on the stance phase leg before landing, and therefore decreasing the knee joint reaction forces.

(I) Changes in foot kinematics[2;49;66-68]

Foot biomechanics are thought to be a contributing factor to PFPS because of its effect on tibial movement. If the tibia rotates, the Q angle will change due to the attachment of the patella tendon to the tibia. Internally rotating the tibia will move the tibial tuberosity medially and thus decrease the Q angle and externally rotating the tibia will move the tibial tuberosity laterally and thus increase the Q angle[14;19;57]. This change in the Q angle may predispose an individual to PFPS.

Tibial rotation is coupled to subtalar joint movement[69]. Pronation of the subtalar joint is coupled with internal rotation of the tibia and may therefore be associated with a decrease in the Q angle[14]. The subtalar joint supination is coupled with tibial external rotation and thus an increase in the Q angle[14]. This presents a conundrum. If an increased Q-angle is presumed to cause maltracking and thus PFPS, then supination, rather than pronation should cause PFPS. But foot pronation has previously been proposed as a predisposing factor in the development of PFPS[14].

A possible explanation for this discrepancy has been offered by Tiberio[14]. His explanation postulates that to obtain extension of the knee in midstance, the tibia must rotate externally in relation to the femur. This enables the knee to obtain the screw home mechanism needed for adequate motion. On a pronated foot however, the tibia is internally rotated. This means the femur must internally rotate even further on the tibia. This will then place the tibia in relative external rotation and result in an increase in the Q angle, increased lateral force acting on the PFJ and possible maltracking of the patella. However, to date there are no kinematic studies that support this theory and further evidence is required to understand the relationship between foot kinematics and PFPS..

Powers[14] states that there is no cause and effect relationship between pronation and PFPS and in a study by Dierks, T. et al[11] they found no significant association between arch height in PFPS participants and control participants, before and after a prolonged run.

In two prospective studies done by Thijs et al[67] on military cadets and by Thijs et al[49] on novice runners, pronation was not shown to cause PFPS. In the study on military cadets the participants that developed PFPS were shown to have had significantly more laterally directed pressure distribution on heel strike, a shorter time to maximal pressure on the fourth metatarsal and a slower maximal velocity of the change in lateromedial direction of the centre of pressure during the forefoot contact phase. In the study on novice runners they found that runners with PFPS exerted a higher vertical peak force under the lateral heel, the 2nd and the 3rd metatarsals. There were no significant relationships between either a pronated foot or supinated foot and PFPS. Another study looked at midfoot mobility in participants with PFPS[19].

They found an association between PFPS and midfoot mobility when a participant moved from non weight bearing; or from weight bearing subtalar joint neutral, to a static relaxed stance.

From the kinematic studies reviewed it seems that the most consistent kinematic changes occurring in individuals with PFPS are found around the hip and knee joints. The changes previously thought to be causative in the ankle and feet are less consistent and it is certainly not due to rear foot pronation as previously hypothesized.

2.7.2.6 Kinematics of PFPS in running

Of interest and relevant to the study in Chapter 3, is the kinematic data found in those studies reviewed, that examined running in particular. It is also of interest to analyse which kinematic parameters were assessed during various stages of the running gait cycle. The studies by Souza et al[8] and Dierks et al[11] investigated peak hip abduction/adduction and peak hip rotation angles during stance phase, while the study by Willson et al[17] investigated hip abduction/adduction and rotation angles at the peak knee extension moment (KEM) and the excursion of those angles from foot strike to peak KEM. Peak KEM was chosen as this is where the quadriceps reaction force is likely to be the greatest. None of the running studies assessed angles at foot strike, toe off or swing phase during running. The main findings of these studies are summarised below in Table 2.6.

Table 2.6 Lower limb kinematics of interest in running related studies.

Lower limb kinematic parameters	**Findings in PFPS group**		
	Souza et al[8]	Dierks et al[11]	Willson et al[17]
Peak knee adduction during stance	-	↔	-
Peak hip adduction during stance	↔	↑	-
Peak hip internal rotation during stance	↑	↔	-
Hip internal rotation at peak knee extension moment (KEM)	-	-	↓
Hip adduction at peak KEM	-	-	↑
Knee internal rotation at peak KEM	-	-	↓
Hip internal rotation excursion from foot strike to peak knee extension moment (KEM)	-	-	↑
Hip adduction rotation excursion from foot strike to peak KEM	-	-	↑
Knee internal rotation excursion from foot strike to KEM	-	-	↓

↓	decreased
↑	increased
↔	unchanged
-	not assessed

2.8 Summary of the literature review

Patellofemoral pain syndrome is the most common musculoskeletal complaint in runners[1;6;7]. Despite this, the pathology and pathomechanics are still not clearly understood[8]. The patella functions to improve the mechanics of the knee joint by increasing the length of the lever arm for the quadriceps[8;25]. This improves the amount of torque the quadriceps can generate during knee extension. And as such, the amount of joint reaction force between the articular surfaces of the patella and femur are high. For this reason the mechanics of the patella over the femur during knee flexion and extension need to be mechanically correct in order to properly absorb these forces[8;26]. Changes in these mechanics can change the distribution of these forces, cause disruption and irritation of structures around the patellofemoral joint and have thus been implicated in PFPS[27].

It has been shown that there are a variety of biomechanical differences found in PFPS sufferers during a variety of activities[6;8;12;17;42;55;57;63;64]. These include anatomical differences, flexibility, and strength, neuromuscular and kinematic differences. The changes in kinematics could be seen as the end result of the other factors. That is, changes in anatomy, strength, flexibility and neuromuscular activity result in a change in kinematics. A variety of studies, either level I or III LOE, have shown these kinematic differences during a variety of tasks[6;12;15;16;42;55;57;63;64]. Of all the studies that were reviewed only three studies (all level III LOE) investigated the changes in kinematics during running[6;8;11;17]. As PFPS is the most common musculoskeletal condition in running it is felt that running needs to be investigated further. The running gait cycle is a very complex movement pattern. It is therefore necessary to investigate the differences in running kinematics at various stages of the running gait cycle in PFPS.

There are many biomechanical factors to consider when assessing and treating PFPS. They may be causative or compensatory. Both local patellofemoral joint biomechanics and biomechanics of the entire lower limb seem to be involved. More research is needed to better diagnose the cause, effect and recommended treatment of this condition. The following study aims to investigate the kinematics during the running gait cycle, in active people with PFPS.

CHAPTER 3: An investigation of potential kinetic factors associated with patellofemoral pain syndrome during running

3.1 Introduction

Patellofemoral pain is a musculoskeletal disorder involving the lower limb, presenting as retropatellar and/or peripatellar pain[1]. It is considered to be the most common injury found in active individuals and the most common lower limb musculoskeletal complaint[1;6;10]. Short term treatment of patellofemoral pain is often successful, but the long term outcomes are less successful, with 80% of sufferers' still experiencing pain 5 years later and 74% of individuals having to reduce their activity level[6]. These poor long term results are thought to be due to a lack of understanding of the aetiological factors in PFPS[8].

The aetiology of PFPS is thought to be multifactorial, but these factors are poorly understood[19]. A better understanding of the causative factors and the possible treatment of them is necessary to better manage PFPS. A commonly accepted hypothesis regarding PFPS aetiology is one of abnormal patella tracking[11;12]. It has been shown that this mal-tracking is affected by local factors, as well as factors proximally or distally[19]. Local factors include those occurring around the knee and PF joint, while proximal factors include those occurring proximal to the knee, for instance at the hip joint and distal factors occur distal to the knee joint, for instance at the foot or ankle. The quadriceps angle (Q-angle) is often discussed in relation to the local, proximal and distal factors, as each factor will have an influence on the Q angle[14]. It has been proposed that a greater Q angle results in a larger lateral force on the patella and therefore predisposes a person to PFPS.

It has been suggested that there are 5 ways in which the Q angle of a patient's knee can be increased[14]. These are contralateral pelvis tilt, increased hip adduction, increased hip internal rotation, increased knee valgus and increased tibial external rotation.

Research that has been done previously to investigate the kinetic changes that occur in PFPS have shown some of these to be valid. Wilson et al[55] found that in runners with patellofemoral pain, there was an increase in the injured groups contralateral pelvic drop and that this was more significant after exertion. Willson et al[55], Dierks, et al[11] and Willson, et al[17] all found an increase in hip adduction in PFPS patients and Willson[55], Souza[42], and Boling et al[50] found an increase in hip internal rotation during a variety of tasks in PFPS participants.

The majority of studies have identified the kinematic changes during stepping or jumping tasks[16;50;55;64;65]. There is limited evidence for kinematic changes during running tasks[8;11;17]. Further, the studies that have investigated running kinematics in participants with PFPS have only examined selected lower limb kinematic variables at peak knee extension moment, and the excursion of angles from foot strike to peak knee extension moment[8;11;17]. Therefore there is a lack of evidence for comprehensive stance phase and swing phase kinematics during the running gait cycle.

Accordingly, the aim of this dissertation was to investigate potential lower limb kinematic factors associated with PFPS during running. The specific objectives of this dissertation have been described in section 1.2.2, page 3.

3.2 Methodology

3.2.1 Study design

The study had a descriptive cross-sectional study design.

3.2.2. Selection of participants

Male and female participants were recruited for the study through advertisements placed at sports medicine and physiotherapy practices, and running clubs in Cape Town, South Africa. Participants included physically active males and females, between 20 and 46 years of age.

Participants were required to be engaging in physical training that consisted of running, cycling or gym training for a minimum of two hours per week for the three-month period preceding the study. Participants were also required to have a Q-angle within the normal range for males (between 8.2º and 14.2º) and females (between 11.3 º and 20.3º) respectively[29].

Participants in the PFPS group were required to have a history of unilateral PFPS that did not exceed a six-month period prior to testing. The diagnosis of PFPS was based on the following criteria:

- A history of anterior or retro-patellar pain of non-traumatic origin[6;63]
- Pain during one or more symptom provocation tests: resisted terminal knee extension, stair descent, or a unilateral partial squat.[6;63]
- Ability to run without pain for a minimum period of 10 minutes. This was important for the running test to be completed without reproducing symptoms of PFPS, and reduced the risk of compensatory gait patterns that may have occurred from the presence of pain during testing.

Potential participants were excluded from the study if they reported any of the following:

- A lower limb or lumbar spine injury or pathology that required treatment or rehabilitation in the six-month period prior to testing.
- Previous lower limb surgery.
- Use of any non-steroidal anti-inflammatory drugs or analgesics in the 12 weeks prior to testing.

Participants in the PFPS and control groups were matched for to age, body mass, stature, body mass index, sum of skin folds, percentage body fat, lean body mass, lean thigh volume and Q-angle.

Potential participants were informed about the purpose of the study, the testing to be undertaken, the possible risks relating to the study, and their right to withdraw from the study at any stage. All participants were required to complete the informed consent form prior to the commencement of testing (Appendix 1). Fifteen participants with unilateral PFPS formed the PFPS group, and the control group consisted of 15 participants without PFPS.

3.2.3 Sample size determination

Data from a previous study that investigated biomechanical variables associated with PFPS in runners were used to ensure that the sample size would provide sufficient statistical power[8]. The peak hip adduction angle during a running trial of the chosen study was selected to determine the required sample size, as it is one of the main outcome measures of this study. The required sample size for peak hip adduction angle was calculated using a smallest meaningful difference of 9º, and a standard deviation of 5º. With statistical significance accepted as $p < 0.05$, groups of 11, 14, and 17 participants provided 80%, 90% and 95% statistical power for the peak hip adduction angle. Therefore, 15 participants were required in each of the PFPS and control groups to ensure sufficient statistical power.

3.3 Testing procedure

The testing phase of the study consisted of one visit to the laboratory. Prior to the visit, an informed consent form (Appendix 1) as well as general health and training questionnaires (Appendices 2 and 3) were emailed to participants. The participants were requested to bring the completed documentation to the laboratory visit.

3.3.1 Informed consent and questionnaires

Participants were required to complete an informed consent form prior to testing (Appendix 1). This form was emailed to the participants along with an information sheet explaining in detail all testing procedures.

They were also required to complete a general health questionnaire, to determine a general health and training history (Appendix 2 and 3). This included information regarding medication, medical and surgical history, musculoskeletal injuries or pathology, and training and racing history. On arrival at the laboratory the completed documentation was checked for inclusion and exclusion criteria.

3.3.2 Familiarisation

All participants underwent a familiarization session prior to the anthropometric measurements and the running test. The participants were familiarized with the laboratory equipment and testing protocols that would be used during the running test. This was to ensure that everything explained in the informed consent was understood and also to minimise any error associated with participants performing unaccustomed exercise.

3.3.3 Anthropometry

Body mass (kg) was recorded using a calibrated scale (Seca model, 708 Germany). Stature (cm) was recorded using a stadiometer (Seca model, 708 Germany). Body fat was expressed as the sum of seven skinfolds (biceps, triceps, subscapular, suprailiac, calf, thigh and abdomen)[70]. Body fat was also expressed as a percentage of body mass. Lean thigh volume was calculated according to the method adapted from Katch and Katch[71], which assumes that the upper lower limb has the shape of a truncated cone (Appendix 4).

3.3.3 Quadriceps angle

Each participant had their Q-angle measured according to previous methods described [17;55]. The measurement was performed with participants in supine, with their knee placed on a small roll, sized so that the knee was in neutral when the Q-angle was measured, and their toes facing directly upwards. A goniometer was used to measure the angle obtained by drawing a line from the anterior superior iliac spine to the mid patella and from the mid patella to the tibial tuberosity[29]. The Q-angle of the affected leg was assessed in the PFPS group and the Q-angle of the right leg was assessed in the control group.

Participants were required to have a Q-angle within the normal range for males (between 8.2º and 14.2º) and females (between 11.3 º and 20.3º) respectively[29].

3.3.4 Patellofemoral pain screening tests

The screening tests for PFPS were then performed for participants with PFPS. These tests were selected from previously validated tests for PFPS[8;57;63]. Patellofemoral pain syndrome was determined by the presence of retropatellar pain reproduced on one of the following tests[6;63]: resisted terminal knee extension performed in supine with the examiner resisting extension from 10° flexion to 0°; stair descent performed down three steps in the laboratory and a unilateral partial squat performed on the symptomatic leg with the participant holding onto a treatment table. Patellofemoral pain measurements were assessed using a visual analogue scale (VAS). Participants were required to have retropatellar pain of three or more on the VAS scale during one or more of the screening tests to confirm the diagnosis of PFPS.

3.3.5 Patellofemoral pain measurements

Patellofemoral pain was measured during the PFPS screening tests, and before and after the running test in participants with PFPS. Participants were required to rate the PFPS according to a "*rating of perceived pain*" on a VAS of 0 to 10, where 0 represented "*no pain*", and 10 represented "*maximal pain*". It has previously been shown that this method of measurement of pain is highly correlated with objective pain measures[5]. Participants were required to have a pain score of zero at rest to participate in the running test. Participants were excluded from testing if the difference between pre-test and post-test pain scores was greater or equal to three points, to eliminate any potential biomechanical changes that may have been influenced by pain inhibition[72].

3.3.6 Running test

After performing the body composition measurements and screening tests, 16 retro-reflective markers were placed on the anatomical sites described in the modified Helen Hayes marker set for dynamic gait analysis[17;55;61].

The key anatomical points were: the lateral malleoli, heels, the second metatarsal heads, lateral aspect of the mid-tibia, lateral femoral condyle in line with the knee joint rotation axis, lateral aspect of the mid-thigh, anterior superior iliac spines, and posterior superior iliac spines (PSIS). The mid-tibial and mid-thigh markers were deliberately placed asymmetrically on the left and right (Figure 3.1) to allow for computerised labelling of the markers. The markers were fixed into place with both double sided tape and transpore tape to ensure no movement during testing.

