Gait kinematics and risk factors for overuse anterior knee pain

By

Andrew K Wills

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Abstract

Overuse anterior knee pain (AKP) is precipitated by activity and affects up to 30% of young and active populations. There is little empirical evidence for the multitude of cited risk factors for the condition and a lack of prospective studies. The main aim of this PhD was to examine the role of gait kinematics as a risk factor for AKP.

The first study examined variables other than gait that may need to be controlled or statistically adjusted for in future studies to avoid masking true risk factors or effects. A prospective study of military recruits was undertaken into the effect of prior activity levels, aerobic fitness and social and medical history on the development of AKP. The incidence of AKP was high (8.6%; 95% CI: 6.8-10.4) despite the short 12-week exposure to training. Heavy smokers (odds ratio (OR): 6.37) and individuals with a previous ankle injury (OR: 2.48) had an increased risk of AKP that was independent of lifestyle factors.

The association between 3D gait kinematics and patellofemoral pain syndrome (PFPS) was then explored prospectively. Principle components analysis was applied to reduce the gait data into its main factors and multivariate logistic regression was used to explore the association between these factors and PFPS. Three factors from treadmill running explained 47% of the variance between individuals who developed PFPS and those that remained injury-free. These factors contained increased hip and tibial internal rotation, increased hip adduction and decreased knee internal rotation during stance. These results contradicted findings from case-control studies.

The association between variability in gait movement patterns and PFPS was assessed using the continuous relative phase method. The main risk factor was reduced inter-stride variability in the joint coordination relationships that contained tibial rotation.

The main limitation of the gait study was the small sample size of the PFPS group (n=7). A study was thus undertaken to cross-validate the findings in a new sample with PFPS. This new sample was captured using a 3-year follow up study of the original gait cohort. The results were not replicated in the new PFPS group, and there were no other gait characteristics correlated with PFPS. The lack of validation was attributed to differences in the symptom-complex between the case groups of the two studies.

To date, all published evidence for an association between gait and AKP originates from case-control studies. The key issue with this design is inferring the correct temporal sequence of a finding. Thus, to assess the effect of PFPS on gait and inform the interpretation of these studies, a repeated measures study of 6 subjects before and after the onset of PFPS was undertaken. Despite the mild symptoms of the group and the absence of pain during testing, the subjects showed some subtle gait inhibition post onset of PFPS. This questions the use of the case-control study to validly quantify risk factors in gait.

Future research should cross-validate the significant risk factors found in these studies, explore other potentially salient variables such as patellofemoral alignment and examine the causes of these risk factors. It is hoped that such work will benefit the prevention and treatment of AKP.
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<th>Description</th>
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<tr>
<td>AKP</td>
<td>Insidious or overuse onset of anterior knee pain</td>
</tr>
<tr>
<td>ASIS</td>
<td>Anterior superior iliac spine</td>
</tr>
<tr>
<td>BMI</td>
<td>Body mass index</td>
</tr>
<tr>
<td>BW</td>
<td>Body weight</td>
</tr>
<tr>
<td>CAST</td>
<td>Calibrated anatomical systems technique</td>
</tr>
<tr>
<td>CI</td>
<td>Confidence interval</td>
</tr>
<tr>
<td>CMC</td>
<td>Coefficient of multiple correlation</td>
</tr>
<tr>
<td>CMS(R)</td>
<td>Common military training syllabus for army recruits</td>
</tr>
<tr>
<td>CRP</td>
<td>Continuous relative phase</td>
</tr>
<tr>
<td>CT</td>
<td>Computerised tomography</td>
</tr>
<tr>
<td>DMS</td>
<td>Daily mean subtraction (used to adjust offsets in joint angles)</td>
</tr>
<tr>
<td>DOF</td>
<td>Degrees-of-freedom</td>
</tr>
<tr>
<td>EMG</td>
<td>Electromyography</td>
</tr>
<tr>
<td>FIQ</td>
<td>Functional index questionnaire (Kujala et al., 1993)</td>
</tr>
<tr>
<td>GAGS</td>
<td>Glycosaminoglycans</td>
</tr>
<tr>
<td>GRF</td>
<td>Ground reaction force</td>
</tr>
<tr>
<td>HH</td>
<td>Helen Hayes</td>
</tr>
<tr>
<td>ICC</td>
<td>Intraclass correlation coefficient</td>
</tr>
<tr>
<td>IPAQ</td>
<td>International physical activity questionnaire</td>
</tr>
<tr>
<td>ISBSV</td>
<td>Inter-stride belt speed variation</td>
</tr>
<tr>
<td>KMO</td>
<td>Kaiser-Meyer-Olkin statistic</td>
</tr>
<tr>
<td>LL</td>
<td>Log likelihood</td>
</tr>
<tr>
<td>LOA</td>
<td>95% Limits of agreement</td>
</tr>
<tr>
<td>PCA</td>
<td>Principle components analysis</td>
</tr>
<tr>
<td>PFPS</td>
<td>Patellofemoral pain syndrome (Thomee, 1999)</td>
</tr>
<tr>
<td>RMS</td>
<td>Root mean square</td>
</tr>
<tr>
<td>ROC</td>
<td>Receiver operator characteristic</td>
</tr>
<tr>
<td>ROM</td>
<td>Range of movement</td>
</tr>
<tr>
<td>RR</td>
<td>Relative risk ratio</td>
</tr>
<tr>
<td>RSA</td>
<td>Roentgen stereophotogrammetric analysis</td>
</tr>
<tr>
<td>SD</td>
<td>Standard deviation</td>
</tr>
<tr>
<td>SE</td>
<td>Standard error</td>
</tr>
<tr>
<td>STA</td>
<td>Soft tissue artefact</td>
</tr>
<tr>
<td>2D</td>
<td>Two dimensional</td>
</tr>
<tr>
<td>3D</td>
<td>Three dimensional</td>
</tr>
<tr>
<td>VAS</td>
<td>Visual analogue scale (Pain)</td>
</tr>
<tr>
<td>VC</td>
<td>Vector coding</td>
</tr>
<tr>
<td>VL</td>
<td>Vastus lateralis</td>
</tr>
<tr>
<td>VMO</td>
<td>Vastus medialis obliquus</td>
</tr>
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</table>
Chapter 1

Relevance, aims and scope of the thesis

1.1 Relevance

The benefits of exercise to health, fitness, and social and mental wellbeing are well known. In the last few decades this has fuelled participation rates, and recent data suggest that more than half of all Europeans now take part in some form of activity (Hayes et al., 2002). Running has become one of the most common leisure activities and whilst the benefits are undisputed, a frequent side effect is musculoskeletal injury. The annual incidence of injury among the running population has been reported as 48-52% (Macera et al., 1989; Walter et al., 1989). The economic cost of all musculoskeletal injury in the US was estimated at $149.4 billion in 1992, which equated to 2.5% of GDP (Yelin and Callahan, 1995).

The knee is the most frequent site of injury, in a retrospective study more than 90% of runners reported a knee injury over a two-year period (Jacobs and Burton, 1986). Among knee injuries, insidious or overuse onset of anterior knee pain is the most common complaint (Taunton et al., 2002). More than one-third of adolescents experience this pain (Fairbank et al., 1984) and it affects 5-15% of military recruits over a time scale as short as 12 weeks (Cowan et al., 1996; Kaufman et al., 1999; Jones et al., 1993; Milgrom et al., 1991). The pain is functionally limiting, often causing a cessation in activity, and in occupations with minimal fitness standards such as the military, a medical discharge (Gemmell, 2002). The long term outcome is varied, as many as 50-90% of patients will experience recalcitrant pain and a cycle of failed interventions (Stathopulu, 2003).

Prevention and treatment of injury has become the focal point for researchers and clinicians (Murphy et al., 2003). Both are inextricably linked to an understanding of the risk factors. A mechanical model has typically been ascribed to the pathogenesis of overuse injuries and in particular anterior knee pain, where the stimulus is thought to be
cumulative stress below the threshold for acute tissue failure (Heino and Powers, 2002; Hreljac et al., 2000a, Powers, 2003; Winter, 1992). The literature suggests that anterior knee pain is caused by a complex multitude of risk factors and inciting mechanisms. However, there is little evidence for many of these anecdotally cited factors, and the level of clinical evidence that does exist is predominantly retrospective and low (Yasuf et al., 1998). There is thus a requirement for well controlled multivariate prospective studies that explore risk factors for anterior knee pain.

1.2 Aims and objectives

The main objective of this PhD was to examine the role of movement kinematics during gait as a risk factor for overuse anterior knee pain. However, given the multifactorial nature of the condition, a multivariate approach was adopted and potential covariates for injury other than gait were also studied.

The investigation was split into 2 phases. The phase I work consists of explorative and hypothesis generating prospective studies to examine potential risk factors for anterior knee pain. These studies were designed to direct the Phase II work. The aims of phase I were to:

- Examine the epidemiology of anterior knee pain and explore variables other than gait biomechanics which may be important covariates to consider in further work.

- Explore the role of gait kinematics as a risk factor for overuse anterior knee pain.

The aim of the Phase II work was to:

- Examine the validity of the findings from the Phase I work
1.3 Scope

1.3.1 Literature review

Chapter 2 contains the background information necessary for a critical understanding of research into risk factors for anterior knee pain. This section covers the biomechanics, pathophysiology and classification of anterior knee pain.

The evidence for the cited risk factors for anterior knee pain is examined in Chapter 3. Weaknesses and limitations of previous research are discussed and the approach for this PhD programme is given. The direction of the Phase I work was derived from the literature reviewed in Chapters 2 and 3 and the rationale for the approach is given at the end of chapter 3. These chapters also form the basis for the approach taken to case classification.

1.3.2 Phase I studies

The Phase I studies used a cohort of subjects undergoing military training. This offered a controlled environment to study risk factors prospectively. To satisfy issues concerning feasibility and validity, the studies were restricted to male cohorts. Unless sufficiently controlled for, gender differences such as the gynacoid pelvis could confound findings from mixed-gender studies, and given the prospective approach, to adjust for such differences would have required unfeasible sample sizes.

The first Phase I clinical study is detailed in Chapter 4. This was a prospective cohort study into the effect of rudimentary anthropometrics, physical fitness, social characteristics (smoking and alcohol intake), previous musculoskeletal injury and pre-enlistment training history on the development of overuse anterior knee pain.

Chapter 5 details the justification for the methods used in the second clinical study into gait kinematics and anterior knee pain. This section covers the choice of kinematic model to derive joint angles, the mode of ambulation and protocol adopted, the signal processing and normalisation procedures and a study to determine the between-day reliability of the protocol.
The second clinical study of the Phase I work is detailed in Chapter 6. This reports the results of a prospective cohort study into the effect of gait kinematics on the development of patellofemoral pain syndrome (PFPS). PFPS is a diagnosis under the umbrella of the symptom complex of anterior knee pain. This study used factor analytic methods and logistic regression to explore the nature and strength of the association of these variables with PFPS.

Recent research has examined the association between inter-stride joint coordination variability and anterior knee pain (Hamill et al., 1999; Heiderscheit et al., 2002). These studies have applied methods such as the continuous relative phase to quantify movement variability, and have found patterns of reduced variability in patients with AKP. However, these data are from case-control studies and so it is uncertain whether these results are a cause or effect of pain. This potentially important factor was investigated in Chapter 7, where the prospective gait data were analysed using the continuous relative phase and vector coding method to examine the relationship between inter-stride joint coordination variability and PFPS.

1.3.3 Phase II studies

The phase I studies found a number of factors in an individual’s gait that were suggested to be risk factors for overuse anterior knee pain. The original intention was to design an intervention study to examine the effect of modifying these factors on the treatment efficacy of anterior knee pain. In order make a valid correlation between treatment outcome and gait, a requisite is that a subject with anterior knee pain can undertake gait that is not compensatory due to pain. Pilot work was unable to validate this assumption.

It was thus decided to cross-validate the results from the phase I work using a new sample of subjects with PFPS. Chapter 8 describes a 3-year follow up study of the control group from the original prospective study to establish a new anterior knee pain group. The gait characteristics of this group were cross-validated with the risk factors found in the phase I studies.

The requirement for a large sample size to capture a case group of sufficient sample size has prohibited the use of the prospective cohort study for investigations into gait
kinematics and injury, and thus to date all published studies have used the case-control design. However, it is not possible to determine the direction of an association that is found using a case-control study because of the unknown effect of injury and/or pain on gait. Chapter 9 follows up the pilot data mentioned earlier and reports the results from a preliminary study into the effect of anterior knee pain on gait. Kinematic data are presented from 6 subjects before and after the onset of anterior knee pain. It was thought that this preliminary study may provide information useful for interpreting the case-control studies into dynamic biomechanical risk factors and injury.

The thesis is summarised in Chapter 10, the relevance, contribution and applications of the work are discussed and suggestions are made for future research.
Chapter 2

Anterior knee pain: Biology, biomechanics and classification

2.1 Introduction

Anterior knee pain, in its literal sense, is a description of symptoms occurring at the front of the knee. At a basic level it is important to separate the acute from the insidious onset of anterior knee pain. Beyond this, is a wealth of literature and clinical opinion detailing different classification systems that are most notable for their lack of agreement. The main hindrance to clearly defined and agreed diagnostic criteria for anterior knee pain is the poorly understood pathology and aetiology. An understanding of the biology and functional biomechanics of the patellofemoral joint and surrounding anatomy is therefore fundamentally important for evaluating and conducting research in this field.

2.2 General patellar biomechanics

2.2.1 Patellar Function

The patella is a sesamoid bone encased within the quadriceps muscle. It is also a common feature in birds and mammals. Interestingly, an evolutionary paradox illustrated by the absence of patellae in the kangaroo, an animal with one of the most effective knee extensor mechanisms, meant it was once considered a regressive feature in humans (Reid, 1993). However, it is now known to afford a considerable mechanical advantage. Biomechanical analysis has shown that through an extension of the patellar tendon moment arm, the patella increases the effective quadriceps force by as much as 50% (Hungerford and Barry, 1979).
The forces that act through the patellofemoral joint are impressive. During weight bearing activities such as running and squatting, knee flexion is accompanied by a posterior shift in the point of application of the ground reaction force relative to the tibiofemoral joint centre, which magnifies the external knee flexor moment. Despite being countered by the added leverage afforded by the patella, these external moments must be overcome by a disproportionate increase in quadriceps force, which increases the stress through the patellofemoral joint. Patellofemoral joint stress is further compounded with increasing knee flexion as the orientation of the patellar tendon and quadriceps force becomes more normal to the patellofemoral joint surface, causing larger joint reaction forces (figure 2.1). These reaction forces are as high as 1-1.5 times body weight (BW) during walking (Nissell, 1985) and up to 7 times BW during running (Scott and Winter, 1990) and occur in a cyclic manner during everyday activities. This example illustrates another important function of the patellofemoral joint, namely, attenuating and distributing compressive forces.

![Figure 2.1 The effect of knee flexion on the patellofemoral joint reaction force (PFJRF) (adapted from Primal pictures 2003).](image)

2.2.2 Anatomical features

Whilst the morphology of the patella is highly variable between individuals, the medial and lateral facets are easily identified. These surfaces articulate with the medial and lateral femoral condyles. Congruency to the joint is augmented by a longitudinal ridge that separates the facets and centralises the quadriceps force, and by a thick covering of hyaline cartilage. Both facilitate a gliding action during knee flexion and extension.
2.2.3 Joint action

Patellofemoral action is a full six degree-of-freedom (DOF) movement (Ahmed et al., 1999; Lin et al. 2003). The patella displays relatively consistent patterns of movement between subjects in the sagittal plane. During knee extension from 90-0°, the quadriceps muscle pulls the patella through the femoral groove resulting in approximately 50mm of posterior translation, 35mm of anterior translation and 60° of extension rotation (Ahmed et al., 1999). The movement of the patella has been described as a gentle lateral to medial curve in the frontal plane during knee extension (Hungerford and Barry, 1979). However, the translations and rotations in the frontal and transverse planes are affected by a complex multitude of forces and are less consistent across subjects (Bull et al., 2002). This is probably one reason why these movements are considered more clinically important.

2.2.4 Contact area

A general description of patellofemoral contact patterns can be described. At full knee extension, the patella rests proximal to the trochlea, it then moves into the trochlea making contact at approximately 20 degrees of knee flexion. Due to the internal femoral rotation that accompanies knee flexion, initial contact is normally made on the lateral facet. The patella is then compressed into the groove where contact is made with the inferior medial and lateral facets. By 45 degrees the middle band of the patella is in contact and at 90 degrees the contact area has shifted to the superior portion of the patella (Hungerford and Barry, 1979).

From 30-90 degrees of knee flexion, the contact area of the patellofemoral joint increases from 20mm² to 47mm² (Hungerford and Barry, 1979). Further, the cartilage is thicker in the superior patellar region (Huberti and Hayes, 1984). These two features accommodate the high joint reaction forces that accompany knee flexion.

2.2.5 Cartilage: Biochemical and biomechanical properties

The hyaline or articular cartilage of the retro-patellar surface has unique properties for withstanding the complex loads induced by patellofemoral joint action. Cartilage has been implicated in a disease model for anterior knee pain so it is important to understand these properties.
Chapter 2. Anterior knee pain: Biomechanics, biology and classification

The biomechanical properties of cartilage are largely a product of the biochemistry of the extracellular matrix. The ground substance is composed of approximately 70% water, 12-21% collagen (mainly type II), and 3-12% proteoglycan aggregates (Martin et al., 1998). The proteoglycans are large units containing branches of core proteins that serve as attachment sites for negatively charged glycosaminoglycans (GAGS) such as chondroitin sulfate. The negatively charged GAGS repel the core protein branches giving the aggrecan a large bottle brush structure. Importantly, the GAGS also make the proteoglycan aggregates highly hydrophilic. The ability of cartilage to withstand high compressive loads is due to the highly repellent protein branches and the hydrophilic properties of the proteoglycans. Under compression water is forced out of the cartilage under the electrical tension created by its attraction to water, this provides the cartilage with visco-elastic properties (Radin et al., 1970).

The organisation of the extracellular matrix provides cartilage with its anisotropic properties that enable it to perform its different functions. At a basic level, three areas can be distinguished. At the articular surface, the lamina splendens comprise very fine collagen, orientated in a parallel manner and thereby resistant to high shear forces. The middle layer contains more randomly arranged bundles of collagen, these strengthen its resistance to shear and provide force absorption. The basal layer is calcified next to the subchondral bone and has perpendicular orientated collagen fibres. These are thought to act as a transition zone, graduating the elastic modulus from the cartilage to the subchondral bone (Schinagl et al., 1996) and giving force absorption and force transfer properties.

2.3 Patellofemoral joint stability

The anatomy, kinematics and joint contact mechanics of the patella that have been discussed so far are relatively generic, and the descriptions are only representative of the mean in a healthy population. However, there is some variability, and pertinent to the patellofemoral joint, even subtle variations can affect the biomechanics and stability of the joint. Stability is concerned with the equilibrium of forces (Hamill and Knutzen, 2003).
was mentioned, the patellofemoral joint has 6 DOF, and is stabilised by a complex multitude of passive and active mechanisms. Generally the more DOF available to a joint the less stable it is (Bell, 1998). Stability is critical to the normal function of the patellofemoral joint because of the magnitude of forces that occur over a small area. This means that even small disturbances in the balance of these forces may cause significant effects on the stress acting through the joint. Figure 2.2 shows an axial MRI scan of a normal patellofemoral joint, the delicately balanced and complex joint congruency is illustrated in this image. Many surgical treatments to the patellofemoral joint such as the lateral retinacular release are based on this premise (Hull et al., 1999). In fact many conservative therapies for anterior knee pain are also aimed at correcting stability, and the concept of stability is frequently alluded to in the medical and scientific literature on anterior knee pain.

![An axial MRI scan of the patellofemoral joint (right limb) illustrating some basic features of the patellofemoral joint, and the poised position of the patella and delicate joint congruency.](image)

**Figure 2.2** An axial MRI scan of the patellofemoral joint (right limb) illustrating some basic features of the patellofemoral joint, and the poised position of the patella and delicate joint congruency.

### 2.3.1 Passive mechanisms

These refer to involuntary mechanisms that may affect joint stability and congruency, such as morphological and anatomical variations.
2.3.1.1 Patellofemoral alignment

There are a number of measurements that describe the position of the patella relative to the femoral groove. Those most studied in relation to anterior knee pain are the medio-lateral translational position in the frontal plane and the patellar tilt angle. Patellar tilt is the orientation of the patella about its supero-inferior axis. Most individuals have a lateral translational alignment and a lateral tilt (Schutzer et al., 1986). However, excessive alignment may cause uneven load distribution on the medial and lateral facets. These can be quantified using the lateralisation distance and patellar tilt angle, obtained from a midpatellar section of an MRI scan or skyline X-ray (figure 2.3).