Figure 3.1 Anterior view of participant with modified Helen Hayes marker set. Markers on both heels and the posterior superior iliac spines are not seen.

Participants performed a standardized warm-up before the running test. The warm-up consisted of five minutes of running on a treadmill at a self-selected comfortable pace. The participants then performed the running test, which consisted of running at a similar self-selected speed as performed on the treadmill for the warm up, on the 10 m pathway of the gait laboratory wearing their normal running shoes. Kinematic data were recorded during each repetition of the test.

An eight-camera motion analysis system (Oxford Metrics Vicon System 370 Version 2.5, Oxford metrics Ltd, Oxford, United Kingdom) was used to collect kinematic data at 250 Hz as the participants ran across an Advanced Mechanical Technology, Inc. (AMTI®Newton, MA, USA) force plate (1000Hz) situated underneath the surface of the 10 m pathway.

Ground reaction force data were collected with the force plate, which was hidden on the floor. Participants in the PFPS group were required to make contact with the force plate with their affected foot, whereas control participants were required to make contact with their right foot. The participants were unaware of the force plate and the need for them to land on it during the running test. The investigator performed a visual assessment to determine the correct foot strike was achieved, and that there were no adjustments in running gait to ensure contact with the force plate. A trial was considered valid if the participant's entire foot made contact with the force plate, from heel strike to toe off, and there was no visual alteration in the running gait pattern compared to their warm up run on the treadmill and practice runs across the test pathway. Ten valid trials were required for data analysis. Patellofemoral pain was assessed at the end of each repetition of the test using the VAS.

3.3.7 Data analysis

Ten valid trials were selected for all participants. In one participant only five valid running trials were recorded due to time constraints, therefore only five trials were used for this participant. A valid trial was a trial with no missing markers, and where the participant had landed on the force plate with their entire foot with a normal gait stride. The data was processed from the time when the participant entered the capture volume of the Vicon cameras until the participant exited the capture volume. In most instances, this included a foot strike of one foot, followed by a full clean strike of the next foot on the force plate, followed by foot strike of the first foot again.

Data when the participant was entering and exiting the capture volume was unreliable, and consequently only the force plate was used to determine the exact moment of heel strike and toe off.

Therefore stance phase was defined as the time from and including heel strike, through to the frame before toe off. Swing phase was defined as all data not during stance phase.

Kinetic and kinematic data were processed using the Vicon Nexus software 1.6.1 (2009 version) (Oxford Metrics Vicon System 370 Version 2.5,Oxford metrics Ltd,Oxford,United Kingdom). The data were reconstructed and labeled automatically. All unlabeled markers were then deleted, gaps were filled using the built in Woltring filter, the plugin gait dynamic gait model was run and the final model outputs, marker positions and force plate data were exported to csv format.

The data were then imported and analyzed by a custom written program in MATLAB (The Mathworks Inc. (2011b)). The timing of the foot strike was defined as the time when the magnitude of the force (force plate data) increased more than one standard deviation of the baseline noise above zero for at least 20 consecutive samples. The timing of the toe off was defined as the first data point after foot strike when the magnitude of the force (derived from the force plate data) decreased to within one standard deviation of the baseline noise of zero. A cubic spline was then applied to the angle data to allow the data points to more accurately be interpolated to the times of heel strike and toe off as the force plate was operating at a higher sample rate. This also allowed for appropriate comparison of timing between participants running at slightly different speeds. Running speed was determined as the average speed of the PSIS markers from one meter prior to heel strike, to one meter after heel strike.

All angle data was represented as X, Y and Z referring to flexion/extension, abduction/adduction (knee adduction/abduction movements are demonstrated in figure 3.2) and internal/external rotation respectively. For each model output the data were extracted at heel strike, toe off, peak during swing and peak during stance. In addition the percentage of stance phase taken to reach the peak range of motion during stance phase, was calculated. The data were then exported to Microsoft Excel 2007 and analysed using Statistica software (StatSoft, Inc. (2011) STATISTICA (Data analysis software system), (Version 10. www.statsoft.com) (Appendix 5).

Figure 3.2: Measurement of knee adduction/abduction relative to anatomical 0° (indicated by solid black line).

The kinematic variables assessed in this study are described in Table 3.1. The kinematic variables were assessed at foot strike, peak stance phase, toe off and peak swing phase. Range of motion of each variable was also assessed during stance phase and swing phase and the percentage of stance phase to reach peak for each variable was also calculated.

Table 3.1 Kinematic variables that were included in the analyses

Variable	**Direction**
Hip Flexion / Extension	+ Flexion - Extension
Hip Abduction / Adduction	+ Adduction - Abduction
Hip Internal / External Rotation	+ Internal Rotation - External Rotation
Knee Flexion / Extension	+ Flexion - Extension
Knee Adduction (varus) / Abduction (valgus) /	+ Adduction - Abduction
Tibial External / Internal Rotation	+ Tibial External Rotation - Tibial Internal Rotation
Ankle Plantarflexion / Dorsiflexion	+ Plantarflexion - Dorsiflexion
Pelvic tilt (Coronal plane)	+ Contralateral Up - Contralateral Down
Pelvic tilt (Saggital plane) Anterior / Posterior	+ Anterior Tilt - Posterior Tilt
Pelvic Rotation (Transverse plane)	+ Ipsilateral Forward - Contralateral Forward

+ indicates movement in a positive direction; - indicates movement in a negative direction

3.3.8 Statistical analyses

Statistical analyses were performed using Statistica software (StatSoft, Inc. (2011) STATISTICA (Data analysis software system), (version 10. www.statsoft.com). Descriptive statistics were performed. Normality was assessed using the Shapiro-Wilkes test. An independent *t*-test was used to determine differences in descriptive variables and kinematic data between the PFPS and control groups (Appendix 5). Chi-squared tests were used to determine differences in training characteristics between groups. Statistical significance was accepted as $p < 0.05$. All data are presented as the mean ± standard deviation. Additional figures for analyses are presented in Appendix 10.

Logistical regression analyses were performed using EPI-INFO 7 (2002) software (Appendix 6). Joint angles at foot strike, peak joint angles during stance phase and the percentage of stance phase to peak joint angles for the hip, knee and ankle joints were coded as painful (1) or control (0) variables. Coding was performed based on the PFPS group mean value for each variable. If the mean value being assessed was higher for the PFPS group than control group, all joint ranges equal to or higher than the PFPS group mean, were coded 1 (pain group) and all ranges lower than the mean were coded 0 (control group). If the mean value being assessed was lower for the PFPS group than control group, all joint ranges equal to or lower than the PFPS groups mean, were coded 1 (pain group) and all ranges higher than the mean were coded 0 (control group). The logistic regressions were performed on this recoded data to determine whether or not kinematic variables were associated with an increased risk of PFPS.

3.3.9 Ethical considerations

The study was performed in accordance with the principles of the Declaration of Helsinki (Seoul, 2008). The study was granted ethical approval by the Faculty of Health Sciences Human Research Ethics Committee of the University of Cape Town (HREC/REF 361/2009) (Appendix 7). Participants were initially emailed an information sheet that described the purpose of the study, the testing to be undertaken, the possible risks relating to the trial, and their right to withdraw from the study at any stage.

Participants were provided with full, adequate and understandable written explanations of the testing procedures, including all possible risks involved in this study. On the day of testing the researcher verbally explained the purpose of the study, the testing to be undertaken, the possible risks relating to the trial, and their right to withdraw from the study. The researcher also showed the participant all equipment that was to be used. Participants had an opportunity to ask questions, and have any queries or concerns regarding the study addressed. All participants were required to provide the written informed consent (Appendix 1) before taking part in the study. All data were kept confidential.

3.3.10 Risks to participants

3.3.10.1 Anthropometry and patellofemoral pain measurement

There were no potential risks to the participants associated with mass, stature, skinfold measurements, or the measurement of patellofemoral pain.

3.3.10.2 Running tests

Running is associated with a minor risk of the participants sustaining a musculoskeletal injury or falling. In this study, the participants underwent a thorough familiarization process. All due care was taken to ensure that participants were both familiar and confident with running along the pathway and across the force plate. Participants were excluded from the study on the basis of medical or surgical history, any musculoskeletal injury, and any medication use or viral infection within the 12 weeks preceding the study. All participants were required to warm up before the running tests, and the warm-up was monitored to reduce the risk of any musculoskeletal injury that may be associated with running. Participants in the PFPS group were monitored with the Visual Analogue Scale (VAS) after each repetition of the running test. Participants were also excluded from further testing if the difference between pre-test and post-test pain scores was greater than or equal to three points. Participants were advised on appropriate treatment for PFPS (Appendix 8), and were referred for physiotherapy management of PFPS.

3.3.10.3 Kinematic data capturing

The use of an eight-camera motion analysis system and a force plate for the measurement of kinematic and kinetic data provided no additional potential risks to the participants.

3.3.11 Benefits to participants

Participants received a detailed pamphlet containing information about PFPS and evidence-based prevention and treatment options. Participants were given individual feedback regarding their general health screening and anthropometric measurements (Appendix 9). The individual results included information regarding body composition measurements, general health screen, kinetic and kinematic data. On completion of the study, the summarized results and recommendations of the study were formally presented to all participants.

3.4 Results

3.4.1 Participants

Thirty-three participants were recruited for this study. Sixteen uninjured participants (eight males and eight females) formed the control group. Initially, 17 participants with anterior knee pain were recruited to the PFPS group. However, one participant presented with iliotibial band friction syndrome and was excluded at the initial evaluation. Another participant was excluded with a Q angle of 33º as this exceeded the normal range for female participants, which was stipulated in the inclusion criteria as being between 11.3º and 20.4º (Section 3.2.2; page 45).

Therefore, 15 participants (eight males and seven females) formed the PFPS group; and a total of 31 participants (16 males and 15 females) completed the running trials. The study sample is summarized in Figure 3.3.

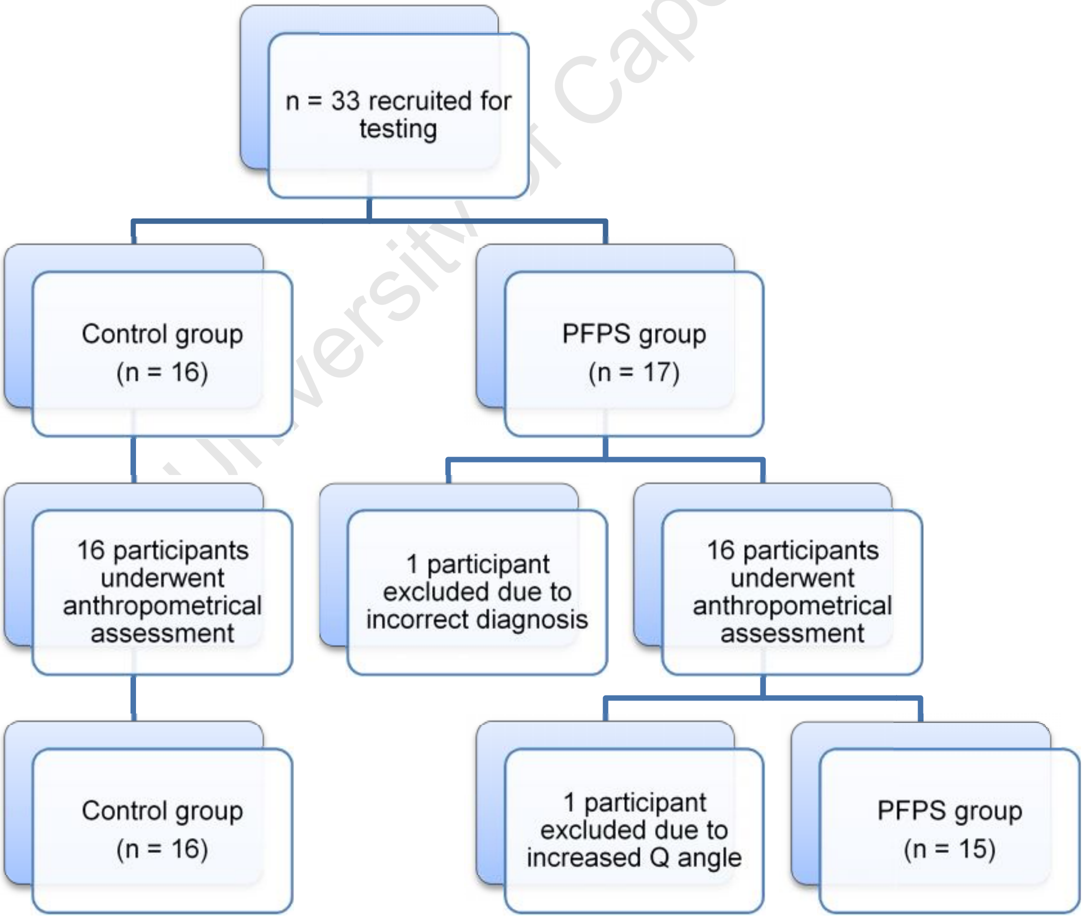

Figure 3.3 Summary of study sample.

3.4.2 Screening tests

The results of the PFPS screening tests for participants in the PFPS group are summarized in Table 3.2. The majority of participants in the PFPS group experienced pain during stair descent and a partial squat. All PFPS screening tests were negative for participants in the control group.

Table 3.2 Summary of results from the PFPS screening tests for participants in the PFPS group (n = 15).

Screening test	Positive (n)	Negative (n)
Resisted terminal knee extension	4	11
Stair descent	11	4
Partial squat	11	4

Participants in the PFPS group had knee pain for an average duration of 16.2 weeks, with a minimum duration of three weeks and a maximum of 52 weeks. Ten participants in the PFPS group had right knee pain, and five participants in the PFPS group had left knee pain. All participants had a PFPS pain score of "*zero*" at rest, and prior to testing. The participants were continually monitored for changes in their pain score during the testing procedure. There were no changes in the pain scores throughout the testing procedure.

3.4.3 Descriptive characteristics

The descriptive characteristics of participants are shown in Table 3.3. There were no significant differences between groups for any of these variables.

Table 3.3 Descriptive characteristics of participants in the PFPS group (n = 15) and control (n = 16) groups. Data are expressed as mean ± standard deviation (SD).

Variables	**Control (n=16)**	**PFPS (n=15)**
Age (years)	36.4 ± 5.5	34.2 ± 7
Mass (kg)	71.6 ± 11.6	70.9 ± 17.1
Stature (m)	1.8 ± 0.1	1.7 ± 0.1
Sum of skinfolds (mm)	91.3 ± 29.4	112 ± 40.4
Body fat (%)	23.3 ± 6	24.8 ± 5
Lean body mass (kg)	55.3 ± 13.5	53.3 ± 13.5
Lean thigh volume (cc)	5378 ± 1376	4762 ± 1012
Q angle (degrees)	14.5 ± 2.9	14.8 ± 3.4

The training characteristics of participants are shown in Table 3.4. There were no significant differences in training characteristics between groups.

***Table 3.4 Training characteristics of participants in the PFPS (n = 15) and control (n = 16)* groups.**

		Control (n)	**PFPS (n)**
Sport	**Running**	16	12
	Cycling	7	3
	Gym	8	6
Years training	**< 5 years**	4	7
	5 - 10 years	4	4
	> 10 years	8	3
Sessions per week	**1**	0	3
	2	1	2
	3	3	2
	4	7	4
	5	5	3
	6	0	1

The average training hours of participants are shown in Table 3.5. The control group had significantly higher average training hours for running, compared to the PFPS group ($p = 0.04$; $t = 2.2$). There were no significant differences in the amount of cycling and gym training between groups.

Table 3.5 Training hours of participants in the PFPS (n = 15) and control (n = 16) groups. Data are expressed as mean ± SD.