![Figure 2.3. Patello-femoral lateralisation distance – distance between the medial margin of the patella and the tip of the lateral facet (A); Congruence angle – formed from a line bisecting the sulcus angle and a line joining the nadir of the groove and the apex of the patella (B); Patellar tilt angle – formed between a line on the anterior tips of both femoral condyles and a line from the apex and lateral tip of the patella (C) (In Bull et al., 2002).](image)

Patellar lateralisation and tilt are affected by the peripatellar retinaculum. This is a dense fibrous connective tissue with high tensile strength. It originates deep in the illiotibial band and is attached to the lateral border of the patella. Stiff lateral retinaculum structures have been implicated as a cause of excessive lateral patellar tilt and lateralisation (Pookarnjanamorakot et al., 1998). Accordingly, the flexibility of muscle and tissue such as the tensor fasciae latae and illiotibial tract which contribute to the tension in the lateral retinaculum has been implicated in the aetiology of anterior knee pain (Puniello, 1993; Thomee et al., 1999). The medial retinaculum connects the medial side of the patella directly to the medial femoral epicondyle without muscle tissue attachment and is thus considered a more benign influence.

Whilst the focus of research in this area has been on tilt and lateralisation, it should be noted that the position and orientation of the patella in the frontal plane and antero-
posterior axis may also affect stability. For example, a high resting patella (patella alta) has been associated with patellar subluxation (Reid, 1992), and frontal plane rotation has been shown to affect the patellofemoral contact pressure (Lee et al., 2001).

2.3.1.2 Patello-femoral morphology
Joint congruency may be affected by variations in the bony morphology of the patellofemoral joint. Morphological factors that may cause poor congruency include a shallow femoral groove (Powers, 2000), an abnormally flat retropatellar surface and lateral femoral condyle dysplasia (McNally, 2001). The congruence angle (figure 2.1) is a measure of this characteristic. The shape of the patella may also affect congruency and has been classified into 3 types (Reid, 1992). A type 1 describes patellae with medial and lateral facets of a similar size. Type 2 are most common and describe patellae with a slightly larger lateral facet. Finally, a type 3 describes a patella with a small medial facet and excessively large lateral facet. Type 3 patellae have been implicated in poor joint congruency (McNally, 2001).

2.3.1.3 Tibiofemoral alignment
A measurement associated with patellofemoral tracking disorders and excessive lateral patellar pressure is the Q-angle (Hungerford and Barry, 1979; Fairbank et al., 1984; Mizuno et al., 2001). This is the angle formed from the intersection of two lines in the frontal plane traced from the anterior superior iliac spine to the centre of the patella, and the tibial tubercle to patella. It describes the lateral force vector of the quadriceps when standing in the anatomical position. Typical values are 10-14° for males and 15-17° for females (Hamill and Knutzen, 2003).

The width of the pelvis and the skeletal morphology of the femur and tibia can affect the Q-angle. The high incidence of insidious anterior knee pain in females has been attributed to differences in pelvic width (Lichota, 2003). The transverse alignment of the femoral neck with respect to the femoral condylar axis, termed femoral torsion, is also a determinant of the Q-angle and has been implicated in anterior knee pain (Krivickas, 1997). An internally rotated femoral condyle axis of approximately 12° with respect to the neck has been cited as normal (Kirtley, 2006). Angles approaching 30° are termed antetorsion, and put the distal end of femur in a more internally rotated position, thereby increasing the Q-angle and possibly affecting patellar tracking. Similarly, hip retroversion,
which is a decrease in the angle between the frontal plane of the pelvis and the femoral neck in the transverse plane, also may cause an internally rotated leg. The morphology of the tibia can also be a factor in patellar stability. In particular, excessive lateral torsion of the tibia may affect the Q-angle through a more laterally placed tibial tubercle (Turner et al., 1981). Finally, tibiofemoral alignment in the frontal plane and in particular genu valgum, may change the dynamics of the patellofemoral joint.

2.3.1.4 Foot posture
Most guidelines on the clinical assessment of anterior knee pain include an examination of the foot as a possible component of patellar stability (Reid, 1992). An adducted posture of the forefoot with respect to the rear (varus) has been linked to compensatory pronation, and excessive pronation is suggested to cause increased internal rotation of the tibia during gait (McClay and Manal, 1997), thereby affecting patellar kinematics (Csintalan et al., 2002). Similarly a varus posture of the rear foot is also thought to contribute to increased pronation (Hamill and Knutzen, 2003).

2.3.2 Active mechanisms

2.3.2.1 Quadriceps muscle action
The quadriceps muscle group encases much of the patella and contributes to the dynamic stabilisation of the tibiofemoral and patellofemoral joint. Each component of the quadriceps has fascicles orientated in different directions. In a study of 18 cadaver specimens, the fibre orientation of the vastus lateralis (VL) relative to the rectus femoris in the frontal plane was 35° (sd: 4°) while in the vastus medialis obliquus (VMO) this was 47° (sd: 5°). Based on the muscle component physiological cross sectional area, it was estimated that the vastus lateralis contributes to 38% of the total quadriceps force and the vastus medialis 25% (Farahmand et al., 1998). Thus each component of the quadriceps exerts a different angle of pull on the patella and has a different maximum force capacity. A weakness of the muscles that exert a medial pull may cause an imbalance of forces and cause the patella to track laterally. In particular, the VMO fibres are thought to function as a medial stabiliser of the patella. Some studies have suggested that the VMO can contract independent of the vastus lateralis (Voight and Wieder, 1991). As such, asynchronous muscle activation of the medial and lateral components has been implicated in abnormal patellofemoral tracking and anterior knee pain (Cesarelli et al., 1999; Tang et al., 2001).
2.3.2.2 Tibiofemoral joint

The patella is attached to the tibia and femur via a network of soft tissue, and dynamic MRI has shown that the femur can move with some independence beneath the patella (Powers et al., 2003b). Movement of the tibia and femur can therefore influence the patellofemoral articulation. This forms the basis for why gait and movement dysfunction is implicated in the aetiology of idiopathic anterior knee pain (Reid, 1992).

Tibiofemoral movement occurs in 6 DOF. The main movements occur in the sagittal plane, where depending on the amount of hip flexion, the knee can move through an arc of approximately 145°. The tibia internally rotates during knee flexion and externally rotates during extension. The amount of internal and external rotation permitted through the flexion-extension arc is approximately 30-45°. The knee is well constrained in the frontal plane, although a few degrees of knee abduction and adduction are permitted (Nordin and Frankel, 2001).

Although the muscles about the knee control some of the transverse rotation at the joint, the structure of the joint dictates that internal rotation will tend to accompany knee flexion and external rotation will tend to accompany extension. The lateral femoral condyle is broader, flatter and projects more anteriorly than the medial condyle. Two slightly concave surfaces comprise the tibial plateau, the medial and lateral facet. The shape of the femoral condyles and tibial plateau mean that the lateral side of the joint moves through a greater excursion during knee flexion, causing internal rotation. During non-weight bearing knee flexion, the tibia predominantly slides and translates on the femur, whereas during weight bearing both the tibia and femur may be moving. Internal tibial rotation is also partly caused by subtalar pronation that accompanies ankle dorsiflexion (Hintermann et al., 1994).

2.3.2.3 Hip joint

The hip is a ball and socket joint, it is comprised of the acetabulum of the pelvis and femoral head. It is freely moveable which makes it an important interacting component with the knee extensor mechanism. In the anatomical position, approximately 140° of hip flexion and 15° of extension, 30° of abduction and 25° of adduction, and 90° of external and 70° of internal rotation are typically available (Nordin and Frankel, 2001).
Hip adduction may contribute to an abducted posture of knee in the frontal plane. This has been associated with anterior knee pain (Bailey et al., 2003; Smith et al., 1991), possibly due to an effect on patellar stability. Previous studies have also shown the hip to favour internal rotation during the load acceptance phase of weight bearing activity (Powers et al., 2003b; Sikorski, 1979). There are a few muscles that act only on the femur to control femoral rotation e.g. piriformis and obturator muscles, this gives the femur some independence from patellar movement (Powers et al., 2003b). It is also why the control of hip rotation is suggested to be an important component of normal patellofemoral function (Ireland et al., 2003).

2.3.2.4 Talocrural and subtalar joint

During weight bearing, movement about the foot and ankle can also affect the function of the knee joint. The main joints about the ankle are the talocrural and subtalar joint. The talocrural is a tightly fitted uniaxial hinge joint where the talus is encased by the tibia and fibula. Its principle function is stability, and the predominant movement is dorsi and plantar-flexion, where normal ranges are 20 and 50 degrees respectively (Hamill and Knutzen, 2003).

Since talocrural movement predominantly occurs in one plane, the main function of the subtalar joint is to absorb the rotational stresses acting at the ankle (Lundberg et al, 1989). The subtalar joint is formed by 3 articulating points on the talus and calcaneal. Its axis of rotation is tilted approximately 27-47 degrees vertically from the heel to the toe, and orientated 8-24 degrees medially from the centre of the tibia in the transverse plane. Movements about this joint are called pronation and supination, and occur in three planes resulting in a combination of 3 movements. Pronation is comprised of dorsiflexion, eversion and abduction, while supination is consists of plantar flexion, inversion and adduction (Lundberg et al., 1989). During weight bearing, the majority of subtalar movement is the talus moving on a fixed calcaneus. As a result of this and the orientation of the subtalar axis, some of the subtalar movement is transferred to the tibia causing internal tibial rotation (Hintermann and Nigg, 1994; Reischl et al., 1999). This is considered functional during gait because it accommodates internal femoral rotation. Since the patella is connected to the tibia via the patellar tendon and the tibia articulates with the femur, movement of the tibia, particularly in the transverse plane may affect normal patellofemoral functioning (Lee et al, 2001). As such excessive subtalar movement during
the load acceptance phase of gait has been implicated in the aetiology of anterior knee pain (Hintermann and Nigg, 1998; Powers, 2003a; Thomee et al., 1999; Tiberio, 1987;).

2.3.3 Summary

This section has given an overview of how the movement of joints about the ankle, knee and hip, and the morphological characteristics of the lower extremity can affect the dynamics of the patellofemoral mechanism, these are summarised in Table 2.1. There are other passive factors not contained in this table which may also affect stability. In fact many of those listed could be sub classified, for example patellar shape has been given a number of sub-iterations (Ficat and Hungerford, 1977). Despite this, it is thought that the main factors have been stated and many of the other variables are derivatives of these.

The multiple degrees of freedom of the joints of the lower extremity allow incredible flexibility in the way that movement can be coordinated to achieve a single outcome. It is clear that the passive and dynamic mechanisms for maintaining normal patellofemoral function and support are complex and intricate. Further, the permutations for upsetting this mechanism and causing instability are numerous. This may partly explain why so many aetiological factors have been implicated in anterior knee pain.

2.4 Joint motion during gait

The movements of the tibia and femur in the sagittal plane during walking and running have been well described and so will not be documented here. However, it is relevant to discuss the translational and rotational movements of the tibiofemoral and patellofemoral joints in the frontal and transverse planes in the context of the previous discussion on patellar stability. The studies described here all used intracortical pins to quantify skeletal movement, which is considered the gold standard.
Table 2.1. Factors affecting the stability of the patellofemoral joint

<table>
<thead>
<tr>
<th>Passive</th>
<th>Active</th>
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<tbody>
<tr>
<td>PF Alignment</td>
<td>Muscular Control</td>
</tr>
<tr>
<td>• Medio-lateral tilt</td>
<td>• VMO muscle weakness/ VMO-VL imbalance</td>
</tr>
<tr>
<td>• Medio-lateral position.</td>
<td>• Muscle activation pattern</td>
</tr>
<tr>
<td>• Patellar alta/ infera</td>
<td>Joint movement</td>
</tr>
<tr>
<td>Femoral/ tibial morphology</td>
<td>• Hip joint</td>
</tr>
<tr>
<td>• Sulcus depth/ angle</td>
<td></td>
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<tr>
<td>• Congruence angle</td>
<td></td>
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<tr>
<td>• Femoral antetorsion</td>
<td></td>
</tr>
<tr>
<td>• Lateral tibial torsion</td>
<td></td>
</tr>
<tr>
<td>Patella morphology</td>
<td></td>
</tr>
<tr>
<td>• Patellar type (Wiberg)</td>
<td></td>
</tr>
<tr>
<td>• Retropatellar surface</td>
<td></td>
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<tr>
<td>Soft tissue</td>
<td></td>
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<tr>
<td>• Peripatellar retinaculum</td>
<td></td>
</tr>
<tr>
<td>• Illiotibial band</td>
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<tr>
<td>Tibiofemoral alignment and morphology</td>
<td></td>
</tr>
<tr>
<td>• Q-angle</td>
<td></td>
</tr>
<tr>
<td>• Femoral retroversion</td>
<td></td>
</tr>
<tr>
<td>• Genu valgum/ varum</td>
<td></td>
</tr>
<tr>
<td>Foot posture</td>
<td></td>
</tr>
<tr>
<td>• Rearfoot/ forefoot varus</td>
<td></td>
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</tbody>
</table>

2.4.1 Tibiofemoral motion

2.4.1.1 Walking

A historical study by Levens and colleagues (1944, cited by Ramsay and Wretenberg, 1999) first showed patterns of femoral and tibial internal rotation from foot contact to midstance, and patterns of external rotation from midstance to toe off during walking. Whilst the pattern of movement appears consistent, Reinschmidt et al. (1997a) found relatively large variability in the amplitude in a study of 5 healthy subjects, illustrated by a range of 5-10 degrees.

La Fortune et al. (1992) showed that the knee remains in an abducted posture during stance with very little excursion.
2.4.1.2 Running

Both McClay (1990) (cited by Ramsay and Wretenberg, 1999) and Reinschmidt et al. (1997b) found a pattern of tibiofemoral internal rotation from contact to midstance during running. However, similar to walking, Reinschmidt et al. (1997b) found a large range in the amplitude across subjects (2-9°).

There was little agreement between studies for the abduction-adduction patterns. McClay (1990) found consistent adduction patterns of approximately 6° from foot contact to midstance, however, Reinschmidt et al. (1997b) found patterns of abduction. Even when both studies used the same anatomical coordinate system and reoentgen-stereophotogrammetric (RSA) method to determine the axis orientation there was still dissimilitude. The abduction-adduction angles are sensitive to measurement error due to incorrectly aligned axis, this may explain the conflicting results.

2.4.2 Patellofemoral motion

It is worth outlining the terminology for describing patellofemoral motion as the authors of the skeletal pin studies used different descriptors to that recommended in a later standardisation paper (Bull et al., 2002). Patellar rotation about a medio-lateral axis in the sagittal lane is described as flexion and extension. Rotation about an anterior-posterior axis in the frontal plane is described as internal and external rotation. Patellar rotation about a supero-inferior axis in the transverse plane is described as medial and lateral tilt, where lateral tilt describes the lateral border of the patella rotating towards the femur. Medial and lateral translations are described as shift. These movements also depend on the axes definition but for the purpose of this review this description should be sufficient.

It is generally agreed that the patellofemoral joint displays relatively homogenous patterns of patellar flexion and extension that correspond with knee flexion and knee extension during walking (LaFortune et al., 1992) and running (McClay, 1990 cited by Ramsay and Wretenberg, 1999).

The walking (LaFortune et al., 1992) and running (McClay, 1990) data showed differences in the pattern of patellofemoral internal and external rotation. Patellar internal and external rotation also showed substantial variance between subjects. The walking study found
patterns of internal rotation from foot contact to mid-stance while the running study found 
an initial external rotation pattern. The differences may be partly explained by the different 
modes of ambulation. But also at a more fundamental level, due to the balance of the two 
competing forces that cause patellar rotation. Internal tibial rotation causes internal patellar 
rotation, and contraction of the vastus medialis fibres on the superior medial border of the 
patella causes external patellar rotation. McClay (1990) suggested that differences between 
these two forces may be responsible for the variability between individuals.

During walking and running, the patella remains in a laterally tilted orientation. However, 
in the walking study (La Fortune et al., 1992), two out of five subjects showed a medial tilt 
movement during the first 50% of stance.

There was a general pattern of lateral patellar shift that corresponded with knee flexion 
during stance in the walking study. However, similar to the rotations in the frontal and 
transverse planes, the magnitude and temporal patterns exhibited wide variance between 
individuals (La Fortune et al., 1992). McClay’s study (1990) showed a slightly different 
pattern during the stance phase of running, here there was an initial small medial shift 
followed by a lateral translation.

It is interesting to note that the movements thought to be most critical in the active control 
of patellar tracking such as the transverse plane rotations are those that occur over smaller 
arcs and distances and exhibit the most between subject variability in both pattern and 
relative magnitude of movement.

2.5 Pathomechanics and physiology of anterior knee pain

Pain occurs when there is a disruption to tissue homeostasis, and can be caused by 
mechanical, thermal or chemical stimuli. The knee has a rich nociceptive nerve supply. 
Free nerve endings (FNE) exist in the subchondral bone, synovium, medial and lateral 
retinaculum, muscle, quadriceps tendon and ligament and all have been cited as a source of 
anterior knee pain (Biedert and Sanchis-Alfonso, 2002; Schneider et al, 2000; Sanchis-
Alfonso et al., 2003; Witonski, 1998). However, basic research into the trigger of anterior knee pain is lacking meaning that the pathophysiology is unknown. The traditional pathological model for anterior knee pain has centred on a mechanical mechanism, recently however, two new biological models have been described.

2.5.1 Mechanical model

Articular cartilage is aneural and so is not a source of pain, however, it has been implicated in a disease model for anterior knee pain through disruption to its material and mechanical properties (Radin et al., 1986). The cartilage is susceptible to lesions from hypopressure and hyperpressure (Goodfellow et al., 1976). Pressure increases the permeability of cartilage and is necessary for diffusion, so that chondrocytes are supplied with adequate nutrition and synovial fluid permeates to the surface layer to reduce friction. Surface fissures and degeneration can thus occur due to hypopressure. These lesions are common in the adolescent and middle aged individual (Goodfellow et al., 1976). They tend to be located on the medial and odd facet (areas of infrequent use) in the young and over more diffuse areas in the middle aged. In fact, they are almost a universal finding in older age groups and are often a secondary chance finding (Goodfellow et al., 1976).

Research has shown that even during everyday activities such as walking and climbing stairs, the patellofemoral contact pressure can exceed 4 MPa (Ahmed et al., 1987). A classical animal study first showed the degenerative effects of high repetitive loading on cartilage (Radin, 1970). In vitro studies have shown that high repetitive loading causes fissures in the basal and intermediate cartilage layers (Hunter, 1995; Weightman et al., 1973; Zimmerman et al., 1988). At higher forces, fissures also develop earlier (Zimmerman et al., 1988). In the patient with anterior knee pain, the locations of these lesions have correlated with patellofemoral alignment. For example, Harilainen et al. (2005) found malacic changes and cartilage disruption on the lateral facet in patients with lateral patellar tilt, and central lesions in patients with patellar alta. However, research has found a poor correlation between arthroscopic findings and symptoms of anterior knee pain (Leslie et al., 1978), and the size and depth of lesions and symptoms (Han et al., 2005). It should be noted that this later study used older patients (mean age: 69 years) undergoing knee arthroplasty and so may not be applicable to the overuse anterior knee pain classically
seen in younger athletic samples. Despite this, the role of cartilage degeneration remains unclear.

The mechanical properties of cartilage are affected by the magnitude and frequency of loading. Zimmerman et al. (1988) found that at frequencies above 20Hz, the cartilage does not recover to its resting thickness. It has been suggested that if the mechanical properties of the cartilage are disrupted by supraphysiological loading, then the decreased capacity of cartilage for force distribution and absorption may stimulate the nociceptive fibres of the subchondral plate triggering anterior knee pain (Fulkerson and Shea, 1990; Reid, 1992; Goodfellow et al., 1976). Bone scans have also shown some evidence of increased bone metabolic activity, a stress reaction, in the patellae of patients with pain (Dye and Chew, 1993).

2.5.2 Neural model

Histological studies have shown a greater density of substance P and type IVa free nerve endings and a higher number of vessels in the excised lateral retinaculum of patients with anterior knee pain (Sanchis-Alfonso and Rosello-Sastre, 2003).

Neural proliferation occurs in response to the release of neural growth factor (NGF), which in turn can be initiated by tissue ischemia and hypoxia. NGF also induces the release of substance P, a neurotransmitter. This pattern of hyperinnervation was found about the vessels in the lateral retinaculum. Vascular innervation has been implicated in other injuries, in particular, achilles tendinosis (Kristofferson et al., 2005). Hyperinnervation has been similarly implicated in the pathophysiology of other conditions such as back pain (Coppes et al., 1997). It has been suggested that vascular torsion caused by patellofemoral malalignment and a tightened retinaculum during knee flexion and high impact activities, may cause these brief episodes of ischemia and hypoxia that result in neural proliferation and possibly anterior knee pain (Sanchis-Alfonso and Rosello-Sastre, 2003). These studies have also documented the presence of neuromas and degenerative changes to nerve in the lateral retinaculum. It was speculated that nerves could also be damaged by excessive stress in a shortened retinaculum.

Whilst the neural approach has so far been confined to the retinaculum as the source of pain, the authors note that this doesn’t preclude the possibility that the pain may migrate to
other structures. In fact, other tissues such as the synovium and subchondral bone also have a rich density of Type IVa nerve fibres and substance P (Biedert and Sanchis-Alfonso, 2002). An extension to the theory is the suggestion that lateral retinacular pain may be an early manifestation of excessive lateral patellar pressure and subchondral pain (Fulkerson and Shea, 1990; Reid, 1993). Sanchis-Alfonso and Rosello-Sastre (2003) also suggested that since Type IVa free nerve endings function as mechanoreceptors and are perfuse in the quadriceps musculature, neural damage may be detrimental to the proprioception and stability of the patellofemoral joint.

The studies supporting this model are case-control so it is not possible to determine whether this is a cause or effect of anterior knee pain. In achilles tendonosis, neovascularisation is considered a response to overuse injury (Kristofferson et al., 2005). Further, these studies compared an AKP group against a group with instability and no pain (Sanchis-Alfonso et al., 2001), which is not an ideal normative comparative group. In one study that did compare against a group with no anterior knee pain or malalignment, neural proliferation in the lateral retinaculum was not found (Witonski and Wagrowsko Danielewicz, 1999).

2.5.3 Venous pooling and intraosseous pressure

Two studies have correlated improvement in anterior knee pain symptoms with decreases in the intraosseous pressure of the patella (Miltner et al, 2003; Schneider et al., 2000). Schneider et al. (2000) demonstrated 3 years post operative pain relief in 90% of patients who underwent intraosseous drilling and decompression. This group also reproduced sensations of pain by raising the intrapatellar pressure. It is important to note that this was not a randomised controlled trial and similar symptom relief could have occurred in a control group.

In relation to the cause of the raised intraosseous pressure, the studies reviewed showed that the pressure was associated with the number of veins (Glotzer, 1993) but not the amount of cartilage damage (Glotzer, 1993; Homminga et al., 1995). The angle of knee flexion has been shown to affect intraosseous pressure, so there could also be a mechanical contribution to this mechanism.
2.5.4 **Summary**

It is noticeable that these models are not entirely independent, for example, there seems to be a requirement for mechanical stress in all of them to disrupt the homeostasis of the tissue. Nonetheless, research should be aware of these different pathophysiological processes and also consider factors that affect them as they may interact with different weighting in a manner that contributes to anterior knee pain and masks other true risk factors. For example, factors that affect pain sensitivity (neural model) may lower the required threshold of factors that affect mechanical stress to trigger anterior knee pain.

2.6 **Classification of anterior knee pain**

Given the myriad of potential mechanisms for anterior knee pain, it is not surprising that there is no universally applied set of diagnostic and classification criteria. However, clinicians have attempted to synthesise the concepts described in this chapter to differentiate between types of anterior knee pain. These classification systems have used a combination of pathological, aetiological, surgical and practical approaches.

2.6.1 **Overview of approaches**

Reid’s (1993) classification is based on grading the level of disruption in the articular cartilage. Reid (1993) suggests excluding anterior knee pain caused by internal derangements and specific peripatellar pathology, and classifying all other pain as patellar pain syndrome. It is recommended that this syndrome group is subdivided into 3 categories based on the amount of cartilage damage. Given the lack of correlation between articular damage and pain (Leslie et al., 1978, Han et al., 2005), one may question the relevance of this approach, particularly since it also relies on arthroscopy.

Holmes and Clancy (1998) developed a classification system based on Merchant’s (1988) criteria. This system is primarily based on aetiology and avoids any description of symptoms. At the highest level, patellofemoral pain is defined based on the presence of instability, the presence of malalignment or no observable malalignment. There are then 47
sub categories, which are defined using a combination of aetiological, clinical, pathological and radiographic criteria. For example, there are subcategories for genu valgum, patellar alta and plica pain. The validity of this system has not been determined. A classification system should be supported by evidence to justify the differential diagnosis. As is discussed in the next section, the inter and intra-observer reliability and evidence for many of the criteria in this system are questionable.

Schutzer et al. (1986) described 3 further sub categories of anterior knee pain in the presence of patellofemoral malalignment. Developed from computerised tomography (CT) scans of 45 patients with malalignment, a type 1 classification describes patellar subluxation without tilt, type 2 describes subluxation with tilt and type 3 describes tilt without subluxation. The congruence and patellar tilt angles were used to classify into type. These criteria were developed to direct surgical treatment and are based on a sample with patellar malalignment. Any individuals without a malalignment were excluded from the study. This is a sensible approach in that the classification system is based on an observable aetiology. However, due to the sample used to develop the criteria, it is not known what proportion of individuals with anterior knee pain have a malalignment, and since only 10 controls were used for comparison, it is also not clear what proportion of asymptomatic individuals have a malalignment. This is particularly important since it determines the specificity of the diagnostic criteria.

A more pragmatic approach to the diagnosis of idiopathic anterior knee pain is provided in a recent review paper (Thomee et al., 1999). Similar to Reid (1993), Thomee et al. (1999) excludes pain due to intra-articular and peripatellar pathology, leaving a group with a clinical presentation of anterior knee pain. A diagnosis of patellofemoral pain syndrome (PFPS) is then given should the pain occur during or after at least three of the following: activity, ascending/ descending stairs, squatting, sitting. It is worth noting that the terms anterior knee pain, chondromalacia patella, patellar pain, patellar pain syndrome and patellofemoral pain have all be used synonymously with PFPS (Thomee et al., 1999). The inclusion criteria in Thomee et al.'s (1999) system help exclude the short episodes of anterior knee pain that may be a normal response to unaccustomed exercise. These criteria can be reliably assessed with a battery of clinical tests and a clinical history. This classification does not exclude malalignments, here the author notes that the exclusion of malalignments requires consideration given the scientific evidence (this evidence is
reviewed in section 3.1.4). This approach to diagnosis seems sensible for research where the hypotheses are concerned with a general patellofemoral stress or mechanical model.

There are other papers describing classification systems, however these were omitted because they are essentially derivatives of the four systems described above.

2.6.2 Classification systems used for research

Previous research into anterior knee pain has employed a range of different inclusion and exclusion criteria for case selection (Table 2.2). Additionally, a few studies did not even report the selection criteria (Dillon et al., 1983; Bennett and Stauber, 1986; Caylor et al., 1993; Hamill et al., 1999; Livingston and Mango, 2003). It is important to provide the diagnostic criteria since they are an important factor for comparing results between studies. It is also possible that the different classification criteria are responsible for the variability of findings from anterior knee pain research (Wilk et al., 1998).

In the papers detailed in Table 2.2, the authors gave no rationale for their choice of selection criteria. Further, no pattern could be discerned between the classification criteria employed and the context of the research or the research hypotheses. Given the circumstances of poorly understood aetiology and numerous classification systems, it is not surprising that studies have used different criteria. This is acceptable given that the most appropriate way to differentiate anterior knee pain is not known. Nonetheless, it seems sensible that the criteria should be explicitly stated along with the rationale for the choice in the context of the research hypotheses. The biomechanics and pathophysiological models of anterior knee pain are possibly the main sources for making this decision.

Two criteria have been universally applied to classify anterior knee pain, these are by onset i.e. overuse, and by age. Although it is possible that there is a disease spectrum for anterior knee pain (Stathopulu, 2003), it is important to study relatively homogenous age groups because of the physiological affects of ageing which may alter the aetiology (Goodfellow et al., 1976). At this basic level then, what remains is a collective diagnosis that is a relatively unspecific description of symptoms and aetiology.
### Table 2.2. Examples of AKP case classification criteria used in previous research

<table>
<thead>
<tr>
<th>Author</th>
<th>Design</th>
<th>Purpose</th>
<th>Inclusion Criteria</th>
<th>Exclusion Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bennett and Stauber</td>
<td>Longitudinal single group follow-up</td>
<td>To determine relationship between strength and AKP treatment outcome</td>
<td>PFPS referral from Orthopaedic Surgeon</td>
<td>Acute or previous ligament trauma or fracture to the knee</td>
</tr>
<tr>
<td>Kujala (1986)</td>
<td>Case-control</td>
<td>To examine risk factors for knee exertion injuries.</td>
<td>Excused from military training due to knee exertion injury</td>
<td></td>
</tr>
<tr>
<td>Messier et al. (1991)   &amp; Duffy et al. (2000)</td>
<td>Case-control</td>
<td>To examine biomechanical, anthropometrical, fitness variables on risk of AKP</td>
<td>1. Patellar pain along the medial or lateral joint capsule</td>
<td>1. Plica syndrome</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2. Pain against the underlying femur</td>
<td>2. Patellar tendinitis</td>
</tr>
<tr>
<td>Milgrom et al. (1991)   &amp; Duffey et al. (2000)</td>
<td>Case-control</td>
<td>To examine biomechanical, anthropometrical, fitness variables on risk of AKP</td>
<td>1. Patellar pain along the medial or lateral joint capsule</td>
<td>1. Plica syndrome</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2. Pain against the underlying femur</td>
<td>2. Patellar tendinitis</td>
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<td></td>
<td>3. Tenderness along the medial facet of the patella</td>
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<tr>
<td>Smith et al. (1991)</td>
<td>Longitudinal single group follow-up</td>
<td>To examine the relationship between flexibility and AKP</td>
<td>Diagnosis of PFPS, Osgood Schlatter disease, patellar tendinitis</td>
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<tr>
<td></td>
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<td></td>
<td>2. Consensus of AKP by Physician and therapist</td>
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<td></td>
<td></td>
<td></td>
<td>3. Duration of symptoms &gt; 6 weeks</td>
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<td></td>
<td></td>
<td></td>
<td>4. Insidious onset</td>
<td></td>
</tr>
<tr>
<td>Finestone et al. (1993)</td>
<td>Longitudinal single group follow-up</td>
<td>To determine which factors predict outcome from treatment of PFPS</td>
<td>Pain on compression, tenderness along medial facet</td>
<td>Tendonitis, inflamed plica or other overuse injury</td>
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<td></td>
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<td>2. Physical examination and suggestive history</td>
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<tr>
<td>Callaghan et al. (1994)</td>
<td>Case-control</td>
<td>To examine gait in AKP patients vs healthy injury free controls</td>
<td>History &gt; 2 years</td>
<td>Other causes of pain e.g., referred from hip</td>
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<td></td>
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<td>2. Unilateral; no previous surgery or trauma</td>
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<td>3. Arthroscopy – normal</td>
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<td>4. Radiographic appearance normal</td>
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<tr>
<td>Kannus et al. (1994)</td>
<td>Longitudinal single group follow-up</td>
<td>To determine which factors predict outcome from treatment in patients with PFPS</td>
<td>1. Unilateral symptoms</td>
<td></td>
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<td></td>
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<td>2. Symptoms of PFPS for &gt; 2 months</td>
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<td>3. Retropatellar pain and crepitation when jumping, running, squatting, up and down stairs</td>
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<td>4. Clinical signs i.e., pain upon compression and positive apprehension tests.</td>
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<td>Thomee et al. (1995)</td>
<td>Case-control</td>
<td>To determine the influence of alignment and activity levels on PFPS</td>
<td>Pain from PFJ if # is fulfilled</td>
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<td>1. During and or after activity</td>
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<td>2. Sitting</td>
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<td>3. Stairs</td>
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<td>4. Squatting</td>
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<td>Nadeau et al. (1997)</td>
<td>Case-control</td>
<td>To examine compensation in gait as a result of PFPS</td>
<td>Referred from orthopaedic surgeon with PFPS</td>
<td>1. Surgery or trauma</td>
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<td>2. Subluxation</td>
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<td>3. Systemic or orthopaedic pathology</td>
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<tr>
<td>Crossley (2002)</td>
<td>Randomised controlled trial</td>
<td>To evaluate the treatment efficacy of physiotherapy for PFPS</td>
<td>1. Anterior or retropatellar pain from at least 2 activities associated with PF.</td>
<td>1. Intra-articular pathology</td>
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<td>2. Insidious onset</td>
<td>2. Ligament laxity</td>
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<td>3. Pain on patellar facets</td>
<td>3. Referred pain from hip</td>
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<td>4. Pain along tendon, illiotibial band or pes anserinus</td>
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<tr>
<td>Powers et al. (2002)</td>
<td>Case-control</td>
<td>To compare the gait in healthy and PFPS subjects</td>
<td>1. Pain from the patellofemoral joint.</td>
<td>5. Previous surgery to the knee</td>
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<td>2. Pain during at least 2 activities associated with PF.</td>
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<td></td>
<td>1. Previous knee surgery</td>
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<td>2. Patellar dislocation</td>
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<td>3. Neurological involvement that would influence gait.</td>
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<td>4. Ligament instability</td>
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<td>5. Internal derangement</td>
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<td>6. Patellar tendinitis</td>
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The purpose of this chapter is to critically evaluate the evidence for the cited risk factors for overuse anterior knee pain (AKP) and to gain an understanding of how these factors may interact with each other. This was undertaken to provide the background information to justify the approach adopted for this PhD.

3.1 Epidemiology and static mechanisms

There is a wealth of research into risk factors for overuse injury and AKP. Much of the research can be described as epidemiological. Epidemiology is a medical science that examines the distribution and determinants of health and disease in exposed and/or diseased populations. It is embedded in classical statistical theory and considered the foundation of an evidence based approach to medicine and health. Epidemiological approaches have been used extensively to quantify the association between an exposure(s) e.g. lower limb alignment, and injury, in an effort to determine risk factors. Fundamental to this is the recognition that a significant association does not imply causation. In this regard epidemiology is principally concerned with inference, and it is the strength of association and underlying path that are of most interest. Methodological weaknesses and statistical limitations are the main factors that influence the inference that can be drawn from epidemiological data, and one aim of this chapter is to highlight the limitations that have most affected the level of inference from studies into risk factors for AKP.

Rather than differentiate between diagnoses, many authors have treated all overuse injuries as a separate comparative group. There are a few reasons for this. Firstly, although the incidence of overuse injury is as high as 40% in some populations (Gemmell, 2002),
subcategorising these injuries can result in very low injury rates which to study prospectively would require an unfeasible sample size. Secondly, some studies have measured factors which are indicators of general cumulative stress e.g. activity level, and if the hypothesis is mechanical and the research variables are concerned with general overuse, then it is reasonable not to sub-classify. Lastly overuse injuries present with a similar history of activity exacerbating their symptoms, accordingly some authors have treated them as a single group distinguishable by their overuse aetiology. This means that there are actually very few epidemiological studies specifically on AKP. As such where the hypothesis was mechanical, the variables relate to general overuse and where studies specifically on AKP are lacking, studies on general overuse injuries have also been included.

3.1.1 Demographics and anthropometry

The incidence of AKP has been reported to be 2-5 times higher in females (Lichota, 2003). Females may be at an increased risk due to a number of gender differences, for example, an increased pelvic width and Q-angle, a higher body fat percentage, less lean muscle mass and decreased muscle strength and aerobic fitness. Mixed gender studies therefore need to be sufficiently powered and use appropriate multivariate analysis to adjust for gender effects, otherwise there is a risk of erroneous conclusions and true risk factors being masked.

Age is hypothesised to be a risk factor for injury due to a decreased capacity for exercise and a greater summated exposure to risk factors. Studies of overuse injuries in military recruits (age range: 17-31 years) have found an increased risk of injury in older age groups (>24 years) (Jones et al., 1993, Knapik et al., 2001). Heir et al. (1997) found similar results in Norwegian military recruits but further analysis suggested age was confounded by BMI, here older age groups also tended to have greater body mass. Generally, AKP has been found to be more prevalent in adolescent (Fairbank et al., 1984) and young populations (Taunton et al., 2002). The authors of the latter study explained that younger patients have a higher propensity to be involved in activity (Taunton et al., 2002).

Height and weight are speculated to be involved in overuse injuries by affecting the length of the joint moment arms and ground reaction forces. However, results have been
Chapter 3. Risk factors for AKP: A review

contradictory. Two studies showed taller people to be more at risk of PFPS (Taunton et al., 2002) and knee exertion injuries (Kujala et al., 1986), one study found a higher distribution of shorter and lighter individuals with AKP (Duffey et al., 2000), and two studies showed no effect for height or weight (Kujala et al., 1986; Witrouw et al., 2000). The differences found in the Duffey et al. (2000) study were small (2.3cm and 0.7kg) and possibly not clinically relevant. The study by Witrouw et al. (2000) was prospective and thus particularly with the influence of weight, is not affected by inverse causality bias, i.e., observing an effect of injury rather than a cause. Further Witrouw et al. (2000) also found no relationship between body shape (endo/ecto/mesomorph) or body fat percentage and PFPS.

Some studies have shown high BMI (>25kg.m$^2$) (Jones et al., 1993) to be a risk for overuse injury, while others have found either no relationship (Knapik et al., 2001; Koplan et al., 1982) or a higher risk in persons with a low BMI (Heir et al., 1997).

Overall, gender does seem to have an association as a surrogate variable for AKP. However, the evidence for a relationship between height, weight or BMI and AKP is equivocal. It is possible that these variables only have a predisposing relationship with injury when combined with other more salient factors.

3.1.2 Training and activity levels

External factors that affect levels of exposure to physiological forces such as the frequency and duration of training have been well studied as a risk for injury. Two large prospective studies on recreational runners found a significant association between increased weekly running mileage and injury (Macera et al., 1989 and Walter et al., 1989). Retrospective studies on similar populations also support this finding (Koplan et al., 1982; Jacobs et al., 1986).

Studies on military training populations, who are exposed to a relatively standardised training program, have found that recruits are more vulnerable to injury if they were less active prior to enlistment (Jones et al., 1993). Although military training is progressive, a proportion of individuals may still be unaccustomed to such intensive exercise. This type of injury mechanism has been termed a training error. In fact these military studies have
also shown low baseline aerobic fitness, muscle endurance and strength to be a risk for injury (Jones et al., 1993; Knapik et al., 2001). These findings indicate the importance of training load relative to an individual’s fitness. One military study examined the risk of injury between two units that were doing different levels of weekly mileage and found an odds ratio of 2.2 (95%CI: 1.16 – 4.17) for injury in the high mileage group (Cowan et al., 1993). Two cross sectional studies on AKP also showed activity levels to be a salient risk factor for AKP in females (Thomee et al., 1995) and adolescents (Fairbank et al., 1984). Only one cross sectional study found no influence of training load on PFPS (Messier et al., 1991). However, the inclusion criteria for the control group used in this study specified involvement in running for at least a year with a frequency of 4 times per week. Thus the PFPS and control group were virtually matched by activity levels making it unlikely that differences would be found in this variable. This is a form of selection bias.

One study showed a positive association between the frequency of hill running and PFPS (Messier et al., 1991). It was speculated that this was due to increased patellofemoral contact forces due to the knee being in a more flexed position. This is reflected in patients with PFPS who tend to report symptoms of pain when going up or down stairs (Salsich et al., 2001; Thomee et al., 1999).

Messier et al. (1991) showed inexperienced runners to be more at risk of PFPS. This factor may be related to a number of other factors such as training errors due to inexperience, increased susceptibility to fatigue, and inefficient running mechanics.

Overall, the literature provides strong evidence for the influence of absolute and relative load on injury and AKP, any contradictions can be explained by differences in study design and sample selection. However, this factor must interact with other risk factors because not all individuals develop AKP at a specified activity threshold.

3.1.3 Fitness

Prospective military studies have shown aerobic fitness as measured by 2.4km run time (Jones et al., 1993) and open circuit spirometry (VO_{2} max) (Knapik et al., 2001) to be a risk factor for overuse injury. A similar study in terms of design and population, specifically on risk of AKP, did not find a significant effect for aerobic fitness (Milgrom et
al., 1991). However, the AKP group did have a slower mean run time and this study used a t-test to assess differences, which is not as suitable or sensitive as the relative risk ratio to detect underlying differences within the frequency distribution. This result requires further clarification in this population. One prospective cohort study on a civilian population also found no association between VO$_2$ max and PFPS (Witrouw et al., 2000). In civilian populations, participants are able to adjust their training load to suit their level of fitness, however, for military recruits where activity is standardised and prescribed, this is not possible. Thus, the less conditioned recruits will be exercising at a greater relative exercise intensity and be more prone to fatigue. It is often cited although not directly proven, that exercising in a fatigued state may cause a shift in the load absorption from the muscle to the skeletal system due to inadequate muscle activity and altered coordination (Chappell et al., 2005; Johnston et al., 1998). Possibly related to this, one military study found aerobic fitness to interact with BMI (Heir et al., 1996), suggesting that heavier and less fit individuals may be more predisposed to injury than lighter colleagues of similar fitness.