Activity	**Control (n=16)**	**PFPS (n=15)**	**p**
Running ($h.wk^{-1}$)	5.1 ± 3.2	2.6 ± 2.6	0.002*
Cycling ($h.wk^{-1}$)	2.5 ± 3.5	2.4 ± 2.7	0.471
Gym ($h.wk^{-1}$)	0.9 ± 1.2	1.4 ± 2.0	0.162

****: indicates significant difference between groups (p<0.05)***

3.4.4 Running velocity

The average running velocity of participants during the running tests are shown in Table 3.6. There were no significant differences in running velocity between groups.

Table 3.6 Running velocity of participants in the PFPS (n = 15) and control (n = 16) groups. Data are expressed as mean ± SD.

Variable	**Control**	**PFPS**	**t**	**p**
Velocity (m/s)	3.40 ± 0.38	3.18 ± 0.34	1.67	0.11

3.4.5 Stance phase kinematics

3.4.5.1 Joint angles at foot strike

The lower limb joint angles at foot strike are shown in Table 3.7. Knee adduction at foot strike was significantly decreased in the PFPS group, compared to the control group (t = 2.72; p = 0.01) (Figure 1; Appendix 10). There were no significant differences in any other lower limb joint angles at foot strike between groups.

Table 3.7 Lower limb joint angles (degrees) at foot strike of participants in the PFPS (n = 15) and control (n = 16) groups. Data are expressed as mean ± SD.

Variable at foot strike	**Control**	**PFPS**	**t**	**p**
Hip Flexion	35.6 ± 5.6	34.5 ± 5.4	0.54	0.60
Hip Abduction	2.9 ± 4.8	4.0 ± 3.6	-0.77	0.45
Hip Internal rotation	15.2 ± 8.8	13.9 ± 7.7	0.41	0.68
Knee Flexion	6.9 ± 2.7	4.6 ± 3.9	1.86	0.07
Tibial Adduction	6.0 ± 3.6	2.8 ± 3.1	2.72	0.01 *
Tibial Internal rotation	-15.7 ± 9.9	-15.1 ± 8.7	-0.21	0.84
Ankle Plantarflexion	7.2 ± 6.3	10.9 ± 7.3	-1.53	0.14
Pelvic tilt (Contralateral down) (Coronal plane)	-2.2 ± 3.1	-1.8 ± 2.9	-0.35	0.73
Pelvic tilt (anterior) (Saggital plane)	17.0 ± 3.7	17.1 ± 3.1	-0.12	0.91
Pelvic rotation (Contralateral forward) (Transverse plane)	-1.6 ± 3.5	-2.3 ± 3.0	0.60	0.55

****: indicates a significant difference between groups (p < 0.05)***

3.4.5.2 Joint angles at toe off

The lower limb joint angles at toe off are shown in Table 3.8. There were no significant differences in lower limb joint angles at toe off between groups.

Table 3.8 Lower limb joint angles (degrees) at toe off of participants in the PFPS (n = 15) and control (n = 16) groups. Data are expressed as mean ± SD.

Variable at toe off	**Control**	**PFPS**	**t**	**p**
Hip Flexion	-7.9 ± 4.9	-11.4 ± 4.7	1.94	0.06
Hip Abduction	-4.7 ± 3.2	-4.2 ± 3.2	-0.44	0.66
Hip Internal rotation	9.4 ± 9.7	9.6 ± 5.8	-0.05	0.96
Knee Flexion	10.9 ± 4.8	9.7 ± 6.7	0.57	0.57
Tibial Adduction	6.5 ± 5.3	4.0 ± 2.7	1.59	0.12
Tibial Internal rotation	-6.0 ± 12.2	-6.4 ± 9.5	0.10	0.92
Ankle Plantarflexion	-20.0 ± 8.6	-20.2 ± 6.5	0.09	0.93
Pelvic tilt (Contralateral down) (Coronal plane)	5.2 ± 2.3	5.0 ± 2.2	0.31	0.76
Pelvic tilt (anterior) (Saggital plane)	18.2 ± 3.2	16.2 ± 3.7	1.67	0.11
Pelvic rotation (Contralateral forward) (Transverse plane)	1.2 ± 2.4	0.1 ± 3.8	0.94	0.35

No significant differences between groups

3.4.5.3 Range of motion during stance phase

The range of motion (ROM) for the pelvis, hip, knee and ankle joints during stance phase are shown in Table 3.9. Hip rotation ROM was significantly increased in the PFPS group, compared to the control group (t = -2.09; p = 0.046) (Figure 2; Appendix 10) and knee abduction/adduction ROM was also significantly increased in the PFPS group, compared to the control group (t = -2.34; p = 0.03) (Figure 3; Appendix 10). There were no significant differences between groups in any other joint ROM during stance phase.

Table 3.9 Lower limb joint range of motion (degrees) during stance phase of participants in the PFPS (n = 15) and control (n = 16) groups. Data are expressed as mean ± SD.

ROM during stance phase	Control	PFPS	t	p
Hip Flexion / Extension	44.8 ± 5.4	47.3 ± 4.4	-1.38	0.18
Hip Abduction / Adduction	14.9 ± 4.0	15.7 ± 4.3	-0.55	0.58
Hip Internal / External rotation	17.4 ± 6.0	21.8 ± 5.7	-2.09	0.046*
Knee Flexion / Extension	32.7 ± 4.6	32.0 ± 2.5	0.47	0.64
Tibial Adduction / Abduction	11.8 ± 3.6	14.8 ± 3.7	-2.34	0.03*
Tibial External / Internal rotation	35.1 ± 5.0	33.6 ± 5.3	0.78	0.44
Ankle Plantarflexion / Dorsiflexion	38.5 ± 9.8	42.2 ± 7.0	-1.21	0.24
Pelvic tilt (Coronal plane)	11.7 ± 3.6	11.9 ± 2.5	-0.18	0.86
Pelvic tilt (Saggital plane) Anterior / Posterior	5.4 ± 2.2	6.1 ± 1.9	-1.01	0.32
Pelvic rotation Medial / Lateral rotation	6.3 ± 2.3	7.9 ± 3.0	-1.71	0.10

****: indicates significant difference between groups (p<0.05)***

3.4.5.4 Peak joint angles during stance phase

The peak joint angles during stance phase are shown in Table 3.10. The peak knee flexion angle during stance phase was significantly decreased in the PFPS group, compared to the control group (t = 2.48; p = 0.02) (Figure 4; Appendix 10). There were no significant differences between groups in any other peak joint angles during stance phase.

Table 3.10 Peak lower limb joint angles (degrees) during stance phase of participants in the PFPS (n = 15) and control (n = 16) groups. Data are expressed as mean ± SD.

Peak angle during stance phase	Control	PFPS	t	p
Hip Flexion	37.5 ± 5.5	35.1 ± 5.1	1.27	0.21
Hip Abduction	10.3 ± 4.3	10.8 ± 3.7	-0.36	0.72
Hip Internal rotation	23.1 ± 8.2	28.3 ± 6.9	-1.91	0.07
Knee Flexion	38.0 ± 3.0	35.3 ± 3.0	2.48	0.02*
Tibial Adduction	17.2 ± 5.6	16.9 ± 4.9	0.13	0.90
Tibial Internal rotation	20.5 ± 8.9	18.6 ± 10.0	0.55	0.59
Ankle Plantarflexion	21.6 ± 3.2	21.9 ± 1.7	-0.40	0.70
Pelvic tilt (Contralateral down) (Coronal plane)	5.3 ± 2.2	5.5 ± 1.9	-0.27	0.79
Pelvic tilt (anterior) (Saggital plane)	19.3 ± 3.1	17.8 ± 3.3	1.27	0.21
Pelvic rotation (Contralateral forward) (Transverse plane)	1.9 ± 2.4	1.1 ± 3.1	0.82	0.42

****: indicates significant difference between groups (p<0.05)***

3.4.5.5 Percentage of stance phase to peak angles

The differences in percentage of stance phase to peak joint angles between groups are shown in Table 3.11. The percentage of stance phase to peak hip flexion angle was significantly decreased in the PFPS group, compared to the control group (t = 2.20; p = 0.04) (Figure 5; Appendix 10). There were no significant differences between groups for any other percentages of the stance phase to peak joint angles.

Table 3.11 Percentage of stance phase to the peak lower limb joint angles during stance of participants in the PFPS (n = 15) and control (n = 16) groups. Data are expressed as mean ± SD.

Percentage of stance phase to peak	**Control**	**PFPS**	**t**	**p**
Hip Flexion / Extension	15.5 ± 11.5	6.5 ± 11.2	2.20	0.04*
Hip Abduction / Adduction	35.8 ± 4.9	35.3 ± 5.6	0.28	0.78
Hip Internal / External rotation	30.1 ± 13.9	34.0 ± 12.7	-0.80	0.43
Knee Flexion / Extension	36.9 ± 17.5	37.9 ± 6.5	-0.41	0.68
Tibial Adduction / Abduction	41.2 ± 4.4	38.8 ± 4.2	1.51	0.14
Tibial External / Internal rotation	46.1 ± 11.9	44.1 ± 10.0	0.51	0.61
Ankle Plantarflexion / Dorsiflexion	56.5 ± 5.0	53.7 ± 2.6	1.85	0.08
Pelvic tilt (Coronal plane)	98.6 ± 2.9	96.3 ± 4.1	1.77	0.09
Pelvic tilt (Saggital plane) Anterior / Posterior	65.5 ± 25.2	51.3 ± 37.0	1.26	0.22
Pelvic rotation Medial / Lateral rotation	75.5 ± 25.8	68.1 ± 33.1	0.71	0.48

: indicates significant difference between groups (p<0.05)

3.4.6 Swing phase kinematics

3.4.6.1 Range of motion during swing phase

The range of motion (ROM) for the pelvis, hip, knee and ankle joints during swing phase are shown in Table 3.12. The knee flexion/extension ROM was significantly decreased in the PFPS group, compared to the control group (t = 2.35; p = 0.03) (Figure 6; Appendix 10). There were no significant differences between groups in any other joint ROM during swing phase.

Table 3.12 Lower limb joint range of motion (degrees) during swing phase of participants in the PFPS (n = 15) and control (n = 16) groups. Data are expressed as mean ± SD.

ROM during swing phase	**Control**	**PFPS**	**t**	**p**
Hip Flexion / Extension	61.0 ± 6.1	58.6 ± 7.7	0.89	0.38
Hip Abduction / Adduction	19.7 ± 6.4	21.9 ± 7.6	-0.88	0.39
Hip Internal / External rotation	28.3 ± 11.6	34.3 ± 13.8	-1.30	0.20
Knee Flexion / Extension	95.4 ± 9.2	85.3 ± 14.1	2.35	0.03*
Tibial Adduction / Abduction	37.7 ± 9.6	31.5 ± 7.9	-1.18	0.25
Tibial External / Internal rotation	38.9 ± 7.2	35.9 ± 7.5	1.06	0.30
Ankle Plantarflexion / Dorsiflexion	41.4 ± 15.6	38.8 ± 11.5	0.53	0.60
Pelvic tilt (Coronal plane)	14.6 ± 4.8	15.9 ± 6.3	-0.62	0.54
Pelvic tilt (Saggital plane) Anterior / Posterior	12.8 ± 3.6	13.1 ± 4.9	-0.19	0.85
Pelvic rotation Medial / Lateral rotation	11.3 ± 3.4	12.4 ± 3.9	-0.76	0.45

****: indicates significant difference between groups (p<0.05)***

3.4.6.2 Peak joint angles during swing phase

The peak joint angles during swing phase are shown in Table 3.13. The peak hip flexion angle during swing phase was significantly decreased in the PFPS group, compared to the control group (t = 2.24; p = 0.03) (Figure 7; Appendix 10) and the peak hip rotation angle during swing phase was significantly increased in the PFPS group, compared to the control group (t = -2.10; p = 0.046) (Figure 8; Appendix 10). There were no significant differences between groups in any other peak joint angles during swing phase.

Table 3.13 Peak lower limb joint angles (degrees) during swing phase of participants in the PFPS (n = 15) and control (n = 16) groups. Values are expressed as mean ± SD.

Peak angles during the swing phase	**Control**	**PFPS**	**t**	**p**
Hip Flexion	51.4 ± 6.7	45.7 ± 7.2	2.24	0.03*
Hip Abduction	6.5 ± 5.0	8.4 ± 6.0	-0.89	0.38
Hip Internal rotation	29.0 ± 9.6	37.1 ± 11.6	-2.10	0.046*
Knee Flexion	96.9 ± 9.5	88.5 ± 18.2	1.62	0.12
Tibial Adduction	30.7 ± 11.1	34.0 ± 9.5	-0.90	0.37
Tibial Internal rotation	16.8 ± 11.4	18.7 ± 11.0	-0.45	0.66
Ankle Plantarflexion	11.2 ± 5.6	14.9 ± 5.1	-1.81	0.08
Pelvic tilt (Contralateral down) (Coronal plane)	7.6 ± 2.7	7.6 ± 2.7	-0.01	1.00
Pelvic tilt (anterior) (Saggital plane)	24.1 ± 3.3	21.4 ± 4.7	1.78	0.09
Pelvic rotation (Contralateral forward) (Transverse plane)	8.6 ± 1.7	7.3 ± 3.9	1.10	0.28

: indicates significant difference between groups (p<0.05)

3.4.7 Summary of stance and swing phase kinematics

In summary, there were several significant differences in kinematic parameters between the PFPS group and the control group at various stages of the gait cycle. The PFPS group had significantly decreased knee adduction at foot strike ($p = 0.01$); increased knee abduction/adduction ROM during stance phase ($p = 0.03$); and increased hip rotation ROM during stance phase ($p = 0.046$), compared to the control group. During the stance phase, the PFPS group also had significant decreases in the peak knee flexion angle ($p = 0.019$), and the percentage of stance phase to peak hip flexion ($p = 0.04$), compared to the control group. During the swing phase the PFPS group had significantly decreased peak hip flexion ($p = 0.03$); and increased peak hip internal rotation ($p = 0.046$) angles, compared to the control group. The PFPS group also had significantly decreased knee flexion ROM during swing phase ($p = 0.026$), compared to the control group. A summary of the significant differences in lower limb kinematics in the PFPS group, compared to the control group is depicted in Table 3.14.

Table 3.14 Summary of significant differences in lower limb kinematics in the PFPS group, compared to the control group.

Gait phase	**Analysis**	**Flexion/ extension**	**Abduction/ adduction**	**Rotation**
Foot strike	**Angle at foot strike**	-	PFPS group has decreased knee adduction (p = 0.011)	-
Angle at toe off	**Angle at toe off**	-	-	-
Stance phase	**Peak angle during stance phase**	PFPS group has decreased peak knee flexion (p = 0.019)	-	-
	Range during stance phase	-	PFPS group knee has increased ROM (p = 0.027)	PFPS group has increased ROM (p = 0.046)
	Percentage of stance phase to peak range	PFPS group reaches peak hip flexion earlier in stance phase (p = 0.036)	-	-
Swing phase	**Peak angle during swing phase**	PFPS group has decreased peak hip flexion (p = 0.033)	-	PFPS group has increased peak hip internal rotation (p = 0.046)
	Range during swing phase	PFPS group has decreased knee flexion ROM (p = 0.026)	-	-

3.4.8 Logistic regression analyses

Selected logistical regression analyses were performed for the pelvis, hip and knee joints at foot strike, peak joint angles during stance phase and the percentage of stance phase to peak angles. The significant regression analyses for knee angles at foot strike, peak hip angles during stance phase and the percentage of stance phase to reach peak hip angles are presented in Table 3.15 – 3.17 respectively.

Table 3.15 Logistic regression analyses of knee angles at foot strike. Data are presented as the Odds ratio and 95% confidence interval (CI).

Term	**Odds Ratio**	**95% CI**	**P**
Knee flexion at foot strike (pain group < 4.6° flexion)	16.16	1.39 – 187.84	0.03*
Knee adduction at foot strike (pain group < 2.8° adduction)	19.44	1.71 – 221.22	0.02*
Knee rotation at foot strike (pain group > -15.1° internal rotation)	0.66	0.11 – 4.19	0.66

: indicates significance (p<0.05)

Table 3.16 Logistic regression analyses of peak hip angles during stance phase. Data are presented as the Odds ratio and 95% confidence interval (CI).

Term	**Odds Ratio**	**95% CI**	**P**
Peak hip flexion during stance (pain group < 35.1° flexion)	1.62	0.31 – 8.43	0.56
Peak hip adduction during stance (pain group > 10.8° adduction)	0.38	0.07 – 2.07	0.26
Peak hip rotation during stance (pain group > 28.3° internal rotation)	8.43	1.21 – 58.96	0.03*

: indicates significance (p<0.05)

Table 3.17 Logistic regression analyses of the percentage of stance phase to peak hip angles. Data are presented as the Odds ratio and 95% confidence interval (CI).

Term	**Odds Ratio**	**95% CI**	**P**
Percentage stance phase to peak hip Flexion (pain group < 6.5% of stance phase)	6.81	1.33 – 34.78	0.02*
Percentage stance phase to peak hip adduction (pain group < 35.3% of stance phase)	0.88	0.18 – 4.39	0.88
Percentage stance to peak hip rotation (pain group > 34.0% of stance phase)	2.4	0.47 – 12.28	0.29

: indicates significance (p<0.05)

With significance accepted as $p < 0.05$, four kinematic variables were identified as being more likely to be present in the group with PFPS. These factors included:

- Knee flexion angle at foot strike of less than 4.6° ($p = 0.03$)
- Knee adduction angle at footstrike of less than 2.8° ($p = 0.02$)
- Peak hip internal rotation angle during stance phase of more than 28.3° ($p = 0.03$)
- Percentage of stance phase to peak hip flexion of less than 6.4% ($p = 0.02$)

However, although regression analyses suggested that these factors may increase the likelihood of experiencing pain associated with PFPS, it is recognized that the 95% confidence intervals are very wide. These results should therefore be interpreted with caution.

3.5 Discussion

The potential causative or contributing factors to PFPS have been widely investigated in current literature, but there are some discrepancies in the conclusions[2;6;8;11;12;14;16;19;33;50;63;64;66;73-76]. Numerous studies have explored potential kinematic factors associated with PFPS[2;8;11;13;14;63;64]. There has been a recent shift in focus from PFJ kinematics and patellar maltracking during movement[2;14;60] to proximal lower limb kinematics[6;14;19]. However, the majority of studies have examined lower limb kinematics only during a stepping task[15;16;57;63], due to the increased PFJ reaction forces associated with this activity[1]. In a few studies where running kinematics have been investigated, only changes in peak ROM during stance phase were reported [6;17;57]. Novel aspects of our study were that 1) we investigated lower limb kinematics during running, 2) we investigated kinematics during the entire gait cycle (stance and swing phases) of the running gait cycle, and 3) we also measured other variables including peak ROM, time to peak ROM, and total ROM travelled during the gait cycle.

The main findings of this study relate to the differences in lower limb kinematics in individuals with PFPS at different stages of the gait cycle. In particular, there is a decrease in knee adduction at foot strike, increased hip rotation range of motion

(ROM) during stance phase, and increased knee adduction ROM during stance phase.

Other changes in lower limb kinematics during running include decreased peak knee flexion angles, and decreased percentage of stance phase to peak hip flexion. During swing phase, changes include decreased peak hip flexion angles and knee flexion ROM.

3.5.1 Participants

Previous research articles analyzing the kinematics of PFPS had study samples of between 10 and 48 participants[1;15;16;57;63]. This study had a sample size of 15 PFPS and 16 control participants, which provided statistical power of 90%. However, it is recognized that a larger sample group would have allowed for a more generalized representation of active individuals with PFPS.

In this study, the PFPS and control groups were matched for gender. Previous studies have often included female participants only[6;17;52;54], as females are reported to have an increased risk of developing PFPS. For example, Boling, et al[22] found that in 1525 participants from the United States Naval Academy, the incidence of PFPS was higher in females (15%) than in males (12%) ($p = 0.09$).

This study tried to achieve a more accurate representation of the general population by having an equal number of male and female participants in the PFPS and control groups. No sub-group analysis according to gender was attempted in this study, due to the small sample sizes for male and female participants respectively. However it is recognised that sub-group analysis should be conducted in future studies with larger sample sizes to determine whether there are gender differences in running kinematics in participants with and without PFPS.

3.5.2 Screening tests

Various screening tests have been used in previous studies[1;6;15;16;57;63], including generalized anterior knee pain or retropatellar knee pain elicited on: ascending or descending stairs[6;15;16;57;63]; running[1;15;16]; prolonged sitting[1;6;15;16;57]; squatting[6;16;57;63]; kneeling[6;16;57]; resisted terminal knee extension[57;63]; or a unilateral partial squat[63]. The majority of participants in the PFPS group experienced pain during stair descent and a partial squat. Interestingly, only 26% of participants in the PFPS group had pain on resisted terminal knee extension. These findings suggest that resisted terminal knee extension may have low sensitivity and specificity for PFPS. No previous research was found investigating the sensitivity and specificity of any of the screening tests and it is therefore recommended that further studies are required to establish the sensitivity and specificity of the screening tests mentioned. Participants with PFPS also had a large variation in duration of symptoms (Section 3.4.2, page 58), with participants having symptoms for between three and 52 weeks prior to testing. This large variation in the duration of symptoms is a possible limitation of the study. Participants with an extended history of pain may have developed chronic pain, which could have resulted in the development of altered movement patterns[77;78].

3.5.3 Descriptive and training characteristics

There were no significant differences between groups for any of the descriptive characteristics. Participants in both groups performed gym, running and cycling training. Participants in the control group performed significantly more running training, compared to participants in the PFPS group (Section 3.4.3, page 60). This may be related to higher patellofemoral joint reaction force (PFJRF) generated during running compared to cycling[79;80]. Chen et al[79] stated that running results in a PFJRF of 58.2 N.kg^{-1}, while Ericson et al[80] found that cycling resulted in a PFJRF of only 1.3 times body weight. Therefore, individuals with PFPS may avoid running training as increased patellofemoral joint reaction forces may lead to symptom reproduction.

3.5.4 Running velocity

There were no significant differences in running velocity between groups (Section 3.4.4, page 61). Maurer et al[81] demonstrated that running biomechanics change at higher velocities. When running at higher speeds, leg kinematics change mainly in the sagittal plane, with the largest kinematic changes between running speeds occurring at the foot, followed by the lower leg and lastly by the thigh. Maximum and minimum values for the heel marker were also reached earlier in the gait cycle at higher speeds when compared to slower speeds[81]. In the current study it was therefore important that the PFPS and control groups performed the running tests at similar speeds, to avoid any potential confounding factors that might influence lower limb kinematics.

3.5.5 Joint angles at foot strike

At foot strike there was a significant difference in the knee abduction/adduction angle with the PFPS group landing with decreased tibial adduction of the lower leg (Section 3.4.5.1, page 62). This decreased tibial adduction results in the PFPS group having an increase in the Q angle, thereby increasing the lateral force exerted on the patella and possibly causing it to track incorrectly[76]. This maltracking is thought to be a possible cause of PFPS as it places undue stress on the joint[76]. This is the first study that has observed changes in knee adduction angles associated with PFPS. Previous kinematic studies have not examined knee angles in this plane of motion. However, Powers[14] stated that an increased valgus of the knee may be a causative factor in the development of PFPS, due to its increasing the lateral force exerted on the patella by the quadriceps muscles.

3.5.6 Joint angles at toe off

There were no significant differences in joint angles at toe off between the two groups. No other studies have investigated angles at toe off and therefore a comparison cannot be made with previous results. A possible explanation for the absence of significant differences in the joint angles at toe off may be related to the position of the knee joint. At toe off the knee is extended, so the quadriceps muscles place minimal posterior directed PFJRF on the patella[1].

The lack of significant findings could also be due to the fact that, at toe off, the moments at the knee joint are related to the hip and ankle joint motion. With this reduced force, there would be a reduced need for the individual to alter their gait pattern to minimize pain.

3.5.7 Range of motion during stance phase

During the stance phase there was a significant increase in the range of motion of hip rotation (Section 3.4.5.3, page 64). Previous studies have shown an increase in the peak hip internal rotation in individuals with PFPS[50;65]. Although there were no differences in peak hip internal rotation ROM in the current study, there was a significant increase in hip rotation ROM travelled during stance phase. The PFPS participants also tended to have had increased hip external rotation at foot strike, although this finding was not significant. An increase in hip external rotation has been hypothesized to decrease the Q angle and thus the lateral force on the patella[14]. It is possible that participants with PFPS tended to land with slightly more hip external rotation than the control group, possibly to minimize the lateral force on the patella. The PFPS participants then travelled through a significantly greater hip rotation range of motion during the stance phase. The fact that the PFPS group is landing more externally rotated as a possible protective mechanism, and then moving through a significantly greater rotation range of motion could indicate a possible weakness of the hip external rotators during stance phase. A decrease in hip external rotation strength has been consistently demonstrated in previous studies have investigated causative factors of PFPS[6;8;11;14;17;50-52;54]. The greater overall rotation range of motion travelled by the hip during the stance phase may also add to the rotational stresses placed on the patella during running.

The knee also moved through a significantly larger range of motion in the abduction/adduction direction in the PFPS group. As discussed in Section 3.4.5.1 (page 62), at foot strike the PFPS group landed with significantly less adduction of the knee. The knee then travels through a significantly greater range of motion in this same direction, possibly in an attempt to reduce the increased Q angle set at foot strike. This finding, lends weight to Powers'[14] hypothesis that an increase in knee valgus increases the predisposition to PFPS.

3.5.8 Peak joint angles during stance phase

There was a significant difference between groups in the peak knee flexion/extension angle during stance phase (Section 3.4.5.4; page 65). The PFPS group had a lower peak knee flexion angle during the stance phase. This finding corresponds to a prospective study that described a decrease in knee flexion during a jump landing task as a predictive factor for the development of PFPS[50]. Boling et al[50] theorized that the reduction in peak knee flexion was a compensatory mechanism to decrease the posterior directed force on the patella. With a reduction in knee flexion the PFJRF will be decreased as the angle between the superiorly directed force of the quadriceps muscle and the inferiorly directed force of the quadriceps tendon on the patella is reduced. Although this change in kinematics is hypothesized as being compensatory, it is not possible for this study to conclude whether the reduction in peak knee flexion in participants with PFPS was due to causative or compensatory mechanisms.

3.5.9 Percentage of stance phase to peak angle

There was a significant difference between groups in the percentage of stance phase to the peak hip flexion/extension angle (Section 3.4.5.5; page 66). The PFPS group reached peak hip flexion significantly earlier during the stance phase than the control group. Time to peak joint angles has been described in previous literature as a means of minimizing load on a joint or of pain avoidance[2]. No previous studies have examined the amount of time to peak ranges during the gait cycle. The fact that the PFPS group reached its peak hip flexion faster may be a method of enhancing the shock absorption in the lower limb and thus decreasing the load placed on the PFJ between foot strike and mid stance[2]. This reduction in load would possibly reduce the pain experienced by the individuals with PFPS. It would be a means of avoiding pain, although at the time of testing, the PFPS participants had no pain.

3.5.10 Range of motion during swing phase

There was a significant difference between groups in the knee flexion/extension ROM during the swing phase (Section 3.4.6.1; page 67). The PFPS group moved through less knee flexion ROM during the swing phase.

This could be a compensatory mechanism of reducing the posterior directed PFJRF during swing phase. With less knee flexion there is a decrease in the angle between the quadriceps muscle and quadriceps tendon forces on the patella. This reduces the pressure on the patella and could possibly minimize pain. The decrease in range of motion travelled during swing phase could also be a compensatory mechanism to use the affected limb less, thus causing less irritation of the PFJ.

3.5.11 Peak joint angles during swing phase

There were significant differences between groups in the peak hip flexion/extension angle and the peak hip rotation angle (Section 3.4.6.2; page 68). During the swing phase the PFPS group had a lower peak hip flexion angle and a higher peak hip internal rotation angle than the control group. The lower peak hip flexion angle may be a mechanism of reducing the PFJRF. This however creates a problem during the swing phase, in that there will now be less ground clearance during the swing through. This potential problem may be compensated for by increasing hip internal rotation. The peak hip internal rotation angle during swing phase was significantly increased in the PFPS group, compared to the control group (Section 3.4.6.2; page 68). This increased internal hip rotation could allow the foot to avoid making contact with the ground during the swing phase, with the decreased peak hip flexion angle and knee flexion ROM. The decrease in range of motion travelled during swing phase could also be a compensatory mechanism to use the affected limb less, thus causing less irritation of the PFJ. Furthermore, no previous studies that described the kinematics of the swing phase of gait were identified; therefore it is not possible to compare the findings of this study with current literature. The large rotational range of motion travelled in the lower limb during stance phase and swing phase could also be a cause of PFPS, due to the frictional forces that result from this increased movement in the sagittal plane.

3.5.12 Logistic regression analyses

This study identified four kinematic variables that may be associated with an increased risk of experiencing PFPS (Section 3.4.8, page 71).

Although these findings are supported by previous investigations into the risk factors for developing PFPS[14;19;50;51;76], the large confidence intervals strongly suggest that the findings of this study should be interpreted with caution. As such no further discussion regarding potential underlying reasons associated with the risk factors identified in this study is warranted. In addition, it is also recognized that the fit of the logistic regression model may be improved with an increased sample size.

3.5.13 Limitations of the study

The main limitation of this study was that it examined lower limb kinematics in participants with pre-existing PFPS. It was therefore not possible to establish whether the changes in lower limb kinematics of participants in the PFPS group were causative, thereby contributing to the development of PFPS; or compensatory, in response to the presence of pain and other factors associated with PFPS. It is recommended that future prospective, longitudinal studies should be conducted to differentiate between causative and compensatory changes in lower limb kinematics associated with PFPS.

Another limitation is that the sample groups were not homogenous in relation to type of exercise. Initially this study planned to only recruit runners, but due to a lack of runners with PFPS, the inclusion criteria were expanded to include any active individuals. Therefore, the control group included more runners and the PFPS group included more participants that took part in other sports. This is indicated by the significantly greater amount of running performed by the control group (Section 3.4.3; page 60). It is recognized that widening the inclusion criteria may have introduced different predisposing factors to PFPS, for example running shoes and bike set up. It is important that future studies clearly identify potential extrinsic predisposing factors to PFPS.

There was also a large variation in duration of symptoms in the PFPS group. This has been discussed in section 3.4.2, page 58. Future studies should place an upper limit on the duration of PFPS symptoms, to avoid the development of chronic pain and the potential for altered movement patterns.

In this study, the size of the force plate and the short running track are also potential limiting factors to the study. Participants may have anticipated stepping on the force plate or may not have been able to get into their proper stride by the time they reached the force plate, which may have altered their running gait.

In an effort to counter this limitation, the participants were not told about the force plate until after the study and numerous trials were performed, but only those trials where the participant landed with their whole foot on the force plate were analyzed. They were also allowed ten practice trials before testing started to get comfortable with the length of the running track. In addition, movement of the skin could cause unwanted movement of the markers. This movement could result in changes in the actual kinematics found. All the participants in both groups had the markers placed in the same way so that any possible variations would be consistent.