Prospective military studies have found decreased muscle endurance to be a risk factor for overuse injury (Jones et al., 1993; Knapik et al., 2001). However, Milgrom et al (1991) found that recruits who went onto develop AKP had higher levels of muscle endurance, although this variable was non-significant when put in a multivariate statistical model due to a correlation with quadriceps isometric strength. All these studies measured muscle endurance using a press-up test, which may be of little relevance to injuries of the lower extremity.

Two cross sectional military studies found reduced quadriceps strength in persons with AKP (Kujala et al., 1986; Messier et al., 1991) and one prospective civilian study found a decreased vertical jump height in those who developed PFPS (Witrouw et al., 2000). It should be noted that these studies could be measuring the effect of pain on strength as opposed to the effect of strength on AKP. There is also a contradictory finding from a prospective study, Milgrom et al. (1991) found that recruits who developed AKP had increased quadriceps isometric strength at baseline. The authors suggested that individuals who develop AKP are able to generate higher reaction forces through the patellofemoral joint.
Ireland et al. (2003) examined the hip abductor and external rotator strength in 15 females with AKP against 15 healthy age and gender matched controls. Subjects with AKP had 26% less abduction strength and 36% less external rotation strength. It is not clear whether this is a cause or effect of AKP and there are no other studies to validate these two findings. Nonetheless the strength discrepancies were quite substantial.

In summary, the role of aerobic fitness, muscle endurance and quadriceps strength is unclear, however, it would seem that relative training load may be an important factor in overuse injury. Hip abductor and external rotator strength may be important determinants of AKP but a prospective study is required to validate these findings.

3.1.4 Lower limb morphology

3.1.4.1 Leg length discrepancy

One previous study found an increased leg length discrepancy (mean difference: 3.9mm) in Finish Army conscripts with a knee exertion injury compared to those without an injury (Kujala et al., 1986). However, three other studies found no relationship between this measure and PFPS (Cowan et al., 1996; Milgrom et al., 1991; Witrouw et al., 2000). The equivocal findings may be due to methodology, the latter studies used a clinical measure of leg length whilst Kujala et al’s (1986) study used a radiographic technique which offers better reliability. If this is a contributing factor to knee pain, the actual mechanism is unclear, since in this study, the pain was partitioned nearly equally between the longer and shorter leg in patients with unilateral pain. It is thought that a leg length discrepancy can cause a number of compensations, such as accentuated frontal and transverse plane movement of the knee and ankle in the longer leg in an effort to equate the discrepancy.

3.1.4.2 Q-angle and constituents

One previous study showed that runners with PFPS had a mean Q-angle that was approximately 6 degrees greater than non-injured runners (Messier et al., 1991). This study examined both males and females, and although females tend to have larger Q-angles than males, the result was still significant when the males were extracted from the analysis. A study of high school athletes also supports this finding (Moss et al., 1992). Insall et al. (1976) found Q-angles in excess of 20° in 40/83 patients suffering from chondromalacia patellae, whilst the mean Q-angle in 50 healthy knees was 14°. Aglietti et al. (1983) found
a smaller but significant relationship in 150 healthy knees and 90 knees with PFPS, concluding that Q-angles in excess of 17° compared to 15° in asymptomatic knees are associated with PFPS. However, Caylor et al. (1993) did not find a relationship between Q-angle and PFPS, a finding supported by a later prospective study (Witrouw et al., 2000). Further, one study found slightly smaller but non-significant Q-angle values in persons with a knee exertion injury (Kujala et al., 1986). Thomee et al. (1995) also found no relationship between Q-angle and PFPS. There is one limitation to this finding. The control group was matched only by age with the PFPS group. It is likely that the controls were more sedentary than the AKP group purely by selection bias. Thus, it is possible that some of the individuals in the control group would develop pain upon exposure to activity. The Q-angle is also prone to measurement error and poor reliability (Neely, 1998), and authors rarely give precise details on the measurement method. Muscle contraction and standing posture can affect this measurement. In fact one study quantified this angle in the supine position (Witrouw et al., 2000) contrary to the typical standing measurement. The range of ‘normal’ values reported in the literature also reflects the lack of standardisation in the methodology. Some authors argue that >15° (Messier et al., 1991) is abnormal while others are more conservative suggesting >20° (Kannus and Nittymarkki, 1994). One study refused to state actual values of Q-angle because of poor agreement with other studies (Kujala et al., 1986). Caylor et al. (1993) reported an intra-observer reliability intra-class correlation coefficient (ICC) of .83 and an inter-observer reliability ICC of only .65. ICCs are affected by sample homogeneity (ICC's are closer to 1 in more heterogeneous samples even when there is the same absolute agreement as a more homogenous sample – Bland and Altman, 1986). Considering that the sample in this study was relatively heterogeneous (range of Q-angle: -6 - 24°), these ICCs are poor.

Static external rotation of the tibia with respect to the femur (knee version) has been cited as a risk factor for AKP. One case-control study of 14 subjects who failed to respond to conservative treatment, demonstrated increased knee version in full knee extension in AKP patients versus healthy controls (Eckhoff et al., 1997). This was examined using computed tomography images, the AKP group had a mean external version of 7° compared to 1° in controls.
Also related to the Q-angle are measures of pelvic width, femoral neck anteversion and external or lateral tibial torsion. Thomee et al., (1995) found no significant differences in pelvic width in females with and without PFPS. Further, although femoral neck antetorsion is often cited a risk for PFPS in females (Krivickas, 1997), there is no epidemiological evidence to support or refute this claim, possibly due to the difficulty in measuring this variable. One study quantified lateral tibial torsion (Cooke et al., 1990) and found greater angles in the PFPS patients that did not respond well to treatment. This study used the thigh-foot angle which is possibly not an accurate or reliable estimate of true tibial torsion. No other studies have assessed this variable.

The Cooke et al. (1990) study also found an increased varum alignment of the knee in patients who didn’t respond to treatment. This result was supported by a study of Israeli Army recruits, which found an increased mean tibial intercondylar notch distance in the AKP group (Milgrom et al., 1991). However, Cowan et al. (1996) found a higher proportion with valgum alignment of the knee in a PFPS cohort and no relationship between a varus alignment. Further, a number of studies did not find a relationship between genu varum/valgum and AKP (Fairbank et al., 1984, Thomee et al., 1995; Witrouw et al., 2000). This variable tends to be measured in a standing posture as the distance between the medial tibial condyles if the knees don’t touch (genu varum), or the distance between the medial maleolus if they do (genu valgum). It is thus a surrogate measure of the actual amount of abduction/adduction of the knee, and is possibly also difficult to standardise.

3.1.4.3 Foot posture
A number of studies have examined the relationship between foot type and overuse injury. Again there is heterogeneity in the findings. One study found a relationship between both pes cavus and pes planus and overuse injury (Dahle, 1991). However, two studies found a protective effect for AKP (Cowan et al., 1993) and overuse injury (Kaufman et al., 1999) in subjects with pes planus. These latter studies were in a military population and both split the group into foot categories by distribution, it is likely that individuals with extremely low arches would have been screened out prior to military selection and biased the categorical groups. One retrospective study attempted to relate arch differences to different sites of pain, and found that persons with pes planus were more likely to have a history of PFPS than injuries to the foot or lateral aspect of the lower extremity (Williams et al.,
2001). However, nearly 50% of the participants in this study were already wearing foot orthoses, which may have biased results.

Powers et al. (1995) attempted to quantify differences in rearfoot posture in female subjects with and without PFPS. A significantly increased varus position of the rearfoot was found in subjects with PFPS (8.9° vs 6.8°). The authors suggested that a varus position may cause compensatory foot pronation. However, other studies have not supported this finding (Kujala et al., 1986; Messier et al., 1991; Thomee et al., 1995). This goniometric measurement relies on finding neutral position of the subtalar joint and is prone to measurement error (Picciano et al., 1993).

3.1.4.4 Patellofemoral alignment

Schutzer et al. (1986) reported findings from a series of computerised tomography (CT) scans of 45 patients complaining of AKP who had suspected patellofemoral malalignment and 10 controls. Cut off criteria for AKP classification were developed from the patellar tilt and congruence angle by comparing data with controls. The sensitivity of these criteria in correctly diagnosing AKP was 83%. A more recent case-control study of 50 AKP patients and 78 controls found a mean difference of 6.5° in tilt angle between groups (Pookarnjamorakot et al., 1998). This variable was put into a regression model to predict AKP, the model had a sensitivity of 70%, specificity of 73% and an overall accuracy of 71.9%. While these results appear excellent, one should consider the effect of selection bias on the accuracy of the models. If cases are selected based on some malalignment then the results may only be representative of a subset of anterior knee pain. This is highlighted in a separate study on the effect of muscle activity on patellofemoral alignment, where without selection of AKP cases by alignment, the authors found no statistical difference in patellar tilt versus a control group (Taskiran et al., 1998). However, the AKP group did have a slightly more lateralised congruence angle.

In a kinematic MRI study, Powers et al. (2000) reported a seven degree difference in tilt between a patellar pain and control group, this difference occurred at 27° of knee flexion, which is within the range where the patella engages with the femur (Goodfellow et al., 1976). It is important to note that these data were from a kinematic study, as such the results could be due to a dynamic abnormality as opposed to a static abnormality. Only one study found no relationship between patellofemoral alignment and AKP, this was in a
sample of females, where the dependent variables were the congruence angle, patellar tilt angle and the lateralisation ratio (Thomee et al., 1995). However, there are a few limitations with this study, firstly PFPS individuals with a history of subluxation were excluded from the pain group, this may have biased the results by screening out the more severe patellofemoral alignments. Further no objective criteria were used to define subluxation, Schutzer et al. (1986) classified a subluxation as a patella that remains in a lateralised angle at 10 degrees of knee flexion as measured by the congruence angle. Thomee et al. (1995) used a clinical examination to classify subluxation, which does not share such rigor. Lastly, the comparison was between the most and least symptomatic knee, which may not be a sensitive or valid comparison. In fact Schutzer et al. (1986) found a specificity (proportion of true negatives classified using a diagnostic test) of only 32% in patients with unilateral pain when comparing patellar tilt and subluxation angles in the involved and uninvolved limb. They note that the other limb may become symptomatic in the future.

Schutzer et al. (1986) also found a decreased trochlea depth, measured from the top of the lateral condyle, in patients with subluxation and tilt. This is tentatively supported by a later study which found the depth of the trochlea as measured by the sulcus angle to be a subtle predictor of patellar tilt and lateral patellar displacement (Powers et al., 2000). Powers et al. (2000) also showed a loss of trochlea depth beyond 27° of knee flexion in individuals with AKP. These findings suggest that there may be a loss in bony stability at the end range of extension in some individuals with AKP.

One study also investigated the size of the patella as a risk factor for AKP, but no association was found (Fairbank et al., 1984).

Overall, there is some good evidence to suggest that static patellofemoral alignment measured using tilt and congruence angles from CT/MRI scans is an important aetiological factor in some types of anterior knee pain. However, it is not known what proportion of AKP sufferers and what proportion of healthy individuals have a malalignment. It is also possible that the malalignment is important only if it manifests dynamically. For variables such as foot type, Q-angle, genu varum/valgum, fore/rearfoot varus/valgus and leg length discrepancy, the lack of measurement standardisation makes it difficult to compare across studies and possibly contributes to the conflicting findings. There is also little agreement
on the criteria for an abnormal alignment classification. Whilst experts and case series reports suggest an association between a number of different lower limb morphologies and AKP, these factors have not been proven in epidemiological studies that have made comparisons with a healthy population. This questions the use of these variables in classification systems for AKP.

3.1.5 Flexibility

Flexibility is commonly implicated in AKP and has been well studied. Flexibility is often discussed in terms of three elements; range of movement (ROM), muscle tone and joint laxity. In essence muscle tone and joint laxity contribute to joint ROM.

Research has not found a relationship between ankle ROM (Messier et al., 1988; Kaufman et al., 1999), subtalar ROM (Kaufman et al., 1999), tibial rotation ROM (Fairbank et al., 1984) or hip ROM (Fairbank et al., 1984; Milgrom et al., 1991) and AKP.

One previous longitudinal study examined the effect of a stretching treatment-intervention on elite skaters with AKP (Smith et al., 1991). This study found that skaters with AKP were more likely to have quadriceps muscle tightness. Further, following a stretching program, the patients that improved their muscle flexibility were more likely to have reductions in pain. This finding was supported by a prospective study which found increased quadriceps and gastrocnemius tightness in physical education students who developed PFPS (Witrouw et al., 2000). The authors of the latter study hypothesise that muscle tightness will increase patellofemoral stress. Iliotibial band tightness was also found in 12/17 patients (Obers test) with patellofemoral dysfunction, however, this study had no control group (Puniello, 1993).

One study found more medio-lateral laxity in the patellofemoral joint in individuals with a knee exertion injury (Kujala et al., 1986). This finding was supported in a prospective study, Witrouw et al. (2000) found medial patellar mobility to be a significant predictor of PFPS in a multiple regression model. No details were provided on the strength of the association.
3.1.6 Previous Injury

Forced rest can change the mechanical properties of the surrounding muscle and ligament, cause muscle imbalances and result in de-afferentiation around a joint and a loss of proprioception. Insufficient rehabilitation from injury is thus thought to increase the likelihood of re-injury. This standpoint is well supported in the epidemiological literature, which has shown injury history to be related to re-injury (Ross, 1994; Macera et al., 1989 and Walter et al., 1989). A more specific relationship was found in one study, with a previous ankle sprain being a risk factor for an overuse injury (Jones et al., 1993). To date, no studies have reported on the effect of previous injury specifically on AKP.

3.1.7 Other factors

The incidence of injury in different social groups is a general line of enquiry in the epidemiology of disease. If particular social groups have a predisposition to injury then it may give aetiological clues to a mechanism. The effects of tobacco on general health have been well studied, and recently, investigators have examined its relationship to injury. Three prospective studies found a significant relationship between smoking and overuse injury that was independent of lifestyle factors such as fitness and activity levels (Jones et al., 1993; Knapik et al., 2000, Heir, 1997). This independence was also demonstrated in a large cross sectional survey of musculoskeletal pain in 12,000 blue and white collar workers (Palmer et al., 2002). Only one study reported no association between smoking and injury (Ross, 1994). Similar to injury history, the effects of smoking specifically on AKP occurrence have not yet been studied. The mechanism by which smoking increases the risk of pain is also unclear. Biochemical, vascular and psychosocial reasons have been attributed (Palmer et al., 2002). Nonetheless, smoking status could be an important covariate to consider in a multivariate study.

3.1.8 Conclusions

Aside from training load, the evidence for the risk factors discussed in this section has been inconclusive. However, certain risk factors have more evidence than others. The strength of association for each factor may be a reflection of its level of association with the actual inciting mechanism for AKP. For example, if the inciting event is biomechanical, then the literature could be reflecting the strength of association between these variables and the
kinematics and kinetics during dynamic activities such as running. In this sense, some measures of anatomical alignment may have a stronger association with dynamic variables that cause higher biological stress than others. The degrees of freedom available to offset the effect of a predisposing factor could then be important, and factors such as gross patellofemoral malalignment may limit the scope for adjusting other degrees of freedom to reduce stress. These factors are also likely to interact with training load, for example if a person has anatomical characteristics that predispose to increased stress on the patellofemoral joint, then this may lower the threshold for developing AKP. Multivariate studies that enable the relationships between variables to be explored and adjusted for may explain some of disparity in the literature.

In fact the failure to account for activity or training load represents a flaw in the design of many of the studies reviewed in this section. Intrinsic biomechanical variables may make an individual predisposed to injury, but without exposure to extrinsic factors/forces such as activity, an individual will not be susceptible to pain. In case-control studies, it is important that the selection criteria for the control group minimises the risk of containing sedentary individuals who upon taking up some physical activity may subsequently develop AKP. Measuring activity levels and controlling for this in the design or analysis of the study would minimise this potential confounding factor. The optimal solution would be a prospective cohort design using a population with a controlled exposure to activity such as the military (e.g. Jones et al., 1993, Knapik et al., 2001).

Pain can also affect the measurement of dynamic variables such as strength and muscle activity, and case-control studies should avoid inferring causality when this is the case.

Some of the conflicting findings may be partly explained by differences in study samples and case groups. As discussed, there is no consensus on the classification of AKP/PFP. The aetiology described in classification systems is numerous, and the results from studies on small populations may only be applicable to the differential diagnosis of that particular case group.

Another criticism of these studies concerns the poor reliability and lack of standardisation of many of the static measurement variables. For example, Hamill et al. (1989) examined the between-day reliability of sixteen static measures of range of movement. Six had
correlation coefficients less than .5 and only one had a correlation greater than .8. Many studies have failed to examine or report the reliability of their measurement variables. Reliability statistics are useful because they enable a more informed interpretation of the results. Lastly, many of the static measurements studied are only surrogate measures of what is actually intended to quantify, and so may lack content validity.

3.2 Static - dynamic inter-relationships

3.2.1 Introduction

Many of the static biomechanical measurements discussed in section 3.1 are measured on the assumption that anatomical structure dictates dynamic function, this assumption has been the focus of a small number of experimental studies. The purpose of this section is to review the evidence for such a relationship. This may provide an explanation for the findings presented in the previous section.

3.2.2 Foot characteristics

Foot morphology has been described using measurements such as arch height and arch index (midfoot area/total foot area). A pes planus foot has been associated with excessive subtalar movement, which in turn has been implicated in injury (Clement et al., 1981). Hamill et al. (1989) examined the relationship between arch index and various static and dynamic measurements in 24 asymptomatic subjects. Multiple regression revealed no association between arch index and subtalar or ankle joint ROM. These latter two movements also had poor reliability (correlation coefficient <0.57). There was no relationship between arch index and the kinematics of the lower extremity during overground walking. The kinematic variables included ankle eversion, knee flexion, knee abduction/adduction and tibial rotation. Surprisingly, arch index was not related to the foot progression angle, which counters the common belief that high arched individuals have in-toeing gait and low arched individuals have out-toeing gait. Later studies also supported these findings (Knutzen and Price, 1994; Nigg et al., 1993). Another study showed that individuals with normal arches exhibited less rearfoot movement compared to individuals with a high or low arch (Kernozek and Ricard, 1990). This study found that the foot
progression angle was a better indicator of pronation than arch index. The relationship between foot morphology and subtalar movement thus appears tenuous.

The inclination of the subtalar joint is thought to be indicative of the amount of movement transfer between calcaneal eversion and tibial rotation. The subtalar joint has been compared to a universal hinge joint, as such high arches with a higher subtalar axis inclination are thought to transfer more rotational movement to the tibia. Nigg et al. (1993) found an association between arch height and the movement transfer coefficient (defined as the ratio of calcaneal eversion to tibial rotation movement). However, whilst arch height explained a significant proportion of the variance in the transfer coefficient (27%), there was still a substantial amount of unexplained variance.

Cavanagh et al. (1997) undertook a comprehensive radiographic study of 50 healthy subjects to determine whether foot structure was related to plantar pressure distribution during walking. Twenty-seven foot measurements were obtained from lateral, anterior-posterior and dorsal static weight bearing x-rays. The reliability of 21 of the x-ray measurements was good (ICCs >.75). Multiple regression was used to examine which variables predicted peak plantar pressure under the heel and 1st metatarsal head. Only three variables were significant predictors of heel pressure and four variables significant predictors of 1st metatarsal pressure. The inclination of the first metatarsal, which is a component of arch height, explained only 11% of heel pressures and 13% of 1st metatarsal pressures. This study suggests that foot type explains only a small amount the variance in plantar pressure characteristics. However, Hamill et al. (1989) found no relationship between arch index and any component of the ground reaction force. This included some of the time history events of the force profile. The functional significance of arch index on total force and timing patterns is thus questionable.

Hamill et al. (1989) also investigated first ray dorsiflexion and plantarflexion ROM, hip internal and external rotation ROM, and static tibiocalcaneal eversion angle during standing for a relationship between walking kinematics and kinetics. Only first ray mobility and hip rotation showed an association. A greater dorsiflexion laxity of the first ray was associated with increased tibial internal rotation values during stance, while greater first ray plantar flexion values were associated with increased tibial external rotation values during the propulsion phase of gait. These effects were also reflected in the
anterior-posterior component of the ground reaction force. The authors suggest that this reflects the function of the midtarsal joint to unlock during load acceptance and lock during propulsion. Hip rotation range of movement had a significant association with the varus/valgus movement of the knee (Hamill et al., 1989), however, the hip rotation ROM measurements showed poor reliability and the authors were reluctant to draw conclusions from these data.

One study examined the effect of rearfoot position on the medio-lateral patella position as measured using x-ray (Klingman et al., 1997). Rearfoot position was manipulated using 6-8° medial heel wedges in a weight bearing 45° squat position. Results showed a mean medial patella shift of 1.08mm (sd: 0.55) in the orthotic condition. The authors suggest that this may be due to decreased femoral rotation in the orthotic condition. This shows the effect of foot position on patellar location during a static posture. However, it does not give information on whether individuals with static rearfoot and forefoot varus malalignment display these characteristics dynamically. Hamill and colleagues (1989) did attempt to quantify this relationship but also reported very poor reliability for this variable.