3.5 Summary

The results from this study demonstrated differences in lower limb kinematics whilst running between active individuals with PFPS compared to healthy controls. Alterations in lower limb kinematics may either contribute to the development of PFPS or may be compensatory to the pain caused by of PFPS. It is difficult to distinguish between causative and compensatory changes in this study. However, it may be postulated that the following changes may be causative in nature: decreased knee adduction at foot strike, increased hip rotation ROM during stance phase, and increased knee abduction/adduction ROM during stance phase. These changes may increase the Q angle and therefore the lateral force exerted on the patella during the running gait cycle. Alternatively, the changes in the lower limb kinematics that may be due to compensatory adjustments in the running gait secondary to pain and potential muscle imbalances associated with PFPS include: decreased peak knee flexion angles, and decreased percentage of stance phase to peak hip flexion during stance phased; and decreased peak hip flexion angles and knee flexion ROM during swing phase. These changes may decrease the posterior directed PFJRF and the frictional forces on the patella, and may be associated with reduced knee pain.

The kinematic findings from this study may be useful for clinicians when performing dynamic biomechanical movement analysis as a reference for common patterns observed in PFPS patients during running. It is recommended that the treatment of PFPS should include gait and running re-education and other strategies to address changes in lower limb kinematics. Further studies are required to differentiate between causative and compensatory changes in lower limb kinematics in participants with PFPS. Additional research is also required to identify optimal interventions which promote symmetrical and efficient running patterns. Further studies combining kinematics, strength and EMG are also indicated.

Chapter 4: Summary and conclusion

Patellofemoral pain syndrome (PFPS) is a musculoskeletal condition affecting the knee joint in a large number of active individuals[1;6;7;10]. It is characterized by retropatellar and/or peripatellar pain[1;7;10]. It has been suggested that approximately 2.5 million runners will be diagnosed with PFPS in a given year and that in the military, 37% of new recruits will develop PFPS during basic training[1;11;19]. It is also prevalent in the work place with 11% of musculoskeletal complaints being due to PFPS[1]. Further, between 70% and 90% of PFPS sufferers will have symptoms up to 20 years after the initial diagnosis[19].

It is important for clinicians to understand the causes of a disorder. PFPS is a multifaceted disorder with both extrinsic and intrinsic predisposing factors[19]. These extrinsic and intrinsic factors may affect an individual's lower limb kinematics. The potential changes in lower limb kinematics associated with PFPS were the focus of this study, due to the lack of evidence regarding kinematic changes during the running gait cycle in participants with PFPS. Therefore, the overall aim of this dissertation was to investigate the potential lower limb kinematics associated with PFPS during running. Based on the evidence provided in this thesis, the study objectives as described in Section 1.2.2 (page 3) may be answered as follows:

> *To determine normative values for lower limb joint angles and range of motion during running in participants with and without PFPS.*

Joint angles were determined at various stages of the gait cycle. These included the angles at foot strike, peak ranges during stance phase, range of motion travelled during stance phase, angles at toe off, peak ranges during swing phase and range of motion travelled during the swing phase. In addition, the study also assessed the percentage of the stance phase to reach peak ranges. The joints included in the assessment were the pelvis, hip, knee and ankle. The sample size had sufficient power, and included both males and females.

To determine whether there were differences in pelvis, hip, knee and ankle kinematics during running in participants with and without PFPS.

In this study there were a number of significant differences in kinematics between groups. These differences included a decrease in knee adduction at foot strike, an increase in hip rotation range of motion (ROM) during stance phase, and an increase in knee abduction/adduction ROM during stance phase. It was hypothesized that these changes may be possible causative factors in the development of PFPS as they increase the Q angle. Further, other changes in lower limb kinematics were thought to be due to compensatory adjustments in running gait. These kinematic changes included a decrease in peak knee flexion angles; a decrease in the percentage of stance phase it took reach peak hip flexion; and decreases in peak hip flexion angles and knee flexion ROM during swing phase. They are thought to be compensatory as they reduce the PFJRF, minimize the time in painful positions and allow for gait even with the other changes.

To determine whether there were any kinematic variables at the pelvis, hip and knee joint during stance phase of running that may predispose an individual to PFPS.

Although this study identified four kinematic variables that may be associated with an increased risk of PFPS, large confidence intervals indicated that these findings should be interpreted with caution. This study was unfortunately not able to unequivocally identify kinematic factors that may predispose to the development of PFPS.

Based on the findings of this study it may be stated that PFPS is a complex condition that involves kinematic changes in the entire lower limb. These kinematic changes could be either causative or compensatory in nature, and further prospective studies are required to establish the pattern and nature of lower limb kinematics changes in participants with PFPS. In addition, although this is a preliminary study, the findings have implications for the assessment and management of PFPS. It is recommended that clinicians perform a holistic biomechanical assessment of the lower limb to determine alterations in alignment or movement patterns. Furthermore, it is recommended that clinicians should address strength deficits and retrain gait patterns for successful long term management of PFPS.

Chapter 5: Reference List

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Appendix 1: Informed Consent

Dear Participant,

The University of Cape Town Division of Physiotherapy will be conducting a study to measure certain biomechanical factors in subjects with and without patellofemoral pain syndrome (PFPS).

Patellofemoral pain syndrome is a condition characterised by pain around the knee cap (patella). It is thought to be caused by an increase in friction between the patella and the groove through which it runs in the tibia. This increase in friction can be caused by various biomechanical factors, like foot pronation and hip position, altering the line the patella tracks along on the tibia.

In this study we will be looking at some of these biomechanical factors thought to predispose a runner to PFPS. They will include ground reaction forces and 3D motion analyses, measured during running. The study will improve our understanding of the biomechanical imbalances that occur with this condition, so that treatment may be appropriately directed in future.

The study will be performed as part completion for the 3 year Masters course in Sports Physiotherapy. The study has been given Ethical Approval by the Faculty of Health Sciences Human Research Ethics Committee at the University of Cape Town (HREC/REF 361/2009)

You will need to report to the gait laboratory at The Sports Science Institute of South Africa for 1 appointment. The visit will take approximately 2 hours. The research project will include the following procedures:

- You will be required to complete a medical questionnaire indicating your level of health and any history of illness.
- Anthropometric measurements involving measurements of your body composition including your weight, height and percentage of body fat, determined by measuring 7 skinfolds, including triceps, biceps, suprailiac, subscapular, calf, thigh and abdomen will be taken.
- A training history of all running activities over the past 6 months will be taken.
- You will be taken through the testing routine and familiarised with all the equipment that is going to be used on you during the process.
- You will then be prepared for the testing protocol. Sixteen reflective markers will be attached using double sided tape to various places on the lower limb.
- You will be asked to do a warm-up of 5 minutes of running at a self selected speed on a treadmill.
- You will be taken through the testing procedure with the equipment attached and allowed a trial run. The test involves running across a 10m track trying to time it so that your affected leg (Right leg in control subjects) hits the middle of the force plate on the ground.
- Testing will take place. This involves running across the 10m track, hitting the force plate with a normal stride 10 times, while all the data is collected.
- The data will include high speed video footage taken by motion analysis cameras and readings taken from a force plate on the ground.
- Between each running trial you will be asked to rate your pain (if any) on a scale of 1 to 10.
- After testing all equipment will be removed and you will be provided with an information pack regarding patellofemoral pain
- All results of your medical screening and the outcome from the research will be forwarded to you as soon as possible.

Potential Risks of participation:

Anthropometry and patellofemoral pain measurement

There are no potential risks to you that may be associated with mass, stature, skinfold measurements, or the subjective measurement of patellofemoral pain syndrome.

Running tests

Running is associated with a minor risk of sustaining a musculoskeletal injury or falling. In this study, you will undergo a thorough familiarisation process. All due care will be taken to ensure that you are both familiar and confident with running along the pathway and across the force plate. You will also be excluded from the study on the basis of medical or surgical history, any musculoskeletal injury, and any medication use or viral infection within the 12 weeks preceding the study. You will be required to warm up before the running tests on a treadmill at a self selected speed. The warm-up will be monitored to reduce the risk of any musculoskeletal injury that may be associated with running. For the injured group, if there is any aggravation of your symptoms, the testing procedure will be stopped immediately and you will be advised on appropriate care and referred to a physiotherapist for treatment.

The use of a six-camera motion analysis system and a force plate for the measurement of kinematic and kinetic data provide no additional potential risks to you.

Potential benefits:

You will not receive any financial compensation for participating in this study. You will receive a detailed pamphlet containing information about patellofemoral pain syndrome as well as normally recommended preventative measures and treatment options. You will be given feedback regarding your general health screen and anthropometric measurements. On completion of the study, the summarised results and recommendations will be formally presented to you.

Questions or Concerns:

If at any time you have any questions about the study please feel free to contact any of the individuals listed below. You are assured that all enquiries will remain confidential.

Chris Allan	082 922 8912
Theresa Burgess	021 406 6171

Should you have any further queries, feel free to contact the project supervisor:

Ms. T. Burgess

Fax number: 021 406 6323

Email: theresa.burgess@uct.ac.za

Tel: 021 406 6171

083 300 7763

You may also contact Professor Marc Blockman of the Human Research Ethics Committee, Faculty of Health Sciences:

Email: marc.blockman@uct.ac.za

Tel: 021 406 6338

Please note that UCT does offer a no-fault insurance that will cover all participants in the event that something may go wrong.

I confirm that the exact procedures and possible complications of the above tests have been explained to me. I understand that I may ask questions at any time during the testing procedures. I realise that I am free to withdraw from the study without prejudice at any time, should I choose to do so. I have been informed that the personal information required by the researchers will be held in strict confidentiality. In addition, I know that the information derived from the testing procedures will remain confidential and will be revealed only as a number in statistical analyses.

I have carefully read this form. I understand the nature, purpose and procedure of this study. I agree to participate in this research project of the Division of Physiotherapy.

____________________ ____________________________

Signature of Volunteer Name (Please Print)

____

Date

____________________ ____________________________

Signature of Witness Name (Please Print)

____

Date

____________________ ____________________________

Signature of Investigator Name (Please Print)

____

Date

Appendix 2

General Health Questionnaire

University of Cape Town Physiotherapy Study

Instructions:

This questionnaire consists of 4 pages

Please read each question carefully as it is important that we obtain accurate information.

Please place information in the appropriate text box e.g. Date of Birth

21/03/1983 Day/Month/Year

If a question is asked, please place an ‘x’ in the appropriate text box.

For example: Which province do you live in?

Western Province ☐ Free State ☐ Kwa-Zulu Natal ☐

Please answer all questions as truthfully as possible. The information gathered will be used in the study but will remain strictly anonymous.

If you have any questions do not hesitate to phone or e-mail any of the individuals below:

Chris Allan	082 922 8912	callan@wam.co.za
Theresa Burgess	021 406 6171	
	083 300 4991 / theresa.burgess@uct.ac.za	

Name: ___________________________________

Surname: ________________________________

Age: _____________________________________

Date of Birth: ___/____/_____

Have you been injured in the past 6 months?

____

If yes what type of injury? i.e.: muscle pulled, broken bone, ligament damage

____

Where was/is the injury? I.e.: Left leg, left hand, right knee

____

How did the injury occur? During your usual exercise routine or other activity

____

____

Have you had any physiotherapy or massage treatment in the last 6 months? If yes please specify.

____

____

Have you been instructed in any form of Rehabilitation (exercise) programme for this or any other injury in the last 6 months?

____

____

Do you have any previous surgical history?

Cardiac Surgery ☐ Other ☐

Spinal Surgery ☐ Fractures ☐

Please specify where: ____

____

Have you been ill in the past 12 weeks? If so, what illness was/ is it? E.g. cold, flu, measles

____

____

If you answered “Yes” above, did you take any medication for the illness? What was it called?

____

____

Is there any medication that you take regularly to manage pain/injuries eg: paracetamol, anti-inflammatories?

____

Are you on any other form of medication at present? If so, what is it?

____

____

Have you ever been diagnosed with any of these disorders?

Coronary Heart Disease ☐

Diabetes ☐

Thyroid Disease ☐

Allergies:__________

Tuberculosis ☐

Osteoarthritis ☐

High Cholesterol ☐

Asthma ☐

Rheumatoid Arthritis ☐

Renal Disease ☐

High Blood Pressure ☐

Osteoporosis ☐

Cancer ☐

Stroke ☐

Other (please specify):

____

Subjects Signature:__Date:______________

Subjects Contact Details:______________________

Appendix 3

Training History

How many years have you been running for?

☐ Less than 5 years

☐ 5 – 10 years

☐ More than 10 years

Do you have a training log that you could supply to us showing the type/distance of your training over the past 6 months?

☐ Yes

☐ No

How often do you run each week?

☐1 ☐2 ☐3 ☐4 ☐5 ☐6 ☐7 ☐more

Give a breakdown on the type of workouts (e.g. tempo, speed, distance, etc) during the week.

____

____

____

Where are you in your training at present (Pre-, mid-, post- event)?

____

What is your average weekly mileage in km's at present?

____

And during the rest of your training (i.e. pre-, post- event)?

____

At what intensity do you do most of your training?

____

Any further comments on your training?

____

____

Give an indication on your times as indicated below.

Distance	Personal Best	Current Estimate
5km		
10km		
21km		
42km		

Appendix 4

Anthropometrical and injury assessment work sheet.

Name: _________________________Date:________________

DOB: ___________

Weight: ___________Height: ________________

Ankle width: Left: _______mm Right: _______mm

Knee width: Left: _______mm Right: _______mm

Leg Length: Left: _______mm Right: _______mm

Q- Angle: _______________ (♂11.2°±3.0°; ♀15.8°±4.5°)

Skinfold Measurements:

Anatomical Areas	1	2	Avg
Triceps Biceps Subscapularis Supra iliac Abdomen Thigh Calf Mid-Thigh circumference Upper________, Middle________, Lower________ Length of quad ________			

Patellofemoral Pain Assessment:

Duration of symptoms: ____________

Cause non-traumatic: Y/N

Area of Pain: Anterior / Retro-patellar / other: _____________

Pain: During exercise Y/N

Then: Eases/Worsens

Only after Exercise Y/N

Type of shoes: ____

Cushioning / Neutral / Stability / Motion Control

Orthotics: Y/N

Provocation tests:

Resisted terminal knee extension:

Stair descent:

Unilateral Partial squat:

Q- Angle: _______________ (♂11.2°±3.0°; ♀15.8°±4.5°)

Pre test VAS score: _____________________________________

Post test VAS score: ____________________________________

Appendix 5: Data for analysis

	Mean 1 (Control Grp)	Mean 2 (PFPS Grp)	t-value	p	Std.Dev. Group 1	Std.Dev. Group 2
Velocity	3.40	3.18	1.68	0.10	0.38	0.34
Hip Angles at foot strike X	35.60	34.53	0.55	0.59	5.55	5.35
Hip Angles at foot strike Y	2.85	4.04	-0.77	0.45	4.82	3.59
Hip Angles at foot strike Z	15.16	13.92	0.41	0.68	8.77	7.65
Knee Angles at foot strike X	6.85	4.63	1.86	0.07	2.73	3.87
Knee Angles at foot strike Y	6.03	2.75	2.72	0.01	3.60	3.05
Knee Angles at foot strike Z	-15.74	-15.06	-0.20	0.84	9.93	8.67
AbsAnkleAngle at foot strike X	7.15	10.87	-1.53	0.14	6.28	7.28
Pelvis Angles at foot strike X	-2.19	-1.81	-0.35	0.73	3.08	2.86
Pelvis Angles at foot strike Y	16.96	17.10	-0.11	0.91	3.65	3.14
Pelvis angles at foot strike Z	-1.56	-2.32	0.63	0.53	3.48	3.04
Hip Angles at toe off X	-7.94	-11.40	1.93	0.06	4.89	4.73
Hip Angles at toe off Y	-4.68	-4.18	-0.42	0.67	3.24	3.20
Hip Angles at toe off Z	9.43	9.59	-0.05	0.96	9.67	5.80
Knee Angles at toe off X	10.90	9.70	0.56	0.58	4.76	6.73
Knee Angles at toe off Y	6.47	4.04	1.59	0.12	5.30	2.70
Knee Angles at toe off Z	-5.99	-6.37	0.10	0.92	12.15	9.51
AbsAnkleAngle at toe off X	-19.95	-20.21	0.09	0.93	8.59	6.48
Pelvis angles at toe off X	5.21	4.96	0.31	0.76	2.34	2.21
Pelvis angles at toe off Y	18.22	16.15	1.69	0.10	3.19	3.65
Pelvis angles at toe off Z	1.22	0.13	0.97	0.34	2.42	3.76

	Mean 1 (Control Grp)	Mean 2 (PFPS Grp)	t-value	p	Std.Dev. Group 1	Std.Dev. Group 2
Peak hip angles during stance X	37.52	35.08	1.28	0.21	5.48	5.09
Peak hip angles during stance Y	10.32	10.84	-0.35	0.73	4.33	3.69
Peak hip angles during stance Z	23.05	28.31	-1.92	0.07	8.21	6.94
Peak knee angles during stance X	37.97	35.30	2.48	0.02	3.04	2.95
Peak knee angles during stance Y	17.19	16.94	0.13	0.89	5.60	4.90
Peak knee angles during stance Z	20.46	18.56	0.55	0.59	8.92	10.02
Peak AbsAnkleAngle during stance X	21.55	21.91	-0.38	0.70	3.19	1.70
Peak pelvis angle during Stance X	5.30	5.51	-0.27	0.79	2.23	1.86
Peak pelvis angle during Stance Y	19.25	17.76	1.29	0.21	3.14	3.28
Peak pelvis angle during Stance Z	1.92	1.07	0.85	0.40	2.39	3.09
Percentage stance to peak hip X	15.46	6.48	2.20	0.04	11.50	11.19
Percentage stance to peak hip Y	35.81	35.26	0.28	0.78	4.92	5.57
Percentage stance to peak hip Z	30.13	33.96	-0.80	0.43	13.90	12.68
Percentage stance to peak knee X	36.94	37.95	-0.39	0.70	7.49	6.50
Percentage stance to peak knee Y	41.15	38.79	1.49	0.15	4.44	4.21
Percentage stance to peak knee Z	46.14	44.07	0.52	0.60	11.90	9.99

	Mean 1 (Control Grp)	Mean 2 (PFPS Grp)	t-value	p	Std.Dev. Group 1	Std.Dev. Group 2
Percentage stance to peak abs ankle X	56.48	53.73	1.85	0.07	4.96	2.60
Percentage stance to peak pelvis X	98.55	96.25	1.76	0.09	2.92	4.07
Percentage stance to peak pelvis Y	65.51	51.28	1.26	0.22	25.18	36.94
Percentage stance to peak pelvis Z	75.48	68.05	0.70	0.49	25.77	33.06
Peak hip angles during swing X	51.36	45.67	2.25	0.03	6.72	7.15
Peak hip angles during swing Y	6.54	8.37	-0.89	0.38	5.01	5.99
Peak hip angles during swing Z	28.98	37.10	-2.09	0.05	9.57	11.63
Peak knee angles during swing X	96.91	88.50	1.63	0.11	9.47	18.23
Peak knee angles during swing Y	30.68	34.02	-0.90	0.38	11.14	9.45
Peak knee angles during swing Z	16.84	18.68	-0.44	0.66	11.43	10.95
Peak abs ankle angle during swing X	11.21	14.91	-1.82	0.08	5.61	5.14
Peak pelvis angle during swing X	7.58	7.59	-0.01	1.00	2.67	2.67
Peak pelvis angle during swing Y	24.05	21.41	1.79	0.08	3.29	4.66
Peak pelvis angle during swing Z	8.56	7.31	1.11	0.28	1.71	3.85
Hip ROM during stance X	44.82	47.25	-1.36	0.19	5.37	4.41
Hip ROM during stance Y	14.88	15.71	-0.56	0.58	4.02	4.25
Hip ROM during stance Z	17.41	21.81	-2.09	0.05	6.03	5.67

	Mean 1 (Control Grp)	Mean 2 (PFPS Grp)	t-value	p	Std.Dev. Group 1	Std.Dev. Group 2
Knee ROM during stance X	32.70	32.03	0.50	0.62	4.63	2.49
Knee ROM during stance Y	11.76	14.82	-2.33	0.03	3.57	3.74
Knee ROM during stance Z	35.08	33.57	0.79	0.44	5.02	5.26
Abs ankle ROM during stance X	38.47	42.19	-1.21	0.24	9.80	7.00
Pelvis ROM during stance X	11.72	11.92	-0.18	0.86	3.57	2.51
Pelvis ROM during stance Y	5.38	6.12	-1.01	0.32	2.21	1.85
Pelvis ROM during stance Z	6.28	7.92	-1.72	0.10	2.26	3.02
Hip ROM during Swing X	60.97	58.61	0.91	0.37	6.09	7.68
Hip ROM during Swing Y	19.69	21.92	-0.87	0.39	6.38	7.55
Hip ROM during Swing Z	28.30	34.29	-1.29	0.21	11.58	13.82
Knee ROM during Swing X	95.36	85.27	2.35	0.03	9.15	14.11
Knee ROM during Swing Y	27.69	31.51	-1.18	0.25	9.46	7.86
Knee ROM during Swing Z	38.87	35.88	1.08	0.29	7.20	7.49
Abs ankle ROM during Swing X	41.39	38.81	0.52	0.61	15.58	11.52
Pelvis ROM during Swing X	14.57	15.86	-0.62	0.54	4.82	6.30
Pelvis ROM during Swing Y	12.76	13.06	-0.18	0.86	3.58	4.94
Pelvis ROM during Swing Z	11.33	12.37	-0.78	0.44	3.42	3.89

Appendix 6: Logistic regressions tables

Logistic regressions analyses at foot strike

Hip Joint

Term	Odds Ratio	C.I.	P
Hip flexion at foot strike	0.58	0.11 – 3.08	0.53
Hip adduction at foot strike	2.36	0.45 – 12.41	0.31
Hip rotation at foot strike	1.32	0.30 – 5.71	0.71

Knee joint

Term	Odds Ratio	C.I.	P
Knee flexion at foot strike	16.16	1.39 – 187.84	0.03*
Knee adduction at foot strike	19.44	1.71 – 221.22	0.02*
Knee rotation at foot strike	0.66	0.11 – 4.19	0.66

p<0.05

Pelvis

Term	Odds Ratio	C.I.	P
Pelvic tilt (Contralateral down) (Coronal plane) at foot strike	0.89	0.19 – 4.09	0.88
Pelvic tilt (anterior) (Saggital plane) at foot strike	2.28	0.50 – 10.49	0.29
Pelvic rotation (Contralateral forward) (Transverse plane)at foot strike Z	1.87	0.38 – 9.13	0.44

Logistic regressions on peak angles during stance phase

Hip joint

Term	Odds Ratio	C.I.	P
Peak hip flexion during stance	1.62	0.31 – 8.43	0.56
Peak hip adduction during stance	0.38	0.07 – 2.07	0.26
Peak hip rotation during stance	8.43	1.21 – 58.96	0.03*

p<0.05

Knee joint

Term	Odds Ratio	C.I.	P
Peak knee flexion during stance	3.59	0.66 – 19.45	0.14
Peak knee adduction during stance	1.63	0.30 – 8.85	0.57
Peak knee rotation during stance	1.58	0.30 – 8.32	0.59

Pelvis

Term	Odds Ratio	C.I.	P
Peak pelvis tilt (coronal plane) during stance	0.44	0.10 – 2.02	0.29
Peak pelvis tilt (Saggital plane) during stance	1.66	0.36 – 7.74	0.52
Peak pelvis rotation (transverse plane) during stance	1.89	0.42 – 8.55	0.41

Logistic regressions done on percentage of stance phase to peak angle

Hip joint

Term	Odds Ratio	C.I.	P
Percentage stance phase to peak hip Flexion	6.81	1.33 – 34.78	0.02*
Percentage stance phase to peak hip adduction	0.88	0.18 – 4.39	0.88
Percentage stance to peak hip rotation	2.4	0.47 – 12.28	0.29

p<0.05

Knee joint

Term	Odds Ratio	C.I.	P
Percentage stance to peak knee flexion	0.80	0.17 – 3.78	0.78
Percentage stance to peak knee adduction	2.87	0.57 – 14.38	0.20
Percentage stance to peak knee rotation	2.01	0.45 – 8.84	0.36

Pelvis

Term	**Odds Ratio**	**C.I.**	**P**
Percentage stance to peak pelvis tilt (Coronal plane)	2.90	0.57 – 14.86	0.20
Percentage stance to peak pelvis tilt (saggital plane)	0.84	0.19 – 3.70	0.82
Percentage stance to peak pelvis rotation (transverse plane)	1.43	0.33 – 6.25	0.64

Appendix 7: Ethics approval

UNIVERSITY OF CAPE TOWN

Health Sciences Faculty
Research Ethics Committee
Room E52-24 Groote Schuur Hospital Old Main Building
Observatory 7925
Telephone [021] 406 6338 • **Facsimile** [021] 406 6411
e-mail: sumayah.ariefdien@uct.ac.za

14 October 2009

REC REF: 361/2009

Mr C Allan
Physiotherapy
Health & Rehab
F Floor
OMB

Dear Mr Allan

PROJECT TITLE: AN INVESTIGATION OF POTENTIALKINETIC, KINEMATIC AND NEUROMUSCULAR FACTORS ASSOCIATED WITH PATELLOFEMORAL PAIN SYNDROME IN RUNNERS

Thank you for submitting your study to the Research Ethics Committee for review.

It is a pleasure to inform you that the Ethics Committee has **formally approved** the above-mentioned study.

Approval is granted for one year until 15 October 2010.

Please submit an annual progress report if the research continues beyond the expiry date. Please submit a brief summary of findings if you complete the study within the approval period so that we can close our file.

Please note that the ongoing ethical conduct of the study remains the responsibility of the principal investigator.

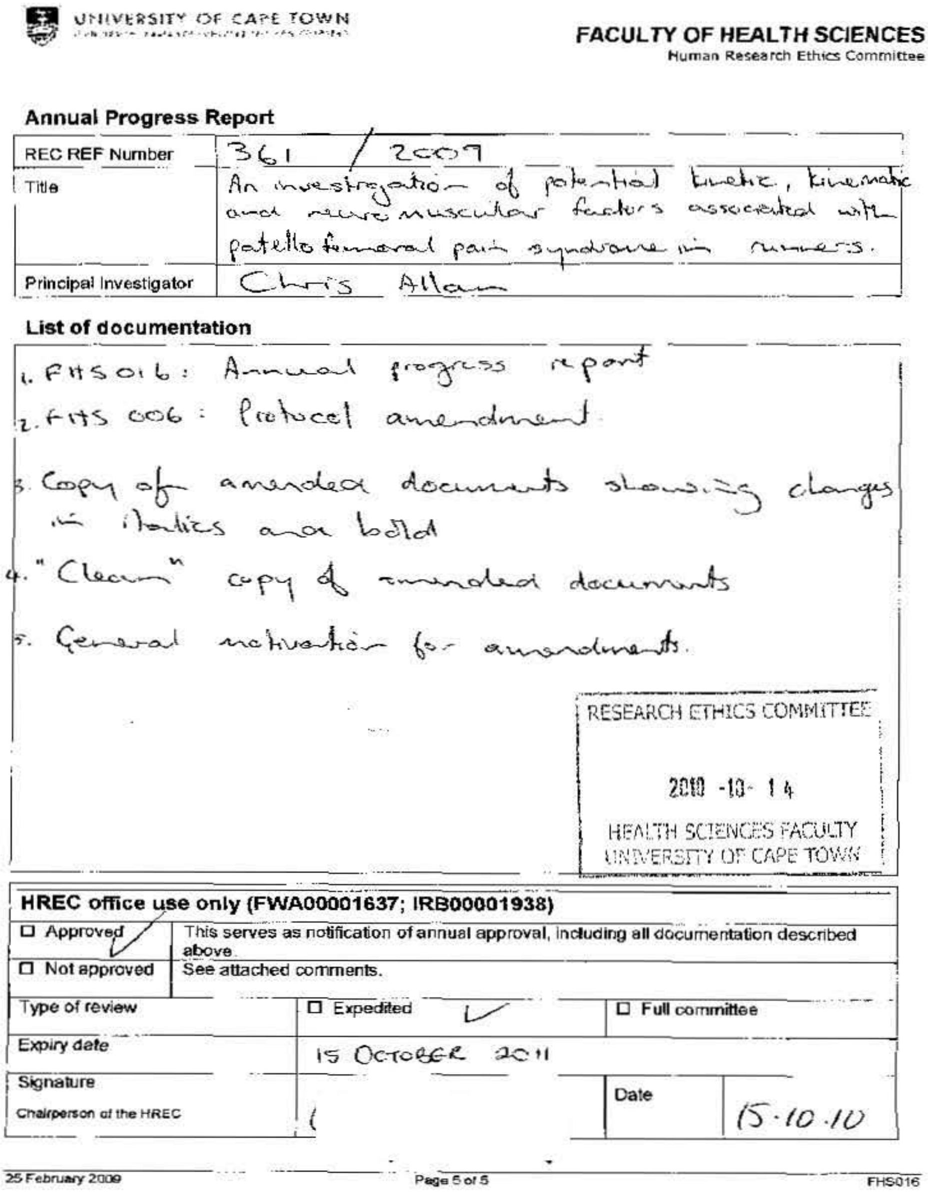
Please quote the REC. REF in all your correspondence.