3.2.3 Q-angle

Mizuno et al. (2001) performed an in-vitro experiment to determine the influence of the Q-angle on patellar and tibiofemoral kinematics. The set-up simulated a squatting movement and was executed with the femur constrained and unconstrained to examine the effects of an altered Q-angle on tibiofemoral kinematics. Six cadaver knees were studied, the Q-angle was manipulated by a 6 cm shift in the medio-lateral position of the quadriceps actuator. This resulted in average Q-angles of 20°, 11° and 4°. The motion of the patella was tracked from 20-90°. An increase in the Q-angle resulted in 3-5° more medial patellar tilt and a 6° of medial (internal) patellar rotation. There was also a lateral shift of the patella from 20-60° of knee flexion when the Q-angle increased. These changes manifested as the patella sliding against the lateral ridge of the trochlea. This in-vitro mechanical test held the Q-angle constant during knee flexion, as such it was actually simulating the effect of a dynamic Q-angle on tibiofemoral and patellofemoral joint biomechanics. It should be noted that the skeletal variations of the tibiofemoral joint that accompany an increased Q-angle were not modelled.
There has been only one in-vivo study that has examined the influence of the static Q-angle on gait kinematics (Heiderscheit et al., 2000). This study analysed the three dimensional kinematics of the hip, knee and ankle joints in 32 subjects. Subjects were divided into two groups based on a cut-off Q-angle of 15°. There were no significant differences in the joint angle peaks or excursions between groups. Multiple regression revealed that the Q-angle explained a small part of the variance in the time to peak tibial internal rotation ($R^2 = .16$), with higher Q-angles taking longer to reach a peak. An earlier study also found no relationship between static Q-angle and rearfoot angle during walking (Kernozek and Greer, 1993). In summary, the static Q-angle seems to have little influence on tibiofemoral or ankle kinematics during gait. Although in-vitro studies suggest higher dynamic Q-angles during knee flexion may predispose to higher contact forces, it is not known whether persons with high static Q-angles exhibit abnormal patellofemoral tracking patterns during gait.

3.2.4 Conclusions

Whilst there are only a small number of studies from which to draw conclusions from, it would appear that static variables are poorly correlated to dynamic function at the knee and that there is much between-subject variation in the relationships. Arch height showed a small association with the transfer coefficient for the subtalar joint and tibia but it was not associated with the magnitude of movement. Only one measure of ROM (first metatarsal mobility) has shown a small association with gait, yet this variable has not been measured in empirical research as a risk factor for AKP. There is also very little evidence to suggest that the static Q-angle is a determinant of dynamic function.

The lack of an association is possibly manifested in the conflicting evidence for these variables as risk factors for injury. Thus, if a mechanical model is hypothesised for AKP, then it may be more productive to consider dynamic variables in future research.
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3.3 Dynamic mechanisms for anterior knee pain

3.3.1 Foot pronation

There are no epidemiological studies to support the view that excessive foot eversion or subtalar pronation are risk factors for AKP. Messier et al. (1991) found no association between peak rearfoot eversion, total excursion or rearfoot velocity during running and AKP in a cohort of runners with AKP. Similar findings were found in later studies employing the same two dimensional methodology during running (Duffey et al., 2000) and walking (Callaghan and Baltzopoulos, 1994). Studies quantifying pronation using three dimensional techniques have also found no difference in magnitude of pronation (Powers et al., 2002). However, Duffey et al. (2000) did find a reduced two-dimensional tibiocalcaneal eversion angle during the first 10% of stance in the AKP group, this parameter has not been examined by other studies.

Foot orthoses have been shown to reduce pain in AKP patients (Eng and Pierrynowski, 1993), and have efficacy rates of 50-90% for treating overuse injuries (Gross and Napoli, 1993; Kilmartin and Wallace, 1994). Therefore, studies into the biomechanical effects of foot orthoses may give an indication into the mechanisms of injury (Gross and Napoli, 1993). Early studies found reduced rear foot movement when wearing orthoses in a group of overpronators during running (Bates et al., 1979; Taunton et al., 1985). In one of the most comprehensive studies, Mundermann et al. (2003) compared three types of orthoses (posting, neutral mould and mould with posting) against a control condition in a group of 21 subjects with overpronation. Foot eversion was less in the posting condition but tended to show an increase in the moulded condition. Other studies have found little or no effect of foot orthoses on peak eversion and eversion velocity in normal subjects (Stacoff et al., 2000a; Williams et al., 2003) and PFPS subjects (Eng and Pierrynowski, 1994). Thus, there seems to be some between-subject variation in the adaptation to foot orthoses that may also be dependent on the orthotic material and construction. The contrasting findings could also be a reflection of the poor association between pronation and AKP/overuse injury.

Pronation is thought to cause internal tibial rotation and excessive internal tibial rotation has been implicated in stress injuries about the knee (Hintermann and Nigg, 1998; Powers, 2003a). Paradoxically, internal tibial rotation reduces the Q-angle (a cited risk factor for
AKP), thereby decreasing the lateral component of the force vector created by the patellar tendon and quadriceps muscle (Powers, 2003a). Tiberio (1987) proposed a mechanism where over pronation can be an implicating factor in lateral stress on the knee. Briefly, pronation of the subtalar joint and flexion of the knee forces the tibia to internally rotate, likewise, in supination and knee extension the opposite occurs and the tibia externally rotates. From heel contact to mid-stance, pronation occurs synchronously with knee flexion. However, if pronation continues after maximum knee flexion when knee extension begins, then two opposing forces will exist, the extending knee will cause external tibial rotation while continued subtalar pronation will cause internal tibial rotation. This may cause compensatory internal rotation of the femur to allow for knee extension, which will encourage relative lateral tracking of the patella. Therefore, excessive pronation could lead to symptoms if it continues into late stance and causes compensatory internal femoral rotation. This model suggests that the timing of pronation may be important. However, only one study has shown later timing of peak pronation in an AKP cohort (Callaghan and Baltzopoulos, 1994). This study was slightly different to others reported in that the case group was older and had a very low recreational status, which the authors suggested may explain their different findings.

One reason why little evidence exists for a relationship between pronation and AKP, could be because pronation is poorly correlated to tibial movement. Much in-vitro work has examined this joint inter-relationship. Hintermann et al. (1994) examined 14 cadaver foot and leg specimens and found a variable movement transfer coefficient from ankle inversion-eversion to tibial rotation that ranged from 0.14 - 0.66. Thus, an eversion input of 20° may result in a range of 3-13° of internal tibial rotation. Dorsiflexion affected the transfer coefficient, here, more dorsiflexion corresponded to less transfer. An increase in axial load put the foot in a more everted position and the tibia in a more internally rotated position, but decreased the movement transfer coefficient. Later studies by the same team examined the contribution of the ankle ligaments to the movement transfer coefficient (Hintermann et al., 1995; Sommer et al., 1996). The ankle ligaments of 8 cadaver specimens were cut to examine their effects. Cutting the lateral ligaments (calcaneofibular and posterior talofibular) significantly increased the movement transfer coefficient while cutting the medial ligament (deltoid) caused a significant reduction in the movement transfer during eversion. Clearly, these findings may not be representative of in-vivo locomotion, for example, the axial loading, sequence of foot movement and musculo-
tendon forces of locomotion were not simulated. However, these studies have proposed the hypothesis that ligaments, ground reaction forces and foot flexion may affect the relationship between pronation and tibial rotation, and this wide between-subject variance in movement transfer has been observed in locomotion studies (Nester, 2000; Reischl et al., 1999; Stacoff et al., 2000b). For example, Reischl et al. (1999) found a non significant regression coefficient of only 0.08 between peak foot pronation and peak tibial rotation in a study of 30 subjects, while Stacoff et al. (2000b) used bone pins and found a coupling coefficient that varied from 0.24-0.96 in 5 normal subjects.

In summary, pronation alone does not discriminate between asymptomatic and symptomatic patients. The influence of subtalar movement on patellofemoral kinematics would appear to depend on the movement transfer with the tibia, which is dependent on a number of factors such as the axial load, dorsi-flexion position, ligament characteristics, joint geometry and muscle contraction. This suggests that knee kinematics cannot be inferred by measuring foot movement alone. It may be that pronation is an important factor only when it is strongly associated with the movement of more proximal joints. Future work should consider the whole lower extremity as a functional unit in relation to injury.

3.3.2 Tibial rotation

Although tibial rotation can affect the patellofemoral mechanism (Lee et al., 2001), only two studies have examined its relationship during locomotion in a group with AKP (Cudderford and Yack, 2000; Powers et al., 2002). Powers et al. (2002) found no differences in either the magnitude or timing of peak tibial internal rotation in a walking comparison between 24 females with PFPS and 17 female controls. Cudderford and Yack (2000) undertook a small pilot study, comparing 3 PFPS patients with 9 controls during running. Internal tibial rotation from initial contact to midstance was present in both groups, however, the mean excursion for the PFPS group was approximately 6° less. Although, the authors suggested this finding was related to patellar tracking abnormalities, it is not sure whether this result was reflecting a pain avoidance strategy.

Unlike the effect of foot orthoses on pronation, nearly all studies have found systematic reductions in internal tibial rotation when wearing a medial orthotic (Cornwall and McPoil, 1995; Eng and Pierrynowski, 1994; Mundermann et al., 2003; Williams et al., 2003).
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Given the efficacy of orthoses treatment for overuse injuries, this could indicate that tibial rotation is an important factor in PFPS.

Lee et al. (2001) examined 6 cadaver specimens and quantified the patellofemoral stress using pressure sensitive film at 0, 30, 60 and 90° of knee flexion with 10° of internal and external tibia rotation. Internal rotation did not alter the pressures significantly for any of the knee flexion angles. However, there was a 30-40% increase in contact pressures with 10° of external tibia rotation. The relevance of this finding at 10° of external rotation is unknown, since individuals tend to display a systematic internal tibia rotation pattern during the load acceptance phase of walking and running (Ramsay and Wretenberg, 1999). Nonetheless, external rotation of the tibia will increase the Q-angle, and an increased dynamic Q-angle has been linked to higher contact pressures due to a lateral shift in patellar contact patterns. This was shown in an earlier in-vitro study that examined the effect of Q-angle on patellofemoral contact pressures. Here, imposing a 10° increase in the Q-angle caused a 45% increase in contact pressure at 20° of knee flexion (Huberti and Hayes, 1984). It should be noted that a later in-vitro study also found increased patellofemoral contact pressures with 10° of internal tibia rotation at 30° of knee flexion (Csintalan et al., 2002). In-vitro studies have also shown that internal tibia rotation affects the kinematics of the patella, causing medial tilt (Hefzy et al., 1992) and internal patellar rotation (Lee et al., 2001).

There is no empirical evidence to suggest that excessive internal rotation of the tibia during stance is a risk factor for AKP. However, foot orthoses do modify tibial rotation, and these have been shown to be efficacious in the treatment of AKP. Further, in-vitro studies have shown how internal and external tibial rotation affects patellofemoral kinematics and kinetics. It is plausible that tibial rotation may have a role in AKP.

3.3.3 Hip/femoral rotation

Femoral rotation is considered critical to normal patellofemoral function (Powers et al., 2003a; Tiberio, 1987). Two previous studies have examined the association between femoral rotation and AKP during walking (Dillon, 1983; Powers et al., 2002) and running (Cuddeford and Yack, 2000). Dillon et al. (1983) found a mean pattern of external hip rotation from foot contact to midstance in a group of patients with chondromalacia patellae.
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compared to a mean pattern of internal rotation in controls. Powers et al. (2002) showed similar findings, in the PFPS group there was a mean external femoral rotation peak of −2.1° compared to a mean internal rotation peak of 1.6° in the control group. However, the pattern of external rotation from foot contact was not uniform in all PFPS subjects, 46% of the 24 PFPS individuals had an internal rotation pattern compared to 72% of the control group. Further, the range of peak hip rotation in the PFPS group was −18.8° to 9.0°. Published data on femoral rotation displays a large amount of between subject variability, another example from Reischl et al. (1999) showed that 27.7% of 30 healthy subjects had an internal rotation pattern. Although, skeletal pin studies have also shown notable between-subject variance for hip rotation (Reinschmidt et al., 1997), it is unknown how much of this variance is true and how much is attributable to measurement error from sources such as skin movement artefact and model weaknesses.

The only published running study found an even larger pattern of external femoral rotation in a PFPS group (Cuddeford and Yack, 2000). However, these results should be treated with caution since only 3 PFPS subjects were studied.

Powers et al. (2002) suggested that the external femoral rotation patterns in PFPS patients were compensatory actions to minimise the lateral forces that act on the patella. In support of a gait compensation strategy, studies of sagittal plane kinematics during walking in PFPS patients have also shown reduced knee flexion (Nadeau et al., 1997, Powers, 1999) and knee extensor moment during stance (Heino and Powers, 2002, Powers et al., 1999) with a compensatory increase in hip extensor moments (Nadeau et al., 1997). This quadriceps avoidance strategy would seem to be aimed at reducing the patellofemoral joint reaction force. Powers et al. (1997b) showed that avoidance patterns at the knee were reduced with patellar taping designed to correct patellofemoral dysfunction. A weakness of the gait studies on AKP is that very few have quantified pain and symptoms or accounted for it in the research design. It is possible that this is correlated with the avoidance patterns. Future work should examine this relationship to aid the interpretation of case-control studies.

Lee et al. (1994) undertook an in-vitro study to examine the relationship between femoral rotation and patellofemoral contact pressures. Thirty degrees of internal femoral rotation caused a significant increase in the patellofemoral contact pressures when the knee was
flexed at 30° and beyond. Typical amounts of knee flexion during running are 35-45° (Milliron and Cavanagh, 1991). A similar in-vitro study also found a decreased contact area resulting from internal femoral rotation at 45° of knee flexion (Fuchs et al., 1999).

In summary, in-vitro studies support the role of femoral rotation on patellofemoral stress. However, the case-control design that has been used by all the published in-vivo studies to test the hypothesis that hip/ femoral rotation is a risk factor for AKP may be flawed due to the effect of compensation. A prospective cohort study would overcome this problem.

3.3.4 Knee valgus

A few studies have examined the position of the femur and tibia projected onto the frontal plane. Studies of jump landing (Smith et al., 1991) and cycling (Bailey et al., 2003) have shown a greater valgus posture of the knee in AKP subjects. This measurement is affected by hip adduction, knee abduction and hip and tibial rotation. Since the knee allows very little abduction or adduction, it is likely these two dimensional representations are reflecting a significant amount of hip adduction and hip and tibial rotation.

3.3.5 Inter-joint coordination

Coordination is defined as the process by which the degrees of freedom are organised in time and sequence to produce a functional movement pattern (Stergiou et al., 2001a). Recently, attention has been directed at trying to understand the coordinative relationship between different joint actions during locomotion (Hamill et al., 1999).

3.3.5.1 Timing differences in peak values

Most of the research into inter-joint coordination has focused on the relationship between foot pronation and transverse plane rotation of the tibia (Bates et al., 1979; Stacoff et al., 2000b; Stergiou and Bates, 1997; Stergiou et al., 1999; Van Woensel and Cavanagh, 1992). Bates et al. (1979) originally plotted the knee flexion angle against the foot pronation angle and showed that peak knee flexion occurred at a similar time to peak pronation. This led some authors to speculate that asynchronous timing between these two movements may be a mechanism for knee injuries (James and Jones, 1990) and excessive soft tissue stress (Hamill et al., 1992; Stergiou and Bates, 1997).
Perturbation designs, where normal locomotion conditions were disturbed through changing the speed (Stergiou et al., 1999), footwear (Hamill et al., 1992; Van Woensel and Cavanagh, 1992) and surface (Stergiou et al., 1999), dominate research in this area. An early study examined the timing difference between calcaneal eversion and knee flexion during treadmill running when wearing shoes of different midsole hardness (Hamill et al., 1992). The softest midsole produced a significant timing discrepancy between these two joint actions where peak rearfoot eversion occurred before knee flexion during stance. This was due to peak pronation occurring earlier rather than knee flexion occurring later. However, the response between subjects was not universal suggesting that there were other anatomical or neuromuscular factors that influenced this adaptation. Van Woensel and Cavanagh (1992) investigated the same joint movements but with three different types of shoe geometry. A valgus and varus shoe were created with 10° of angulation over the entire midsole region. The normal shoe showed similar timing patterns between peak rearfoot eversion and knee flexion, while both the valgus and varus shoe caused peak pronation to occur after peak knee flexion. Results were similar to Hamill et al. (1992) in that the major change to the perturbation occurred at the foot rather than the knee. The lack of adaptation about the knee in these two studies is perhaps surprising given the magnitude of the perturbations. However, a criticism of these studies is that they were based on the assumption that rearfoot movement and knee flexion are indicative of the transverse plane rotations about the knee, later studies raises significant concern over this assumption (Nester, 2000; Reischl et al., 1999; Stacoff et al., 2000b). Soutas-Little et al. (1987) showed that measuring rearfoot eversion from a projection on the frontal plane overestimates this joint angle. Van Woensel and Cavanagh (1992) also noted that the time to peak pronation may not be a meaningful or reliable parameter to describe the pronation waveform because it occurs over a plateau when angular velocity is low.

McClay and Manal (1997) examined the timing relationships in 18 runners with normal and excessive pronation. Unlike the two previous studies that measured the mean time to the peak value (Hamill et al., 1992; Van Woensel and Cavanagh, 1992), McClay and Manal (1997) calculated the individual timing difference between joint actions on a stride by stride basis, noting that calculating the ensemble mean difference may mask individual differences. In the pronation group, eversion occurred earlier than knee flexion causing a timing disparity. The pronation group also showed slightly more internal tibial rotation but
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3.3.5.2 Curve correlation and slope differences

Stergiou and Bates (1997) used the curve correlation coefficient, as described by Derrick et al. (1994), to assess the relationship between eversion and knee flexion when running over surfaces of different hardness. It was hypothesised that since higher ground reaction forces are associated with running injuries, there may be coordinative changes in the function of knee and ankle movement that are factors for knee injuries. The authors observed that on average, there was a shift from a unimodal to bimodal pronation curve when running on harder running surfaces. This resulted in a slightly lower curve correlation coefficient (.78 vs .85). It was suggested that the bimodal transition may cause prolonged joint coordination asynchronicity and that this information could be lost if the joint angle plot was analysed using discrete parameters. Stergiou and Bates (1997) also quantified joint coordination by subtracting the knee flexion and rearfoot eversion angular velocities at each data point and calculating a mean and maximum difference from this curve. This method was a better discriminator of coordination differences than the curve correlation coefficient. A later study by the same authors also showed that higher running speeds and running over obstacles of increasing height induced a bimodal transition (Stergiou et al., 1999). The authors suggested that an increase in the ground reaction force may be the control mechanism for this transition.

Nester (2000) quantified the transverse plane rotations of the hip, knee and foot complex in 20 healthy subjects. The curve correlation coefficients for tibial rotation with knee and hip rotation during the stance phase of walking were .41 (range: .04 – .92) and .37 (range: .05 – .78) respectively. The low correlations should be interpreted with a little caution due to the limitations of these statistics. Derrick et al. (1994) demonstrated that while a high correlation coefficient always corresponds to similar temporal characteristics, a low correlation coefficient can be caused by differences in amplitude, and so does not necessarily mean dissimilar timing characteristics. However, the results from Nester (2000) agreed with an earlier study (Reischl et al., 1999) that used a simpler method to quantify timing difference. In a study of 30 subjects, the timing of peak foot pronation, tibial internal rotation and femoral internal rotation was 26.8%, 15.2% and 14.1% of the stance phase respectively. Here angles were referenced to the global coordinate system rather than statistically similar knee internal rotation, which suggests that there was some compensatory internal femoral rotation.
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the proximal segment (Reischl et al., 1999). Given the results of these two studies, the pattern of foot pronation would seem to have only a subtle and highly variable association with tibiofemoral kinematics.

The lack of synchronicity between subtalar movement and hip and knee transverse plane kinematics suggests that inferring transverse plane knee kinematics from knee flexion and subtalar pronation curves may be problematic. To support the proposed injury mechanism of altered joint coordination between pronation and knee flexion (Tiberio, 1987), the knee would have to move in a more constrained manner in the transverse plane to transfer the rotation to the hip. These later studies (Nester, 2000; Reischl et al., 1999) suggest that the tibiofemoral joint may move in a less constrained manner during gait than previous studies have inferred (Hamill et al., 1992; Stergiou and Bates, 1997; Stergiou et al., 1999; Van Woensel and Cavanagh, 1992). Future studies should thus avoid making assumptions about the kinematics of more proximal segments from the movement of distal segments.