Yours sincerely

PROFESSOR M BLOCKMAN
CHAIRPERSON, HSF HUMAN ETHICS

Federal Wide Assurance Number: FWA00001637.

lem jedi



UNIVERSITY OF CAPE TOWN

FACULTY OF HEALTH SCIENCES
Human Research Ethics Committee

Annual Progress Report

REC REF Number	361 / 2009
Title	An investigation of potential kinetic, kinematic and neuromuscular factors associated with patellofemoral pain syndrome in runners.
Principal Investigator	Chris Allan

List of documentation

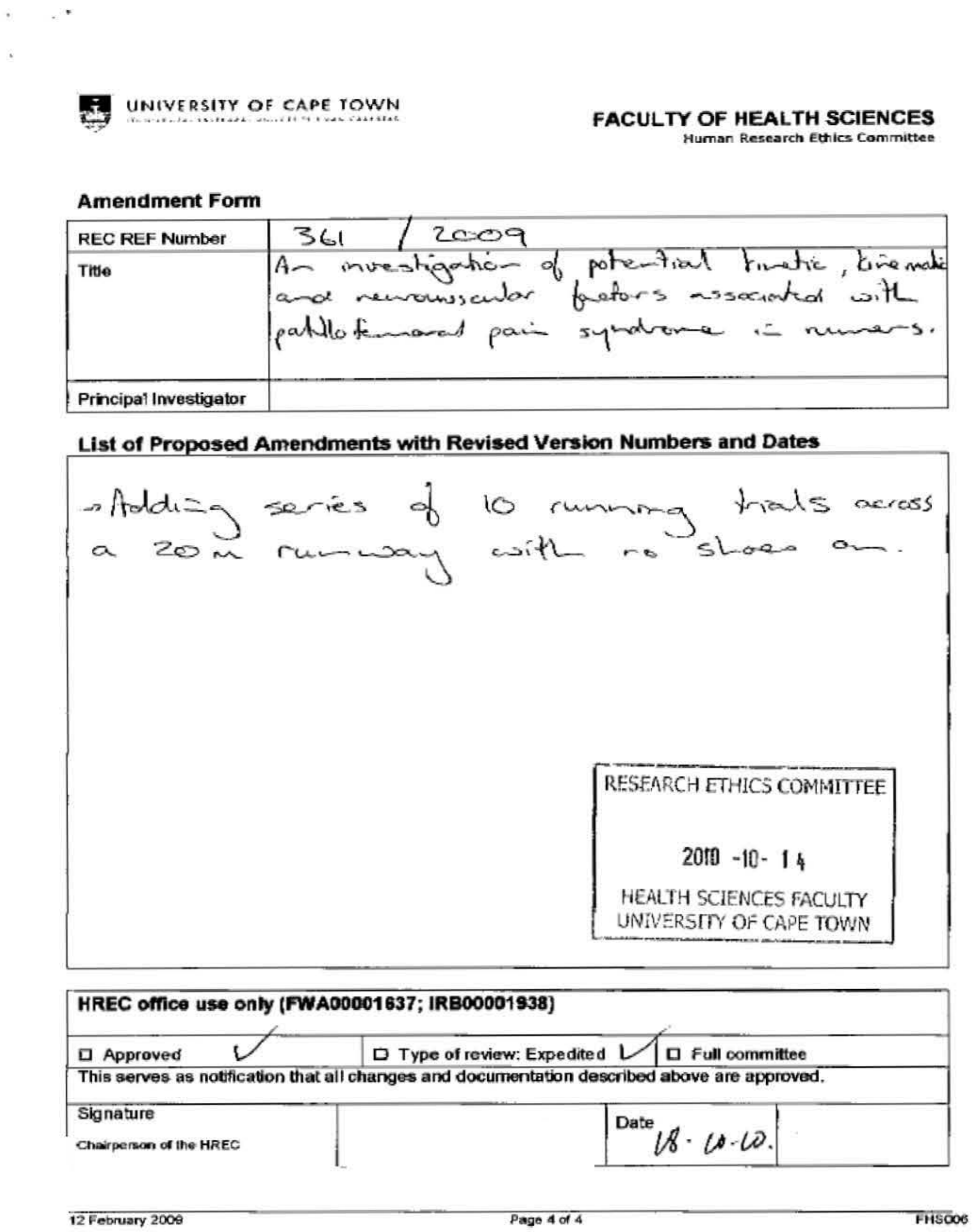
1. FHS016: Annual progress report
2. FHS 006: Protocol amendment
3. Copy of amended documents showing changes in italics and bold
4. "Clean" copy of amended documents
5. General motivation for amendments.

RESEARCH ETHICS COMMITTEE
2010 -10- 14
HEALTH SCIENCES FACULTY
UNIVERSITY OF CAPE TOWN

HREC office use only (FWA00001637; IRB00001938)

☐ Approved ✓	This serves as notification of annual approval, including all documentation described above.		
☐ Not approved	See attached comments.		
Type of review	☐ Expedited ✓	☐ Full committee	
Expiry date	15 OCTOBER 2011		
Signature Chairperson of the HREC		Date	15.10.10

25 February 2009 Page 5 of 5 FHS016



UNIVERSITY OF CAPE TOWN

FACULTY OF HEALTH SCIENCES
Human Research Ethics Committee

Amendment Form

REC REF Number	361 / 2009
Title	An investigation of potential kinetic, kinematic and neuromuscular factors associated with patellofemoral pain syndrome in runners.
Principal Investigator	

List of Proposed Amendments with Revised Version Numbers and Dates

→ Adding series of 10 running trials across a 20 m runway with no shoes on.

RESEARCH ETHICS COMMITTEE
2010 -10- 14
HEALTH SCIENCES FACULTY
UNIVERSITY OF CAPE TOWN

HREC office use only (FWA00001637; IRB00001938)

☐ Approved ✓	☐ Type of review: Expedited ✓	☐ Full committee

This serves as notification that all changes and documentation described above are approved.

Signature Chairperson of the HREC		Date 18·10·10.

12 February 2009 Page 4 of 4 FHS006

UNIVERSITY OF CAPE TOWN

FACULTY OF HEALTH SCIENCES
Human Research Ethics Committee

Annual Progress Report

REC REF Number	361/2009
Title	An investigation of potential kinetic, kinematic and neuromuscular factors associated with patellofemoral pain syndrome in runners
Principal Investigator	Chris Allan

List of documentation

HREC office use only (FWA00001637; IRB00001938)

☑ Approved	This serves as notification of annual approval, including all documentation described above.	
☐ Not approved	See attached comments.	
Type of review	☑ Expedited	☐ Full committee
Expiry date	28 OCTOBER 2012	
Signature Chairperson of the HREC		Date 26/10/2011

25 February 2009 Page 5 of 5 FHS016

Appendix 8: Information to participants regarding PFPS

Patellofemoral pain syndrome

Patellofemoral pain syndrome (PFPS), also known as runner's knee or anterior knee pain, is a syndrome characterised by pain or discomfort seemingly originating from the contact of the posterior surface of the patella (back of the kneecap) with the femur (thigh bone). It is the most frequently encountered diagnosis in sports medicine clinics.

The cause of PFPS often results from prolonged repetitive compressive or shearing forces on the PF joint, sustained during running or jumping. The result is thinning and softening or irritation and inflammation of the articular cartilage and synovial linings under the patella or on the medial or lateral femoral condyles. Secondary causes of PFPS are fractures, internal knee derangement, OA of the knee and bony tumors in or around the knee.

Specific populations at high risk of primary Patellofemoral Pain include runners, cyclists, basketball players, young athletes and females especially those who have an increased angle of genu valgus (aka "Q-Angle" or commonly referred to as "knock-knees").

Typically patients complain of localised anterior knee pain which is exacerbated by sports, walking, sitting for a long time, or stair climbing. Descending stairs may be worse than ascending. Unless there is an underlying pathology in the knee, swelling is usually mild to nil.

Treatment

Exercises

Quadriceps strengthening is commonly suggested because the quadriceps muscles help to stabilise the patella. Proper form is very important. Strength of the hip muscles has also been suggested to improve recovery. Inflexibility has often been cited as a source of patellofemoral pain syndrome. Stretching of the hip, hamstring, calf and iliotibial band may help restore proper biomechanics. Furthermore, the use of a foam roller may help to add flexibility and relieve pain from sore or stiff muscles in the leg.

Rest

Patellofemoral pain syndrome may also result from overuse or overload of the PF joint. For this reason, knee activity should be reduced until the pain is resolved. Those with pain originating from sitting too long should straighten the leg or walk periodically. Those who engage in high impact activity such as running should consider a nonimpact activity such as swimming or aerobics on an elliptical machine.

Ice

To reduce inflammation, ice can be applied to the PF joint after an activity. The ice should be kept in place for 10 to 15 minutes.

Taping

In addition to physical therapy, tape could be used to stabilise the patella. This will not correct the underlying source but may prevent further injury and assist in pain free rehabilitation. For this reason, they should be used in conjunction with physical therapy.

Arch support

Low arches can cause overpronation this means the feet roll inward too much increasing the Q angle and genu valgus. Poor lower extremity biomechanics may cause stress on the knees and ultimately patellofemoral pain syndrome. Stability or motion control shoes are designed for people with pronation issues. Arch supports and custom orthotics may also help to improve lower extremity biomechanics.

Appendix 9: Research feedback.

Dear ________________________

Thank you for assisting me with my research on the biomechanics of patellofemoral pain. Below are the results from your anthropometrical tests performed on the day, as well as some feedback about your gait analysis.

Once the dissertation has been finalised I will send you a summary of the results found. If you have any further queries, please don't hesitate to contact me on callan@wam.co.za.

Thank you again,

Chris Allan.

Name:______________________________ Date of test:____________

Weight: ____________Height: _________________

Q- Angle: ________________ (This is a measure of the angle of your knee influencing the position of your patella and may have an impact on Patellofemoral pain. Normal values are as follows: Males: 11.2°±3.0°; Females: 15.8°±4.5°)

Skinfold Measurements:

Anatomical Areas	Value
Triceps	
Biceps	
Subscapularis	
Supra iliac	
Abdomen	
Thigh	
Calf	
Sum of skin folds	
Percentage body fat	

Gait assessment: ____

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Appendix 10: Figures of significant results

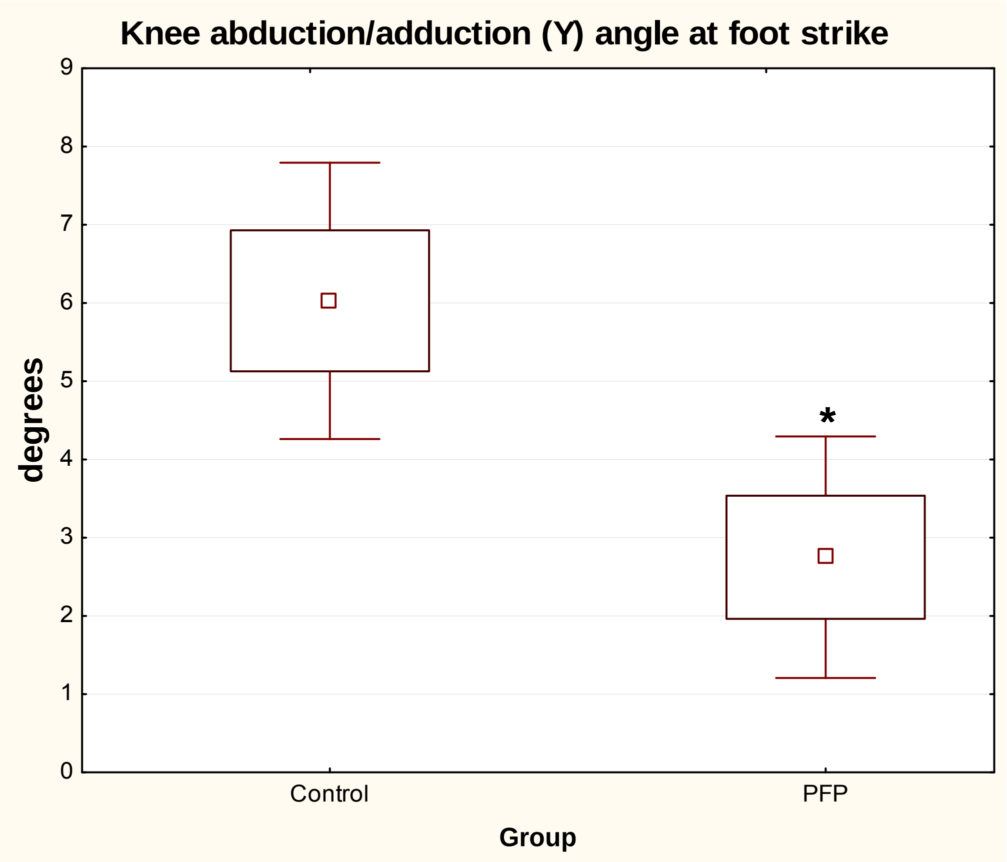

Figure 1: Knee abduction/adduction (Y) angle at foot strike of participants in the PFPS and control groups. Data are expressed as mean ± standard error (SE).

** p = 0.01*

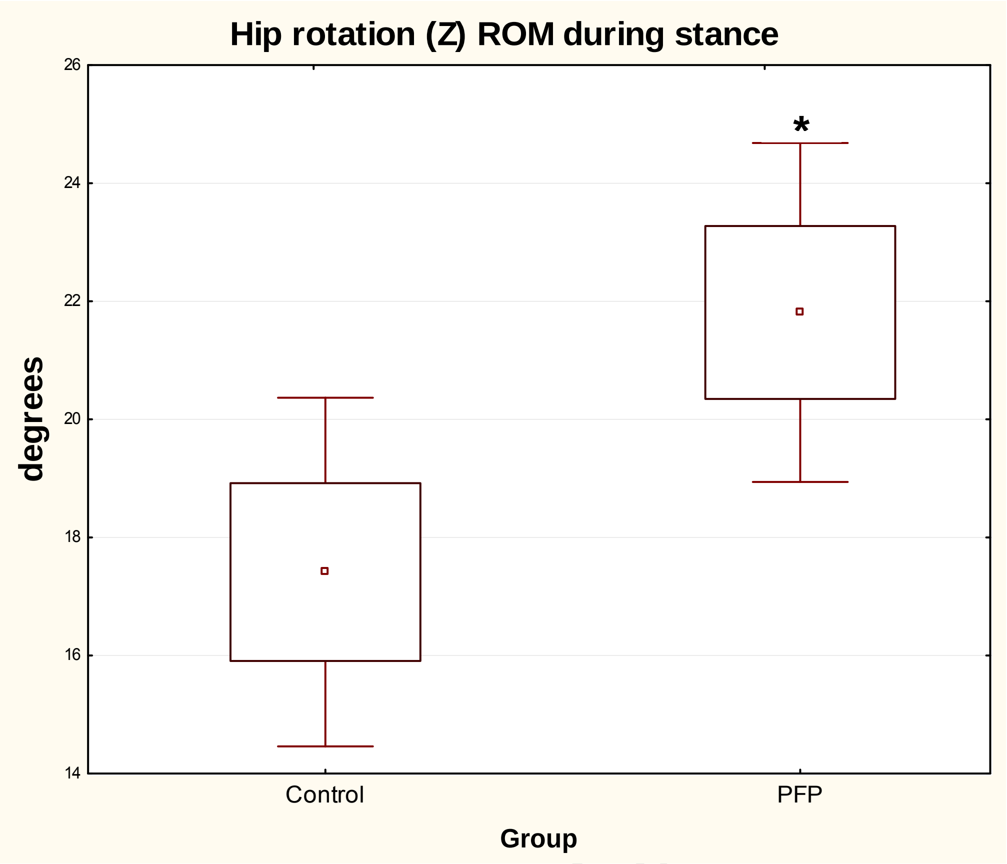

Figure 2: Hip rotation (Z) range of motion during stance phase of participants in the PFPS and control groups. Data are expressed as mean ± SE.

** p = 0.046*

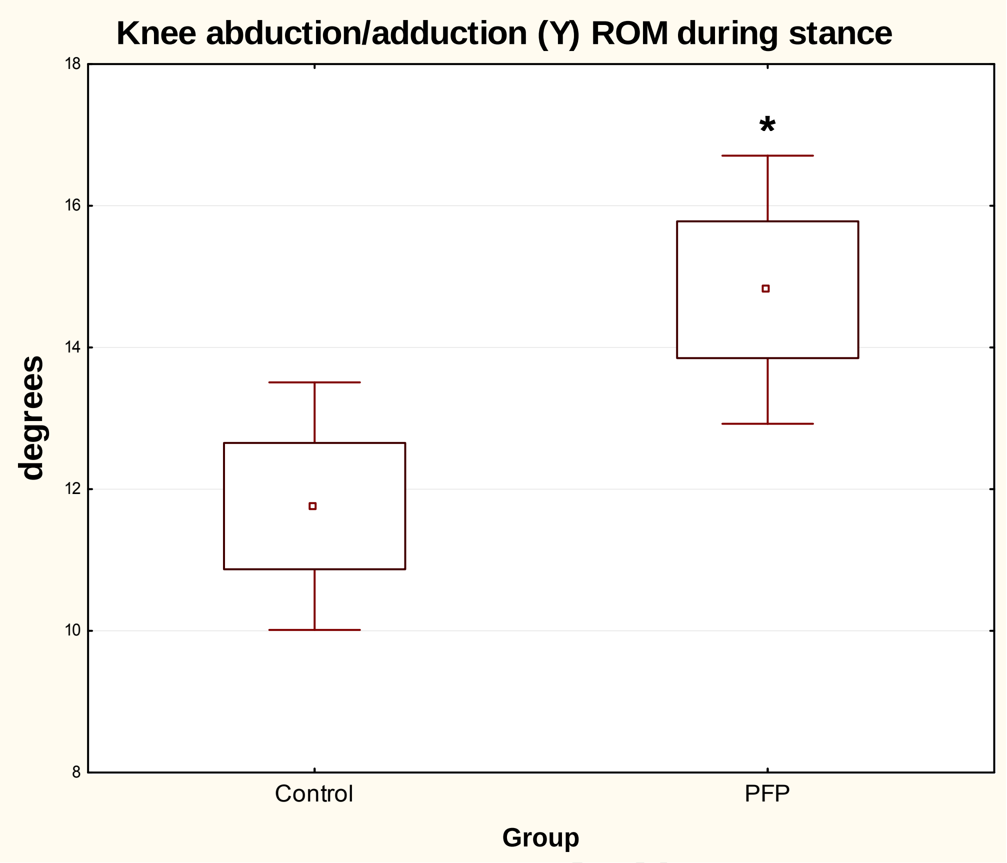

Figure 3: Knee abduction/adduction (Y) range of motion during stance phase of participants in the PFPS and control groups. Data are expressed as mean ± SE.