3.3.5.3 Continuous relative phase

Recently biomechanists have adopted methods from motor control theory to study joint coordination. The continuous relative phase (CRP), an analytical tool developed from dynamical systems theory (explained in section 3.3.6), has recently been used in biomechanics research. There are a few techniques to calculate the CRP, but the underlying principle is based on plotting the segment velocity against displacement, and converting these coordinates into a phase angle. This phase angle contains spatial and temporal information and demarcates the qualitative state of the segment. The phase angle from two joint angles can be subtracted to provide a continuous measure of the synchronicity of two joint movements, this is called the CRP.

Hamill et al (1999) used the CRP to quantify the joint coordination patterns in five runners with patellofemoral pain. The thigh adduction — tibial rotation values were more in phase from foot contact to midstance in the injured group. Based on a similar premise to earlier work, Stergiou et al. (2001a) also used the CRP to investigate the changes in sagittal and frontal plane knee and ankle coordination to running over different obstacle heights. Similar to Hamill et al. (1999) the segments were more in phase during the impact period of the gait cycle. More in phase inter-segmental relationships were also seen at the higher
obstacle heights. It was suggested that these adaptations may occur to attenuate higher forces.

It is possible that coordination has a role in the aetiology of injury. However, little is known about the coordinative and phasing relationships between joint angles during locomotion in healthy populations. A number of different methods to quantify joint coordination have been presented, these include; the relative timing of joint actions, the curve correlation coefficient, the velocity difference between joint angles and the continuous relative phase and as reported, each of these has limitations. The CRP appears to have theoretical advantages, but it is difficult to interpret when used to quantify coordination. Further study is needed to clarify the biomechanical information contained in this approach (Burgess-Limerick et al., 1993) and the boundaries of normal values (DeLeo et al., 2004).

3.3.6 Movement variability

3.3.6.1 Background

The within-subject or inter-stride variability in gait has traditionally been treated as random error. This variance has two sources, instrumental and biological; of interest here is the biological variability. The inherent biological variability in movement has been largely ignored because it was historically equated with noise and considered to have no theoretical importance. Both research-based and clinical gait analyses typically take the mean of several strides and discard the inter-stride variance, this is highlighted in the literature where standard deviations are rarely given or discussed. It is only in the last decade that effort has been made to examine the structure of variability in gait and biomechanics. The impetus for this has come from the dynamical systems theory of motor control (Hamill et al., 1999).

Dynamical systems theory considers variability to be functional at a behavioural level (Deidrich and Warren, 1995; Glazier et al., 2006; Hamill et al., 1999), in that it can reflect a more flexible, adaptive and developed sensorimotor system. This is in contradiction to the traditional schema paradigms of motor control that suggests variability reflects errors in central and peripheral control and noise in selecting appropriate motor programs (Glazier et al., 2006). The historical view that variability is detrimental has been attributed to the
biomechanical simplicity of the tasks used to study performance in these early experiments (Glazier et al., 2006). These experiments typically measured outcome from simple tasks using variables such as error scores and accuracy. Variable outcomes were linked to unskilled performance but also to variable movement behaviour. However, in more complex tasks that utilise greater biomechanical degrees of freedom, the outcome is not necessarily a reflection of the variability of the movement patterns. In this respect variability of motor behaviour can reflect a capacity to use a greater range of coordinative structures to achieve a similar outcome. This is seen in skill acquisition where a novice performer simplifies the task by constraining the available degrees of freedom resulting in rigid and mechanised movement, whereas an experienced performer utilises more coordinative structures resulting in more fluid, unconstrained movement that is flexible and responsive to changes in the external environment. In dynamical systems theory, patterns of coordination are thought to develop from exploration and a process of self organisation. Thus, certain types of behavioural variability are considered to be functional and reflective of a healthy sensorimotor system (Deidrich and Warren, 1995; Glazier et al., 2006; Hamill et al., 1999). These functions can be stated threefold. Firstly, variability can allow flexibility to adjust to new movement patterns to foster learning. Secondly, it may allow the transition to previously learned movement patterns to meet new task constraints, and thirdly, variability provides stochastic sampling of different movement patterns that allow the selection and monitoring of the most appropriate behaviour. There is evidence to support these functions in walking and postural studies of aging (Buzzi et al., 2003; van Wegen et al., 2002; van Emmerik and van Wegen, 2000) and studies of patients with neuromuscular disease such as Parkinsons (van Emmerik et al., 1999). For example, elderly individuals at risk of falling have demonstrated reduced variability in foot centre of pressure movements during quiet standing compared to young individuals (van Emmerik and van Wegen, 2000). Comparisons of pelvic-thorax coordination during gait and gait speed transitions in patients with Parkinsons disease and controls, have shown less variability in the diseased group and more rigid coordination profiles over different test speeds (van Emmerik et al., 1999).

The background given so far has concentrated on the beneficial role of variability. However, it has been acknowledged that variability can also be detrimental. This is highlighted in the study of postural sway, where elderly patients had reduced variability during quiet standing but higher variability compared to young individuals when asked to
shift their centre of pressure forward. Here, it could be interpreted that the elderly subjects were unable to meet the task constraints and variability was having a negative effect on stability (van Emmerik and van Wegen, 2000). The consensus is that variability is only functional so long as it is occurs within the constraints and boundaries of the task (van Emmerik et al., 2000; Hamill et al., 2006). Therefore the consequences of variability on the task also need to be considered.

3.3.6.2 Variability and Musculoskeletal health

These applications and findings have led biomechanists to examine whether inter-stride variability during gait has a functional role in the preservation of musculo-skeletal health. The possible biomechanical consequences of variability to injury have been described twofold. Firstly and alluded to earlier, variability may enable greater adaptability to environmental and external perturbations and thereby aiding a transition to more optimal movement strategies (Kurz and Stergiou, 2004; Stergiou et al., 2001b). Secondly, variability may provide anatomical protection to bone and soft tissue (e.g. cartilage) by dispersing the forces among different and broader areas of the body (James et al., 2000, 2004).

The first function that variability is necessary for a flexible and adaptive neuromuscular system is at the core of dynamical systems theory and has been demonstrated in perturbation studies of gait. For example, perturbations to gait speed (Deidrich and Warren, 1995), obstacle height and ground reaction force (Stergiou et al., 2001b) were shown to cause an increase in gait variability followed by a change to a new coordinative state before variability returned to stable values. In dynamical systems theory, a perturbation that causes an increase in variability and a shift to a new coordinative state is termed a control parameter. The physiology (e.g. morphology, anatomical alignment) and neuromuscular health of an individual are hypothesised to act as control parameters, hence the rationale for the role of variability in overuse injury (e.g. Hamill et al., 1999; Heidersheith et al., 1999, 2002; Selles et al., 2001).

At a macroscopic level the second function concerning the characteristics of loading is demonstrated in the design of training programs to encompass varied activities and so avoid overuse injury caused by training errors (Dufek, 2002). In-vitro and animal studies have also shown the importance of frequency and magnitude of load in causing tissue
damage (Hunter, 1995; Radin, 1970; Weightman et al., 1973; Zimmerman et al., 1988). Accordingly, inter-stride joint coordination variability in gait may offer biomechanical protection from overuse injury through an alteration of these loading characteristics (James, 2004).

The standard deviation from the CRP has been the most common method used to quantify inter-stride coordination variability. Hamill et al. (1999) calculated the CRP in a healthy group of subjects and a group with patellofemoral pain (n=5) during treadmill running. The following CRP joint couplings were compared; thigh flexion/extension – tibial rotation; thigh rotation – tibial rotation; thigh abduction/adduction – tibial rotation and tibial rotation – foot eversion/inversion. Interestingly the CRP variability tended to be greater during the first half of stance, a finding tentatively supported by a similar study (Heiderscheit et al., 1999). The authors reasoned that increased variability at the beginning of stance when impact forces are high could be an indicator of healthy gait (Hamill et al., 1999). It was suggested that this enables the system to explore the environment to maintain stability, and helps to distribute stress among different anatomical areas (Heidersheit et al., 1999). The main purpose of Hamill et al.’s (1999) paper was to demonstrate the use of a dynamical systems approach to the study of the biomechanics of injury, as such no statistical analysis was done. However, the CRP variability plots showed reduced variability in the pain group during the late stance and late swing phase and there were also some small and inconsistent disparities during initial stance (Hamill et al., 1999). In a later study by the same group, Heiderscheit et al. (2002) used the vector coding method (see ANNEX E for an explanation of vector coding) to quantify joint coordination variability in 8 females with unilateral patellofemoral pain during treadmill running. There were no differences in the mean variability over the entire gait cycle. However, the injured limb showed less variability in the thigh rotation – tibial rotation variable from ±10% of heel contact compared to the healthy limb and to the healthy control group, which conformed to their earlier work (Hamill et al., 1999). Since all these studies used the case-control design, it is not known whether these subtle differences reflect a pain state or were risk factors for pain. In support of the former, Heiderscheit et al. (2000, cited by Hamill et al., 2006) found that with relief of pain induced by patellar taping, the variability of the injured limb increased. It was suggested that persons with pain may have less flexibility from which to coordinate movement and achieve pain-free patterns. However, a prospective study is required to determine the temporal pathway of these associations.
3.3.6.3 Summary

Dynamical systems theory explores the nature of variability in much greater depth than this brief overview (e.g. see Davids et al., 2006). However, the purpose of this section was to briefly outline the theoretical background and most importantly, to highlight possible biomechanical implications of altered variability in relation to the development of overuse injury and AKP. It is possible that inter-stride variability has a functional role in maintaining musculoskeletal health via an effect on anatomical loading characteristics. Further, the application of a dynamical systems approach to biomechanics may also provide a better insight into the mechanisms of injury than the traditional discrete single joint studies (Glazier et al., 2006). Some relationships between movement variability and patellofemoral pain have been found (Hamill et al., 1999; Heiderscheit et al., 2002), however, prospective cohort studies are required to determine the correct temporal sequence of these associations.

3.3.7 Kinetics

3.3.7.1 Ground reaction forces

The passive impact force from locomotion has historically been associated with overuse injury and degenerative joint disease, although much of the belief in the role of these forces has been derived from expert opinion (e.g. Nigg, 2001) and a classical animal study (Radin et al., 1972). However, epidemiological studies of runners dispute the role of the vertical impact force on joint disease (van Mechelen, 1992; Konradsen et al., 1990). Case-control studies using AKP subjects have also shown no relationship (Duffey et al., 2000; Powers et al., 1999). Further, no other parameters of the vertical ground reaction force have been associated with AKP (Duffey et al., 2000; Messier et al., 1991). One of the problems with quantifying load that may cause an overuse syndrome is that the forces involved are below the limit for acute failure. Further, since the ground reaction force reflects the accelerations of all the body segments during stance, it is unlikely to have the sensitivity to show an injurious loading pattern in a particular joint.

Two case-control studies have examined the anterio-posterior (a-p) ground reaction force in patients with AKP. Both studies found results that were interpreted as compensations, specifically, the uninjured limb showed an increase in the propulsive phase of the a-p force compared to the injured limb (Duffey et al., 2000; Messier et al., 1991).
The time to peak lateral force during the stance phase of walking (Callaghan and Baltzopoulos, 1994) and running (Messier et al., 1991) was significantly longer in an AKP versus a control group. It has been suggested that this variable might reflect the frontal plane movement of the lower extremity, which in turn has been implicated in AKP (Bailey et al., 2003; Powers, 2003a; Smith et al., 1991). However, a larger study of AKP found no relationship (Duffey et al., 2000). The medio-lateral ground reaction force shows an inconsistent pattern between subjects (Hamill and Knutzen, 2003), maximum values tend to be only 10% of body weight (BW) during running (Ounpuu, 1990). Although, these forces are likely to affect the moments about the knee in the frontal plane, in a similar manner to the vertical force, they may not have the sensitivity to demarcate between persons with and without AKP. In fact, whilst Duffey et al. (2000) found seven significant ground reaction force variables between an AKP (n=99) and an uninjured group (n=70), the differences were very small and when the variables were combined in a discriminant function analysis with other risk factors such as pronation during the first 10% of stance, they were no longer significant predictors of AKP.

3.3.7.2 Joint moments

Joint moments are considered a useful diagnostic indicator of injured gait (Winter, 1983). Two studies that have examined the gait in patients with AKP have shown a reduced extensor moment about the knee (Nadeau et al., 1997; Salsich et al., 2001). Persons with AKP also showed a tendency to increase the hip extensor moment, a compensatory mechanism to maintain the same net extensor moment but reduce the patellofemoral joint reaction force. There are no prospective studies relating muscle moments to those who develop AKP, or on AKP patients during a pain remission, thus the role of these moments as a precursor to AKP is unknown.

3.3.7.3 Joint reaction forces

Scott and Winter (1990) calculated the patellofemoral joint reaction force (PFJRF) during running using a mathematical model. Two-dimensional kinematic and kinetic data were collected from three individuals and used as input parameters for the model. The locations of the muscle origin and tibial tuberosity were obtained from previously published data. The patellar tendon length and position during knee flexion were obtained from cadaver data. Data relating to the tension ratio in the patellar tendon and quadriceps muscle were incorporated from a previous study. The co-contraction of the gastrocnemius muscle was
also modelled, and the patellar tendon and quadriceps muscle force calculated. The PFJRF reached a peak value of 7.6 times BW during running. This occurred during midstance when the knee flexion angle was greatest. These high reaction forces may help explain the susceptibility for pain in the patellofemoral joint.

3.3.7.4 Patellofemoral contact stress
In-vitro studies have suggested that many of the cited risk factors such as the dynamic Q-angle and femoral rotation may result in increased retro-patellar stress (Csintalan et al., 2002; Hefzy et al., 1992; Huberti and Hayes, 1984; Fuchs et al., 1999; Lee et al., 1994; Lee et al., 2001). Whilst the PFJRF has been quantified in a few in-vivo studies, only two have quantified the contact stress of the patella in-vivo (Heino and Powers, 2002; Matthews et al., 1977). The study by Heino and Powers (2002) quantified the patellofemoral stress in 10 healthy and 10 PFPS subjects during walking. The groups were matched by gender, and three walking trials at a self-selected and fast speed were undertaken. The sagittal plane knee joint moment was calculated from 3-dimensional kinematic and kinetic data. Static non-weight bearing MRI scans were taken at 0, 20, 40 and 60° of knee flexion, and sequential 2mm sagittal plane slices were used to calculate the patellofemoral contact area at each angle. Patellofemoral joint stress was calculated using a two-dimensional biomechanical model. The input variables were knee flexion angle, knee extensor moment and patellofemoral contact area. Similar to Scott and Winter (1990) the tension ratio between the quadriceps force and the PFJRF at different knee flexion angles was modelled. Patellofemoral stress over the entire gait cycle was calculated. At the self-selected pace, there was no significant difference in peak contact stress, but the PFPS group had a stress-time integral more than twice as high as the control group. During fast walking, both the peak contact stress and stress time integral were twice the magnitude in the PFPS group. At the self-selected pace, the PFJRF and knee moment were significantly less in the PFPS group, similar but non-significant results were found at the faster pace. The knee flexion angle was similar between groups, thus the increased stress patterns in the PFPS group were attributable to a reduced contact area rather than higher joint reaction forces or altered movement patterns. The authors noted that some subjects complained of pain and discomfort during testing, thus, it is likely that the reduced knee moments in this study were due to a gait compensation effect. The transverse and frontal plane movements were not modelled, so it is not clear how these would affect the PFJRF. It is also possible that the transverse and frontal plane kinematics of the tibiofemoral joint influenced the
contact area of the patella during the MRI scan, as has been shown in in-vitro studies (Csintalan et al., 2002; Fuchs et al., 1999; Hefzy et al., 1992; Lee et al., 1994; Lee et al., 2001). The sample studied had a mean age of 38 years, thus, the results may not extrapolate to anterior knee pain in younger more athletic populations. Despite these limitations, this was an important study that provides some evidence to suggest that supraphysiological patellofemoral stress may be a precursor to anterior knee pain.

3.3.8 Neuromuscular factors

A wealth of literature espouses the clinical belief that retarded muscle activation and weakness of the vastus medialis obliquus (VMO) contributes to anterior knee pain. Additionally, much of the literature concerning the rehabilitation of AKP advocates the need to focus on the VMO (Davies et al., 2001), and biofeedback and short arc quadriceps exercises are almost universally administered in the conservative treatment of AKP (e.g., Crossley et al., 2001). The neuromuscular involvement in AKP is a substantial area of study, however, some general findings and issues will be summarised here.

Many EMG studies have examined the ratio of muscle activity in the VMO compared to the vastus lateralis (VL) during isokinetic and isometric exercises. Some have reported an inhibited VMO in AKP patients indicated by a VMO:VL normalised muscle activity ratio of less than one (Cesarelli et al., 1999; Tang et al., 2001) and others have reported similar normalised muscle activity between the VMO and VL where both components are inhibited (Grabiner et al., 1994).

There are a number of studies that have shown delayed muscle onset patterns in the VMO in AKP patients (e.g. Cesarelli et al., 1999; Voight et al., 1991). However, there are also studies that have displayed synergistic VMO:VL onset patterns (e.g. Karst and Willett, 1995).

Due to the case-control study designs employed, it is not possible to determine whether VMO inhibition was present before the onset of pain. Thus, the applications of this research can only be related to whether there is a need to selectively rehabilitate the VMO. There is some supplementary evidence to support the selective inhibition of VMO due to pain. For example, the VMO is known to atrophy quicker than the VL following injury
(Gerber et al., 1983) and it is more sensitive to inhibition from swelling. One study showed that only 20ml of a hypertonic saline solution is required to inhibit the VMO compared to 50-60ml in the VL (Spencer et al., 1984)

To support the hypothesis that retarded VMO activation contributes to altered patellofemoral tracking and AKP, studies must be able to prove that the VMO can be selectively activated. Some authors contend that the evidence argues against selective activation (Grabiner et al., 1994).

The methods used in these EMG studies have been scrutinised and challenged (Grabiner et al., 1994). There are still many basic questions concerning the role of neuromuscular factors in healthy individuals. The basic control strategies used by the central nervous system to control the quadriceps musculature under different conditions is not known, likewise, it is not known how this control strategy affects patellofemoral movement. Lastly, the relationship between control strategies and pain has been equivocal and requires further investigation. This is a complex area of study, it has even been suggested that the EMG approaches used thus far may not be sensitive enough to answer these questions (Grabiner et al., 1994).

3.3.9 Conclusions

In summary, the evidence from case-control studies, perturbation studies and in-vitro work suggests that dynamic factors may have a stronger association with anterior knee pain than the static factors reviewed in section 3.1. Despite the belief that pronation is an important risk factor for knee injuries, there is little evidence for an association and laboratory studies suggest that pronation has only a tenuous relationship with the movement of the tibia and knee. In contrast, the kinematics of the hip, knee and tibia were shown to have a stronger relationship with patellofemoral kinematics. In support of a pathomechanical basis for AKP, one study demonstrated an increase in patellofemoral stress induced by a decrease in patellofemoral contact area. A prospective multivariate study that quantifies the joint kinematics that most affect the patellofemoral joint could be a more fruitful and productive approach to determining risk factors for AKP. Movement variability may also have a functional role in maintaining joint health and patterns of reduced variability were found in a group with PFPS. Whilst some studies into dynamic mechanisms for injury have adopted
Chapter 3. Risk factors for AKP: A review

a more integrated and multivariate approach, to date all these studies have used case-control designs which suffer from the unknown effect of inverse causality bias.

3.4 The nature of risk factors for injury

Throughout this review, the distinction has been made between extrinsic and intrinsic risk factors and static and dynamic variables. However, these factors have not been put in a mechanistic order to describe how they may influence injury. The purpose of this section is to describe a previously published model for injury (Meeuwisse, 1994) that has been adapted to describe the risk factors for AKP reviewed in this chapter. This model gives reference to the multifactorial nature of injury, the sequence of events that may lead to an injury, the proximity of a risk factor to the actual injury mechanism and the nature of a risk factor (figure 3.1). It should also help put the studies contained in this thesis within the context of previous research.

Meeuwisse’s (1994) injury model separates the inciting event for an injury from other risk factors that may predispose an individual to injury. Based on the mechanical model for AKP and the evidence reviewed, the inciting event is described as a biomechanical mechanism, in this case altered patellofemoral stress (figure 3.1). Although only one study has quantified increased stress in persons with AKP, amongst the cited aetiological factors for AKP, the common factor is that they may affect patellofemoral kinematics and kinetics.

The first factors along a chain of events to injury are suggested to have an intrinsic predisposing influence (figure 3.1). The intrinsic factors listed in figure 3.1 are those that were shown to be a risk factor for AKP, and have not been refuted by research using a similar study design. These intrinsic factors make an individual more or less vulnerable to an injury beforehand, however, there still needs to be an exposure to an external factor to cause an injury. When exposed to an external factor, an individual becomes sensitive or susceptible to an injury. The most conclusive external risk factor for an overuse injury was training load (e.g.; Thomee et al., 1995). A previous study also showed increased amounts of hill running to be a risk factor for AKP (Messier et al., 1991).
Chapter 3. Risk factors for AKP: A review

The dynamic risk factor/ mechanism box describes variables that were shown, or suggested to affect patellofemoral stress. Training load was also included in this box, since it is a characteristic of load. In-vitro studies have shown that lower extremity kinematics have an effect on the patellofemoral contact stress, through an altered contact area (e.g. Mizuno et al., 2001) and an altered joint reaction force (e.g. Huberti and Hayes, 1984). Subjects with AKP were shown to have less inter-stride variability than healthy controls (Hamill et al., 1999), and although not proven, it was suggested that this may subject a joint to more localised stress patterns. These variables can also be described as predisposing factors, which is the reason for the additional connecting arrow to the predisposed category. However, they are also dynamic determinants of patellofemoral joint stress, and are thus described as a mechanism as well as a predisposing factor (figure 3.1).

Figure 3.1. A dynamic multi-factorial model of risk factors for sports injury, adapted for anterior knee pain (adapted from Meeuwisse, 1994).

The factors listed in figure 3.1 are not definitive or conclusive. It is possible that sample distribution within the case group and study design were reasons why some factors were
non-significant in certain studies, and one feature of this review was the difficulty in comparing results between studies for these reasons. Further, some factors in the model such as hip strength and knee version have not been well studied, and may be refuted with further research.

Despite the lack of clear evidence for risk factors for AKP, the model is useful for differentiating between factors that predispose to AKP and factors that are mechanisms for AKP. This distinction was highlighted in section 3.2, where the relationship between static biomechanical variables and dynamic function was shown to be tenuous and have wide between-subject variability. Thus with reference to the injury model, there is likely to be some association between the internal risk factor and dynamic mechanisms box but the strength of association varies between individuals. If PFPS is caused by altered stress patterns as was indicated by the Heino and Powers (2002) study, then the kinematics of locomotion may have a significant influence on whether a person develops AKP, which provides a good rationale for investigation.

3.5 Research design and levels of evidence

Given the clinical nature of this thesis and the wealth of epidemiological studies reviewed, it is worth outlining the research governance guidelines regarding the levels of evidence of different research designs in the context of this research.

The Oxford Centre for Evidence-Based Medicine has described 5 levels of evidence with a number of sub-levels that depend on the variance of findings (Yasuf et al., 1998). For an aetiological study, level 1 represents randomised controlled trials; levels 2 are prospective cohort studies; levels 3 are case-control studies; level 4 is a case series report and level 5 represents expert opinion without critical appraisal or research based on first principles. In examining the epidemiological evidence presented in this chapter, the majority of results would be classified in the lowest sub-category of level 3, which represents case-control studies with heterogeneous findings. Examples of the weaknesses of the case-control design for studying risk factors were highlighted in this review, these weaknesses apply
particularly to dynamic mechanisms since the effect of pain on the dependent variables is unknown. Similarly the benefits of the prospective cohort design were also advocated. However, this review found only two published prospective cohort studies specifically on AKP (level 2). These guidelines are an indication of the nature of the evidence rather than the strength of a relationship. For example, certain dynamic mechanisms such as femoral rotation are well founded by biomechanical theory and cause and effect research but have low level empirical evidence since all the published data are from case-control designs. There is thus a requirement for prospective cohort studies that examine risk factors and in particular dynamic mechanisms for AKP.

3.6 Conclusions: A multivariate, prospective and hypothesis generating approach

This review (Chapters 2 & 3) has discussed the functional anatomy and physiology of the lower limb and detailed some clinical aspects of anterior knee pain such as the classification and possible pathogenesis of the symptom complex. Some key concepts of an epidemiological and an evidence based approach to injury research were discussed in the context of previous research into biomechanical and non-biomechanical risk factors for anterior knee pain. Information has thus been sought from other medical sciences in addition to biomechanics. However, this was considered necessary given the interdisciplinary field of musculoskeletal injury and the multifactorial nature of anterior knee pain. It is also fundamental to the approach developed for this work.

Despite the numerous studies, the most appropriate way to classify anterior knee pain is not clear, the pathophysiology is poorly understood and the evidence for the cited risk factors is equivocal. From experimental studies and a theoretical standpoint, it is plausible that the kinematics of gait may be a risk factor for anterior knee pain. This provides a sound rationale to focus this project into gait and AKP. However, the evidence to support this association and other factors is of a low level and suffers from research design and statistical limitations. In addition to the predominant use of the case-control study and the problem of determining the temporal sequence of an association because of the potential
effect of the condition on the measurement, insufficient consideration has been given to the multi-factorial nature of anterior knee pain.

This latter point is a general criticism of the methodological design of previous studies. It applies to issues such as the selection of the control group, the choice of measurement variables, the control of confounding factors and the choice of statistical analysis. These limitations have been recognised by members of the scientific and medical community (e.g. Bahr and Holm, 2003; Murphy et al., 2003) and are not unique to the study of anterior knee pain. For example, Bahr and Holm (2003) emphasise the multi-factorial nature of injury and note that an individual’s likelihood of injury is the sum and interaction of a number of risk factors. In this manner, the univariate, less integrated and overly descriptive approach to injury research that has been a feature of many studies may mask true factors. Given the lack of understanding of risk factors for anterior knee pain, a multivariate approach that compliments the multifactorial nature of the injury is thus recommended. This should be executed by considering other variables which may or may not be biomechanical as possible covariates, some examples are activity levels, fitness and injury history. Thus while the research hypothesis may be principally concerned with gait kinematics, it is important to consider these other risk factor variables because they may affect the strength of a relationship or interact with the main variables, and if not controlled for in the design or analysis could cause real effects to be masked. In this sense, it has been recommended that injury research should measure variables beyond the inciting mechanism(s) (Bahr and Holm, 2003).

It is also important to use multivariate statistical analysis so that the inter-relationships among risk factors can be explored. For example, some variables may only be a risk factor when others are controlled for, while the addition of a factor may increase the strength of a predictive relationship. Univariate analyses, which have been common in the literature, may hide these potentially important suppressor and mediating variables. It is possible that the univariate approach is partly responsible for some of the conflicting findings. A multivariate approach to the design and analysis of injury research may thus offer a more rigorous and productive approach. Further, given the lack of evidence and paucity of prospective studies, an explorative and hypothesis generating approach is also recommended.
Lastly and most importantly given the focus on movement kinematics, is the requirement for prospective studies. This is the most preferable design given the aim to quantify risk factors and the problem of obtaining valid baseline exposure data once the injury has occurred. This approach is considered logical in light of the lack of empirical evidence for risk factors for anterior knee pain.
Chapter 4

The epidemiology of AKP: A prospective cohort study

4.1 Introduction

4.1.1 Rationale

The literature review (Chapters 2 and 3) emphasised the need for a multivariate approach to research into anterior knee pain. Thus, it is important to consider factors that whilst may not be the primary trigger for the onset of pain, could have a mediating effect that alters the strength of an association between a risk factor and an injury. This is important for designing future research to avoid masking true risk factors due to ill considered study design and confounding. Prospective cohort studies and an explorative hypothesis-generating approach were also recommended.

To date there are no published data on the effects of social characteristics (e.g., smoking), injury history and training history on AKP. The effect of relative training load was also considered important and needs investigation in relation to anterior knee pain. The literature suggests that these intrinsic and extrinsic factors could be mediating variables and important covariates for future empirical research into AKP.

4.1.2 Military training

British Army recruits undergo an initial 12-week physical training program (common recruit training syllabus (CMS(R))). This can place a substantial physical demand on certain individuals and is reflected in injury rates of approximately 40% (Gemmell, 2002). Army recruits are 17-31 years old and by virtue of selection procedures and training protocols are considered a young, highly active and athletic population. Most importantly, the 12-week training program is standardised across recruits. For example, all recruits undertake similar levels of activity, exercise on the same training surfaces, have similar nutritional intake and
are exposed to equal periods of activity, rest and sleep. Military candidates undergoing CMS(R) are thus an ideal population to study risk factors for injury.

4.1.3 Aims

The aims of this investigation were to:

- Obtain a measure of AKP occurrence in an Army training population undergoing a short duration exercise program.

- Examine the association between prior activity levels, aerobic fitness, body mass index, smoking and alcohol intake, prior musculo-skeletal injury and the development of AKP.

4.2 Method

Ethical approval was obtained from the Defence Medical Services Clinical Research Committee, UK. All participants were verbally briefed about the methods and aims of the study and gave written informed consent.

4.2.1 Study design and subject selection

A prospective cohort study of 960 male Army recruits enlisted onto the CMS(R) training syllabus at the Army Training Regiment, Pirbright was undertaken. All recruits underwent a structured and broadly similar 12-week training program. Exposure status was calculated for a number of demographic, social, training and medical variables at the beginning of training, and the cohort was followed up for AKP occurrence over the training period. Sixty-three subjects were still in training at the end of the study due to extended absence and back-trooping, these subjects had no follow up or outcome data and so were removed from the study, leaving 897 subjects for the main analysis into risk factors.
4.2.2 Baseline measurements and procedures

The independent variables were collected on the first two days of the training syllabus when all subjects were healthy and free of injury.

4.2.2.1 Entry Questionnaire

A questionnaire was developed to obtain information on each subject's demographics (gender, age, height and weight), social (alcohol intake and smoking habits) and training history (pre-enlistment running and strength training). All subjects completed this questionnaire on entering training. A non-military investigator (Author) briefed subjects on each section of the questionnaire. Subjects were asked to give honest answers and informed that their responses would be kept strictly confidential and would not affect their military training or service.

To ensure that the questions were unambiguous and had face and content validity, the questions were piloted on 27 recruits before the start of the study. Questions to quantify social and training history were subsequently simplified into a number of short questions to obtain direct closed responses (ANNEX A). Since some of the questions required numeric responses about lifestyle and past events, it was thought that there could be some error in recalling this information. Thus to gain an indication of the stability of these measurements an intra-subject reliability study was undertaken, this is explained below.

Questionnaire reliability:

50 randomly selected subjects completed the AKP study questionnaire (ANNEX A) on two separate occasions at least 5 hours apart. There is the risk that some responses on the questionnaire at time point 2 may have been influenced by their memory of earlier responses, and normally these studies repeat methods on two separate days to minimise this risk. However, logistical constraints prevented a repeat assessment on a separate day, and so to minimise this bias, individuals were selected at random at least 5 hours after completing their first questionnaire without any prior warning. The limits of agreement (LOA) method (Bland and Altman, 1986) was used to assess the within-subject reliability. This is considered a more valid and meaningful measure of reliability compared to other statistical methods based on correlation and hypothesis testing.
Smoking and alcohol intake showed excellent agreement, the 95% LOA were within limits that can be considered to have a negligible effect on the study (Table 4.1). The LOA were poor for the strength training variable (Table 4.1) and were considered large enough to affect the results of the main study. On inspection of the raw data, this poor result was due to discrepancies in both the ‘number of sessions per week?’ and ‘minutes per session?’ question. To improve the reliability for this factor, the responses were categorised for the main analysis and the mins.session\(^{-1}\) variable was removed. Categorisation was also applied to the variables relating to running history. This should avoid magnifying errors as occurs when deriving the mins.week\(^{-1}\) variable, and should also reduce the effect of outliers. The percent agreement following categorisation was considered excellent (Table 4.2).

### Table 4.1. Response bias and 95% LOA for the intra-subject reliability of the social and training history derived continuous variables.

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Mean Bias (T2 - T1)</th>
<th>95% CI for Bias</th>
<th>95% LOA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking (cigs.day(^{-1}))</td>
<td>0.12</td>
<td>-0.00 - 0.24</td>
<td>-0.73</td>
</tr>
<tr>
<td>Alcohol Intake (units.week(^{-1}))</td>
<td>0.37</td>
<td>-0.00 - 0.74</td>
<td>-2.20</td>
</tr>
<tr>
<td>Running (miles.week(^{-1}))</td>
<td>0.22</td>
<td>-0.39 - 0.83</td>
<td>-4.00</td>
</tr>
<tr>
<td>Strength training (mins.week(^{-1}))</td>
<td>-4.29</td>
<td>-23.03 - 14.46</td>
<td>-134</td>
</tr>
</tbody>
</table>

### Table 4.2. Percent agreement for the intra-subject reliability of the variables relating to running and strength training history.

<table>
<thead>
<tr>
<th>Variable*</th>
<th>% agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Running</td>
</tr>
<tr>
<td>Yes/ No</td>
<td>100</td>
</tr>
<tr>
<td>Frequency</td>
<td>88</td>
</tr>
<tr>
<td>Miles per week</td>
<td>90</td>
</tr>
<tr>
<td>Since</td>
<td>96</td>
</tr>
</tbody>
</table>

*Please see Table 4.3c for a list of categories within each variable

4.2.2.2 Musculoskeletal injury history

Recruits underwent an entry medical by a General Practitioner (GP) and were asked to give information about any previous injuries in the last 5 years that were reported to a GP,
physiotherapist or other healthcare professional. This was also corroborated with their medical record, and injuries were categorised depending on the site and type of injury (Table 4.3D).

4.2.2.3 Anthropometry
Height and weight were measured and Body Mass Index (BMI) was calculated using the following equation: \[ \text{BMI} = \frac{\text{weight (kg)}}{\text{height}^2 \text{ (m)}} \]

4.2.2.4 Aerobic Fitness
All recruits did a 2.4 km timed run administered by the regiment training personnel. This has been shown to be a good indicator of maximum oxygen uptake (Burger et al., 1990). It was considered that ‘prior activity levels’ and ‘aerobic fitness’ would provide an indication of each subject’s relative training intensity and potential acclimatisation to the training program.

4.2.3 Physical training program
During the 12-week training period, all subjects wore similar clothing and footwear. In addition to the daily undertaking of common military tasks such as marching and tabbing (walking and running with weight), all subjects undertook a progressive standardised exercise training program. This consisted of 5-6 one-hour sessions per week of specific physical training aimed at improving aerobic capacity and anaerobic endurance and strength. Recruits did not participate in additional individual activity outside the normal training syllabus.

4.2.4 Follow-up
4.2.4.1 AKP diagnosis and case-capture
The literature details a wealth of differential diagnosis for AKP, some of which requires advanced imaging methods impractical for a study of this size. AKP is predominantly distinguished by the location of pain and the characteristics of onset. The variables examined in this study were considered markers that may indicate the potential for general overuse of non-specific tissue (e.g. activity levels, aerobic fitness, anthropometry) or markers that may elicit a mediating response affecting sensitivity to injury (e.g. smoking status). Since these are non-specific to a hypothesised biomechanical mechanism, it was
deemed valid to capture the symptom complex of AKP as opposed to a more precise diagnosis. Following discussion with two consultant physicians (Rheumatology and Sports Medicine), the following criteria were developed and used to define a case: 1. Pain around the anterior aspect of the knee; 2. Insidious onset; 3. No evidence of trauma. Clinical histories were taken to exclude pain due to knocks, twists or falls, and any swelling was noted and excluded. Medical records were reviewed twice per week to ascertain cases. Any medical notes where a case was difficult to define according to these criteria were followed up by communication with the GP, physiotherapist or recruit. As part of a separate study, twelve patients were also seen in clinic by a Sports medicine physician and the medical notes produced here resulted in the same AKP classification thereby cross-validating the approach employed for this study.

4.2.4.2 Exclusions
The following criteria were applied to exclude subjects that did not develop AKP from the risk factor analysis: 1; Training failure and 2; Development of a lower limb overuse injury other than AKP during training. The first criterion was used to control the training exposure ‘dose time’ and the second criterion was applied to minimise the possibility of other overuse injuries with similar aetiology masking true risk factors. Data relating to other overuse injuries were obtained by reviewing all medical notes and electronic hospital database records.

4.2.5 Statistical analysis
The cumulative incidence of AKP over the 12 week training program was calculated. Univariate risk factor analysis was performed using relative risk ratio (RR) statistics and confidence intervals. Categorical variables were created from continuous variables and referenced to a common-sense baseline category (e.g. RR of AKP in smokers = % incidence of AKP in smokers/ % incidence of AKP in non-smokers). The chi-squared statistic was used to test whether the RR ratio was significant. Alpha was set at .05. Multivariate logistic regression was used to assess for confounding. Logistic regression allows for the calculation of the odds of an outcome (AKP) when adjusting for the effects of other variables. Variables were selected for the multivariate model if they showed a significant relationship or a trend in the univariate analysis. A backwards stepwise logistic
regression (likelihood ratio procedure) was used (SPSS v10). The statistical criterion for removal of a variable was set at .05.

4.3 Results

4.3.1 Incidence and morbidity

Seventy-seven (8.6%, 95%CI: 6.8 – 10.4) subjects presented to the medical centre complaining of AKP (denominator = 897, see section 4.2.1). Generally, the cases of AKP were mild resulting in a median of 3 days (IQR: 3-5) on partial sick leave. However, 26% of the AKP cases were held back in training and 10.4% were medically discharged from the Army.

4.3.2 Exclusions

Of subjects who didn’t develop AKP (n=820), 132 (15% of the total cohort) were excluded from the ‘risk factor’ analysis due to training failure and 176 (20%) were excluded due to an overuse injury other than AKP (see section 4.2.5.2 for the justification of exclusion criteria). 589 subjects thus comprised the main analysis into risk factors (n of AKP = 77; n of healthy and no lower limb overuse injury = 512).

4.3.3 Cohort descriptives

The median age of the sample for the risk factor analysis was 20.6 years (IQR: 17.7 – 22.4), the mean height was 1.79m (sd: 0.06), the mean weight was 72.7kg (sd: 10.0) and the mean BMI was 22.7 (sd: 2.6). The age distribution was positively skewed with a maximum age of 32.7 years.

4.3.4 Risk factors

4.3.4.1 Demographics and physical characteristics

Age did not show an association with AKP (Table 4.3a). There was no association between height, weight or BMI and AKP. The least aerobically fit subjects, as measured by the 2.4 km run time, had a higher RR ratio but this was non-significant (Table 4.3a). This trend
Chapter 4. Epidemiology of AKP

was also non-significant in the multivariate analysis and the variable was removed from the final model (Table 4.4). Unfortunately, due to an administrative problem the 2.4km run data were unavailable for 173 (29%) of the subjects used in the analysis.

4.3.4.2 Social history
Current smokers were at an increased risk of AKP (Table 4.3b). There was also a significant dose-response relationship, heavy smokers (>20cigs.day⁻¹) had nearly fourfold the risk of AKP compared to non-smokers. Subjects who had given up smoking in the last month, were not at an increased risk of AKP. Smoking was an independent risk factor when adjusting for physical fitness (2.4 km run time) and pre-enlistment activity levels (running miles.week⁻¹) in the multivariate analysis (Table 4.4). Alcohol intake showed a non-significant trend where the heaviest drinkers were more prone to AKP (Table 4.3b).

4.3.4.3 Training history
There was no significant association between pre-enlistment running and AKP, however, a large proportion of the RR ratio confidence interval was below 1 in those that undertook some running (Table 4.3c). This pattern was also evident with strength training (Table 4.3c). Table 4.3c also shows that the incidence of AKP was lower although not significant in those that undertook some activity for as little as 4 weeks before entering training.

4.3.4.4 Musculoskeletal injury history
Individuals with a previous ankle injury were at a greater risk of developing AKP (Table 4.3D). A previous ankle injury was also an independent risk factor for AKP when modelled with other variables measured in this study (Table 4.4). Table 4.5 provides a summary of the side the previously injured ankle against the limb which developed AKP for each subject, in all cases where data were available, the limb that had an ankle injury also developed AKP. The incidence of AKP was also greater in individuals who had a previous episode of AKP or a previous knee ligament injury, however, the results were not significant (Table 4.3D).

4.3.4.5 Multivariate model
The Chi-squared statistic and Hosmer Lemeshow test both suggest that the two predictor variables for the multivariate model (smoking and previous ankle injury) explained a significant amount of the variance in AKP outcome above that of the constant. However,
the amount of explained variance from the predictor variables in the multivariate model was only 6% ($R^2$) (Table 4.4). The residuals and influence statistics for the model were satisfactory.