$p = 0.03$

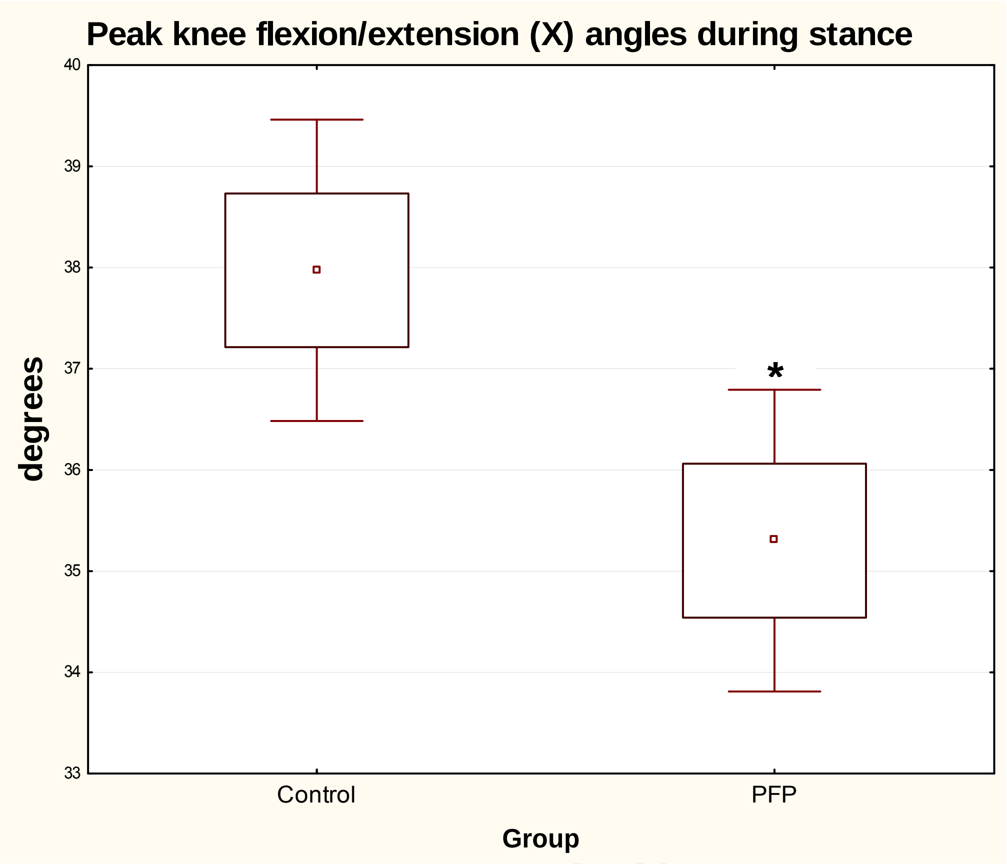

Figure 4: Peak knee flexion/extension (X) range of motion during stance phase of participants in the PFPS and control groups. Data are expressed as mean ± SE.

** p = 0.02*

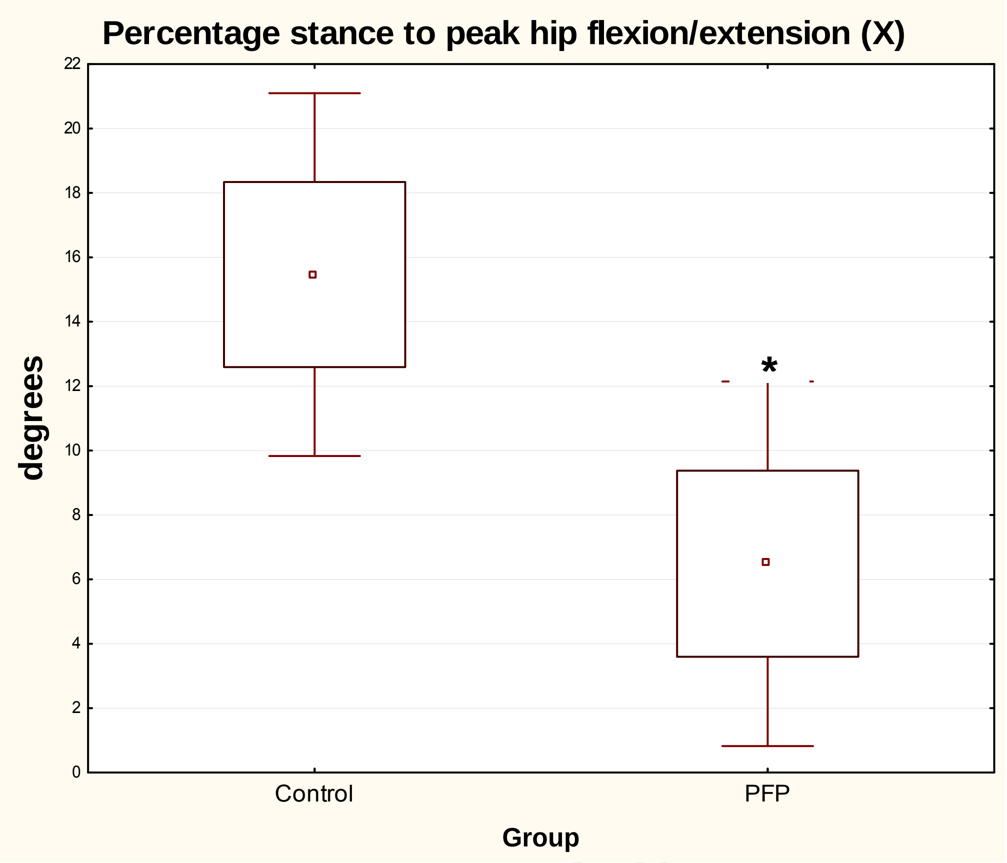

Figure 5: Percentage of stance phase to peak flexion/extension (X) hip angles of participants in the PFPS and control groups. Data are expressed as mean ± SE.

**** p = 0.04***

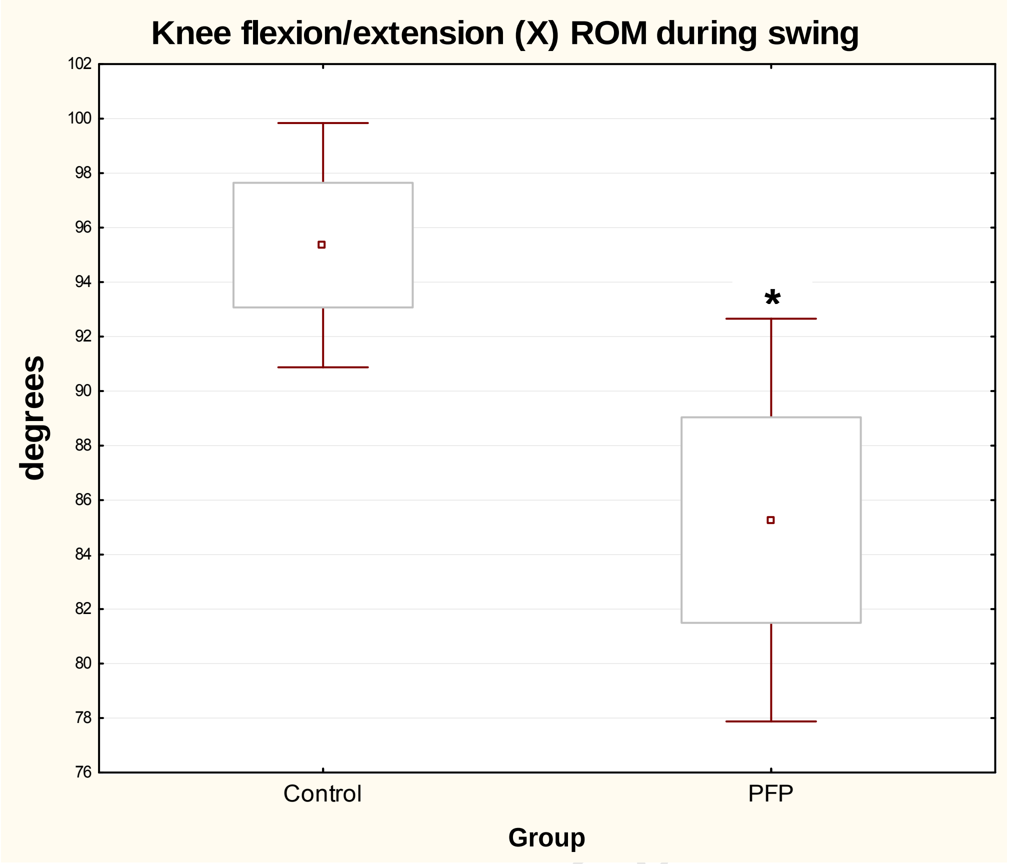

Figure 6: Knee flexion/extension (X) ROM angles during the swing phase of participants in the PFPS and control groups. Data are expressed as mean ± SE.

**** p = 0.03***

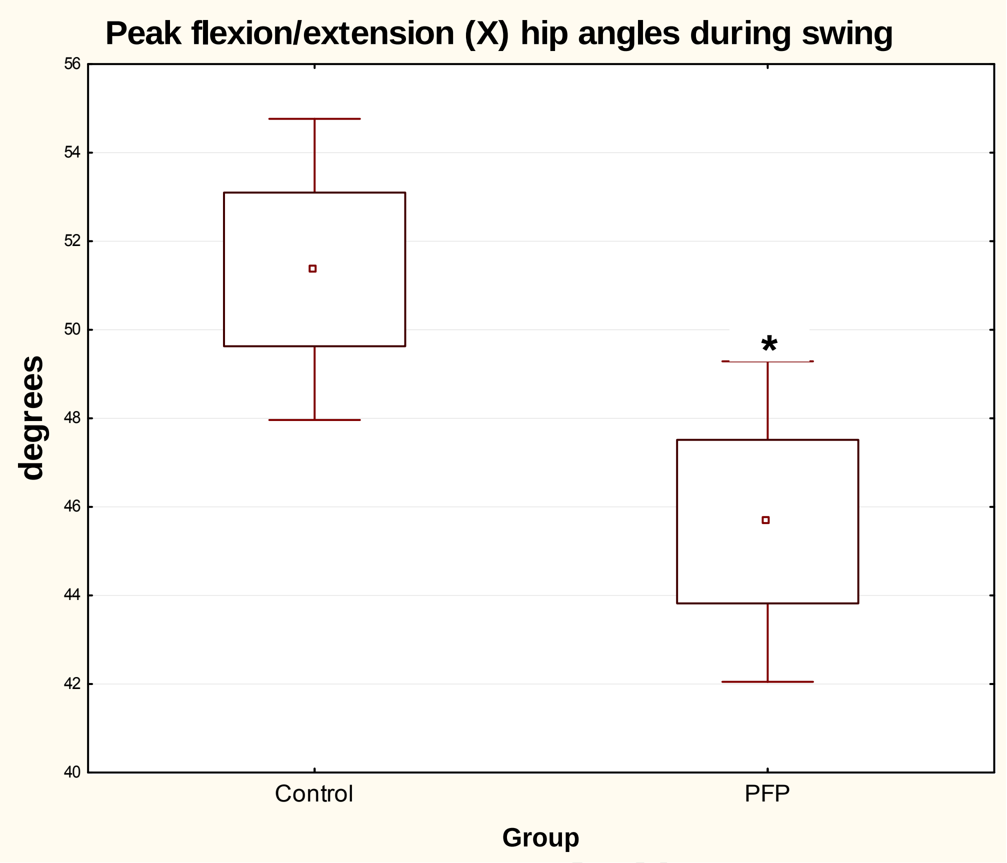

Figure 7: Peak hip flexion/extension (X) angles of participants during the swing phase in the PFPS and control groups. Data are expressed as mean ± SE.

**** p = 0.03***

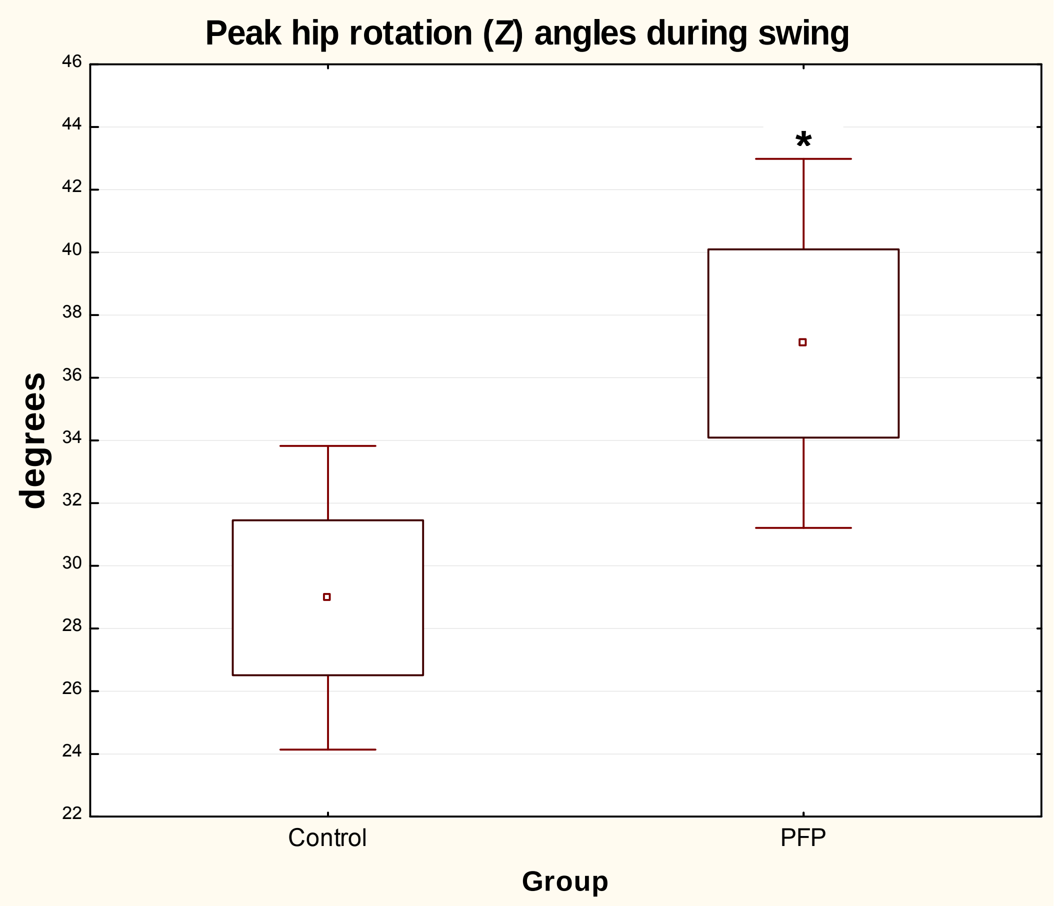

Figure 8: Peak hip rotation angles (Y) of participants during the swing phase in the PFPS and control groups. Data are expressed as mean ± standard error (SE).

** p = 0.046*