### Tables 4.3 a – d. Incidence of AKP by risk factor, with associated RR ratios and 95% CI.

(a) Demographics and physical characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Incidence (%)</th>
<th>RR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (yrs)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16-19</td>
<td>327</td>
<td>13.1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>20-21</td>
<td>99</td>
<td>14.1</td>
<td>1.08</td>
<td>0.62 – 1.88</td>
</tr>
<tr>
<td>22-23</td>
<td>60</td>
<td>10.0</td>
<td>0.76</td>
<td>0.34 – 1.71</td>
</tr>
<tr>
<td>24-25</td>
<td>38</td>
<td>10.5</td>
<td>0.80</td>
<td>0.30 – 2.11</td>
</tr>
<tr>
<td>26+</td>
<td>65</td>
<td>15.4</td>
<td>1.09</td>
<td>0.58 – 2.05</td>
</tr>
<tr>
<td><strong>Height (m)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 1.7</td>
<td>41</td>
<td>17.1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>1.7 – 1.74</td>
<td>89</td>
<td>7.9</td>
<td>0.46</td>
<td>0.17 – 1.22</td>
</tr>
<tr>
<td>1.75 – 1.79</td>
<td>179</td>
<td>13.4</td>
<td>0.79</td>
<td>0.36 – 1.69</td>
</tr>
<tr>
<td>1.8 – 1.84</td>
<td>169</td>
<td>13.0</td>
<td>0.76</td>
<td>0.35 – 1.66</td>
</tr>
<tr>
<td>&gt; 1.85</td>
<td>108</td>
<td>13.9</td>
<td>0.81</td>
<td>0.36 – 1.85</td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 60</td>
<td>46</td>
<td>13.0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>60 – 69</td>
<td>176</td>
<td>10.8</td>
<td>0.83</td>
<td>0.35 – 1.95</td>
</tr>
<tr>
<td>70 – 79</td>
<td>219</td>
<td>13.7</td>
<td>1.05</td>
<td>0.46 – 2.38</td>
</tr>
<tr>
<td>80 – 89</td>
<td>102</td>
<td>17.6</td>
<td>1.35</td>
<td>0.57 – 3.18</td>
</tr>
<tr>
<td>&gt; 90</td>
<td>41</td>
<td>2.4</td>
<td>0.19</td>
<td>0.02 – 1.49</td>
</tr>
<tr>
<td><strong>BMI (kg.m$^2$)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20 – 24.99</td>
<td>377</td>
<td>14.1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>&lt; 20</td>
<td>87</td>
<td>10.3</td>
<td>0.74</td>
<td>0.38 – 1.43</td>
</tr>
<tr>
<td>&gt; 25</td>
<td>119</td>
<td>10.1</td>
<td>0.72</td>
<td>0.40 – 1.30</td>
</tr>
<tr>
<td><strong>2.4 km run time (s)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 569</td>
<td>102</td>
<td>9.8</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>570 – 659</td>
<td>252</td>
<td>12.3</td>
<td>1.25</td>
<td>0.64 – 2.46</td>
</tr>
<tr>
<td>&gt; 660</td>
<td>62</td>
<td>17.7</td>
<td>1.81</td>
<td>0.82 – 4.01</td>
</tr>
</tbody>
</table>
### (b) Social history

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Incidence (%)</th>
<th>RR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoke (current)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>325</td>
<td>9.8</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>264</td>
<td>17.0</td>
<td>1.73</td>
<td>1.13 – 2.64*</td>
</tr>
<tr>
<td>Smoke (cigarettes.day(^{-1}))</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None in last year</td>
<td>299</td>
<td>10.4</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>1 – 9</td>
<td>100</td>
<td>15.0</td>
<td>1.45</td>
<td>0.82 – 2.57</td>
</tr>
<tr>
<td>10 – 19</td>
<td>147</td>
<td>15.6</td>
<td>1.51</td>
<td>0.91 – 2.49</td>
</tr>
<tr>
<td>&gt;20</td>
<td>17</td>
<td>41.2</td>
<td>3.97</td>
<td>2.06 – 7.67*</td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>117</td>
<td>11.1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>472</td>
<td>13.6</td>
<td>1.22</td>
<td>0.70 – 2.14</td>
</tr>
<tr>
<td>Alcohol (units.week(^{-1}))</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>117</td>
<td>11.1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>&lt;5</td>
<td>79</td>
<td>11.4</td>
<td>1.03</td>
<td>0.46 – 2.28</td>
</tr>
<tr>
<td>6 – 14</td>
<td>208</td>
<td>12.0</td>
<td>1.08</td>
<td>0.58 – 2.03</td>
</tr>
<tr>
<td>15 – 24</td>
<td>108</td>
<td>15.7</td>
<td>1.42</td>
<td>0.72 – 2.78</td>
</tr>
<tr>
<td>&gt;24</td>
<td>58</td>
<td>19.0</td>
<td>1.71</td>
<td>0.82 – 3.57</td>
</tr>
</tbody>
</table>

### (c) Training history

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Incidence (%)</th>
<th>RR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Running</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>34</td>
<td>20.6</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>555</td>
<td>12.6</td>
<td>0.61</td>
<td>0.31 – 1.23</td>
</tr>
<tr>
<td>Running (frequency.week(^{-1}))</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>34</td>
<td>20.6</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>59</td>
<td>8.5</td>
<td>0.41</td>
<td>0.14 – 1.20</td>
</tr>
<tr>
<td>2-3</td>
<td>348</td>
<td>12.9</td>
<td>0.63</td>
<td>0.31 – 1.28</td>
</tr>
<tr>
<td>≥4</td>
<td>148</td>
<td>13.5</td>
<td>0.66</td>
<td>0.30 – 1.43</td>
</tr>
<tr>
<td>Running (miles.week(^{-1}))</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>34</td>
<td>20.6</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>1-5</td>
<td>210</td>
<td>12.9</td>
<td>0.62</td>
<td>0.30 – 1.32</td>
</tr>
<tr>
<td>6-10</td>
<td>221</td>
<td>11.8</td>
<td>0.57</td>
<td>0.27 – 1.21</td>
</tr>
<tr>
<td>11-15</td>
<td>48</td>
<td>20.8</td>
<td>1.01</td>
<td>0.43 – 2.39</td>
</tr>
<tr>
<td>≥16</td>
<td>76</td>
<td>9.2</td>
<td>0.45</td>
<td>0.17 – 1.18</td>
</tr>
<tr>
<td>Running (since when)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>34</td>
<td>20.6</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>&lt;1 month</td>
<td>125</td>
<td>11.2</td>
<td>0.54</td>
<td>0.24 – 1.24</td>
</tr>
<tr>
<td>1 – 2 month</td>
<td>261</td>
<td>13.8</td>
<td>0.67</td>
<td>0.32 – 1.39</td>
</tr>
<tr>
<td>≥3 month</td>
<td>164</td>
<td>11.6</td>
<td>0.56</td>
<td>0.26 – 1.23</td>
</tr>
<tr>
<td>Strength training</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>112</td>
<td>16.1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>477</td>
<td>12.4</td>
<td>0.73</td>
<td>0.47 – 1.25</td>
</tr>
<tr>
<td>Strength training (sessions.week(^{-1}))</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>112</td>
<td>16.1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>41</td>
<td>14.6</td>
<td>0.91</td>
<td>0.39 – 2.13</td>
</tr>
<tr>
<td>2-3</td>
<td>293</td>
<td>12.6</td>
<td>0.79</td>
<td>0.47 – 1.32</td>
</tr>
<tr>
<td>≥4</td>
<td>143</td>
<td>11.2</td>
<td>0.70</td>
<td>0.37 – 1.30</td>
</tr>
<tr>
<td>Strength training (since when)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>112</td>
<td>16.1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>&lt;1 month</td>
<td>97</td>
<td>11.3</td>
<td>0.71</td>
<td>0.35 – 1.42</td>
</tr>
<tr>
<td>1 – 2 month</td>
<td>120</td>
<td>10.8</td>
<td>0.67</td>
<td>0.35 – 1.31</td>
</tr>
<tr>
<td>≥3 month</td>
<td>260</td>
<td>13.5</td>
<td>0.84</td>
<td>0.50 – 1.41</td>
</tr>
</tbody>
</table>
Chapter 4. Epidemiology of AKP

(d) Previous Injury

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Incidence (%)</th>
<th>RR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee Ligaments</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>571</td>
<td>13.0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>18</td>
<td>22.2</td>
<td>1.74</td>
<td>0.71-4.23</td>
</tr>
<tr>
<td>AKP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>578</td>
<td>12.9</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>11</td>
<td>18.2</td>
<td>1.40</td>
<td>0.39-5.00</td>
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<tr>
<td>Ankle Injury</td>
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<td></td>
</tr>
<tr>
<td>No</td>
<td>557</td>
<td>12.4</td>
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<td></td>
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<tr>
<td>Yes</td>
<td>32</td>
<td>25.0</td>
<td>2.02</td>
<td>1.06-3.83*</td>
</tr>
</tbody>
</table>

*p<.05

Table 4.4. Summary of risk factors from the backwards stepwise multivariate logistic regression analysis. Beta coefficients (β) and their significance, odds ratios (exp β) and the significance of the change in the model (LL = log likelihood) if the variable is removed are presented.

<table>
<thead>
<tr>
<th>Variable</th>
<th>β</th>
<th>se</th>
<th>P</th>
<th>exp β</th>
<th>95% CI</th>
<th>sig. of change in LL if term removed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None in last year</td>
<td>-1.11</td>
<td>1.04</td>
<td>.285</td>
<td>0.33</td>
<td>0.04</td>
<td>2.52</td>
</tr>
<tr>
<td>None in last month</td>
<td>0.39</td>
<td>0.34</td>
<td>.252</td>
<td>1.48</td>
<td>0.76</td>
<td>2.88</td>
</tr>
<tr>
<td>1-9</td>
<td>0.44</td>
<td>0.30</td>
<td>.145</td>
<td>1.45</td>
<td>0.86</td>
<td>2.77</td>
</tr>
<tr>
<td>10-19</td>
<td>1.85</td>
<td>0.53</td>
<td>.000</td>
<td>6.37</td>
<td>2.26</td>
<td>17.96</td>
</tr>
<tr>
<td>&gt; 20 cigs.day</td>
<td>0.91</td>
<td>0.44</td>
<td>.037</td>
<td>2.48</td>
<td>1.06</td>
<td>5.81</td>
</tr>
</tbody>
</table>

Previous ankle injury

| No | Yes | 0.91 | 0.44 | .037  | 2.48  | 1.06  | 5.81 | .050 |

Constant   | -2.21 | 0.19 | .000  | .11   |        |        |        |      |

R^2 = .06 (Nagelkerke)

Model χ² = 17.848 (p=.003)

Hosmer and Lemeshow Test: χ²=192 (p=.979)

Table 4.5. A comparison of limb of previous ankle injury and limb that developed AKP (note; data on site of ankle injury were missing for subject 5).

<table>
<thead>
<tr>
<th>Subject</th>
<th>Previous ankle injury</th>
<th>AKP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Bilateral</td>
<td>Bilateral</td>
</tr>
<tr>
<td>2</td>
<td>Left</td>
<td>Left</td>
</tr>
<tr>
<td>3</td>
<td>Left</td>
<td>Bilateral</td>
</tr>
<tr>
<td>4</td>
<td>Left</td>
<td>Bilateral</td>
</tr>
<tr>
<td>5</td>
<td>-</td>
<td>Left</td>
</tr>
<tr>
<td>6</td>
<td>Right</td>
<td>Right</td>
</tr>
<tr>
<td>7</td>
<td>Right</td>
<td>Right</td>
</tr>
<tr>
<td>8</td>
<td>Right</td>
<td>Bilateral</td>
</tr>
</tbody>
</table>
Chapter 4. Epidemiology of AKP

4.4 Discussion

4.4.1 Incidence

The incidence of AKP was within the range of 5-15% reported in previous studies (Cowan et al., 1996; Kaufman et al., 1999; Jones et al., 1993; Milgrom et al., 1991). The proportion of the AKP cases that were first onsets demonstrates how a bout of intensive activity for as little as 12 weeks can precipitate symptoms of anterior knee pain in a previously asymptomatic individual. This has implications for studies into risk factors that fail to account for individual activity levels in their research design or analysis.

4.4.2 Age and anthropometry

There was no association between age and AKP. One previous cross sectional study reported a higher risk of PFPS in individuals under 34 years of age (Taunton et al., 2002). This study cannot support or refute this finding due to the homogeneity of ages studied.

Body weight, height and BMI did not show an association with AKP. As highlighted in section 3.1.3, these variables have failed to demonstrate consistent findings in numerous studies regardless of research design and study population. And where differences have been found these have been small and possibly clinically irrelevant. However, it is worth noting that similar to this study, the samples studied have been relatively homogenous in terms of height and weight (Duffey et al, 2000; Kujala et al., 1986; Witrouw et al., 2000). It is possible then that these variables could be a risk factor in outlying cases.

4.4.3 Fitness and prior activity levels

Although aerobic fitness (2.4 km run time) was not a significant risk variable in this study, the least fit individuals had approximately twice the incidence of AKP compared to the fittest group. Unfortunately, the missing data for this variable and small sample size in the least fit category may have reduced the power of this comparison.

Whilst the incidence of AKP was consistently lower in individuals that had undertaken some pre-enlistment activity in running and strength training, the results were not significant. A previous prospective study of all lower extremity overuse injuries found
significant results for this factor (Jones et al., 1993). It should be noted that this study measured general markers of prior activity levels, it is possible that exercise which is specific to the knee extensor mechanism may offer some protection.

Combining the results for aerobic fitness and previous activity levels suggests that relative training load may only be of small importance in this population. Given these findings, it is intuitive to speculate that intrinsic factors may be more dominant predictors of AKP than training errors in populations with a short exposure to activity. However, these results do not preclude the possibility that training errors are a more important risk factor in other populations where the training progression is more extreme.

4.4.4 Smoking

The independence of smoking from lifestyle factors and aerobic fitness agrees with results from previous studies into training injuries (Heir and Eide, 1997; Jones et al., 1993). A cohort study of more than 12,000 subjects found that smokers had higher odds of musculoskeletal pain irrespective of the physical or economic nature of their occupation (Palmer et al., 2003). These results suggest there are explanations other than lifestyle factors, which may be important in this effect. Other suggested explanations are a pharmacological effect on pain perception, pathological changes around a joint through altered nutrition and/or demineralisation of bone, and prolonged tissue healing (Hoogendoorn et al., 2002). Smoking has also been related to arteriolar vasoconstriction and tissue hypoxia so a vascular mechanism could exist (Harvey et al., 2002; Michaud et al., 2003). Confounding should also be considered. For example, neuro-psychological factors that may attract certain persons to smoking may also make these people more likely to report symptoms of pain. Interestingly, subjects that had recently given up smoking were not at an increased risk of AKP compared to non-smokers, which suggests some reversibility. Lincoln and Callahan (2003) examined the effect of smoking on long term disability from musculoskeletal injury and found similar results for subjects that had quit smoking. The authors reasoned that former smokers may have also undergone behavioural changes that contribute to improved health and lowered risk.
4.4.5 Previous musculoskeletal injury

There was an increased risk of AKP in recruits who had suffered an ankle injury in the past 5 years. This effect of previous injury being a risk for re-injury is well documented (Jones et al., 1993; Macera et al., 1989; Walter et al., 1989) but this was the first study to show an association specifically with AKP. De-afferentiation around the joint and inadequate rehabilitation could be explanations for this risk factor. Healthy ankle joint function is critical to locomotion and any dysfunction could affect the normal action of more proximal joints (Kirtley, 2006). Interestingly, the side of previous injury also closely matched the side of AKP, which may hint to such a mechanism. Another explanation may be a subtle alteration in lower extremity kinematics as a result of the injury, although this could also be related to inadequate rehabilitation.

Previous sufferers of AKP had a higher incidence of AKP during training, but the number with a history of AKP was too small to show any statistical significance. Selection and response bias may have existed with this variable since there were very few previous AKP sufferers in the cohort. This was surprising considering the prevalence of AKP in adolescents has been reported as 30% (Fairbank et al., 1984), and suggests some subjects may have been unwilling to report previous symptoms of knee pain, possibly for fear of occupational reprisal.

4.4.6 Limitations

A large proportion of subjects from the original sample were excluded from the analysis. Although the reasons for exclusion such as training failure (e.g. Cowan et al., 1996) and other overuse injury are in accordance with previous studies (e.g. Duffey et al., 2000; Messier et al., 1991; Callaghan and Baltzopoulos, 1994), the number excluded may introduce some bias. The biggest cause of exclusion was ‘other overuse injuries’. These were omitted from the analysis to avoid masking risk factors due to some injuries sharing similar aetiology to AKP, it was thought that this could diminish the hypothesis-generating purpose of this study.

Self-reporting and medical records were used to capture cases. Although this is the most widely used method, it is possible that it introduces some selection bias to the case group through the capture of certain social types more likely to report pain. However, there was
little benefit for subjects to feign injury because it could result in being back-trooped or medically discharged. Likewise, the demanding and intensive nature of Army training makes it very difficult for a recruit to continue training with a knee injury. The alternative to self-reporting was to use a screening method. However, in addition to the practical problems, a concern with screening was the generation of false positives due to post exercise discomfort or aches that are normal with a progressive training programme being interpreted as AKP. Ideally one trained observer would have diagnosed all AKP cases, however, this was considered impractical and too expensive for the large prospective cohort used in this study. In fact, there was 100% agreement in a cross-validation of 12 cases with a single physician, as such the case capture method was considered reliable.

One other possible limitation is the short follow up time used this study. Over a longer training period, other subjects may also have developed AKP. However, the short-follow up time and captive study sample allowed for a near complete injury surveillance with few cases lost to follow-up.

Recall bias may have influenced some of the questionnaire responses which asked recruits to give details relating to past events. Given that Army recruits are by nature a competitive population, pre-enlistment activity levels may have been over estimated. However, categorising these variables would have reduced the effect of the outliers on the analysis. Additionally, the questionnaire was piloted and revised to maximise face and content validity, and results from the reliability study were considered acceptable.

4.4.7 Conclusions

There is a paucity of prospective cohort studies of AKP in the literature (Milgrom et al., 1991; Witrouw et al., 2000) and this was the first to examine social and training history and previous injury characteristics as risk factors for overuse anterior knee pain. There was good evidence from this study linking smoking and previous ankle injury to an elevated risk of AKP. However, the odds ratios should be considered in conjunction with the fit of the regression model which showed that these factors explained only a small proportion of the variance in individuals who developed AKP versus those who remained free of lower limb overuse injury. Despite this, the purpose of the study was to explore associations with
AKP and not to develop a predictive model. There is no theoretical basis or evidence to suggest that these factors may be strong predictors of AKP.

Despite the wealth of studies into risk factors and the treatment of AKP, the aetiology of this condition remains poorly understood. AKP is likely to be caused by a complex interaction of intrinsic risk factors, extrinsic exposures, and inciting biomechanical/physiological mechanisms. Factors such as smoking status, ankle injury and activity may be mediating variables that alter the strength of a relationship between another factor and AKP. Future research into AKP may wish to consider some of the variables measured in this study as covariates in the design and/or analysis research stage. For example, a randomised controlled trial into an intervention for anterior knee pain may wish to include smoking as a covariate to adjust for in the analysis, and a case-control study into risk factors may wish to consider a minimum activity level for control group inclusion to avoid individuals who may sustain AKP upon taking up activity entering the control group. These approaches may minimise the risk of masking true risk factors due to the confounding effect of other significant covariates. Future experimental research may also wish to examine the mechanisms for the significant risk factors highlighted in this study.
Chapter 5

Gait analysis method development

5.1 Introduction

Chapters 6-9 of this thesis are concerned with the relationship between lower extremity gait kinematics and anterior knee pain. Kinematics is a broad area of study, applied to answer many questions in biomechanics, and the kinematic methodology employed in measuring gait is an ongoing area of research in itself. The accuracy of kinematic measurement is dependent on a number of factors, of which many can be carefully selected by the researcher to minimise error. These need to be discussed and addressed in the context of the requirements and constraints of this programme of research.

The objective of this chapter is to detail the rationale and key issues concerning the kinematic methodology employed for the studies contained in the rest of this thesis. This section contains a description and discussion of the kinematic model used, a summary of a literature review into the differences between treadmill and overground locomotion, the choice of filter parameters for signal processing and a description and brief discussion of the normalisation procedure to enable between subject comparisons. Finally a brief report from a between-day reliability study of the kinematic protocol is given.

5.2 Data acquisition and kinematic model

5.2.1 Data acquisition

A six camera opto-electronic motion capture system (ProReflex, Qualisys Medical AB) was used to capture three dimensional (3D) marker coordinates. The resolution of the
system is 1:60,000 pixels, which provides an average absolute system accuracy of ±0.2mm for a 1m³ measurement volume. The actual accuracy of the system is dependent on the effectiveness of the calibration. The system uses a dynamic calibration sequence and direct linear transformation approach to scale the 2D coordinates of each camera to the required measurement volume. The system was set-up to ensure calibrations for each camera had average residuals less than 1.5mm (<0.5%). Factors such as lens distortion, whilst corrected using linearization files, can affect the accuracy of the system at different locations in the measurement volume. The approximate calibrated measurement volume used for the prospective gait study was 1.2m x 0.5m x 1.2m. The accuracy of the system at identifying the known length of a rod throughout this measurement volume was checked (ANNEX B). The accuracy ranged from 0.02 to 0.65% for all points checked. These instrumental errors were considered excellent.

5.2.2 Kinematic model

The selection of the model to generate lower extremity kinematics was based upon the requirements of the prospective cohort study reported in Chapter 6. Firstly, and stemming from the literature review, the study was designed to be explorative and hypothesis generating, so the model had to be able to measure all three angular rotations of the hip, knee and ankle joint. Secondly, since the study was prospective and used a military sample, a large sample size was required and the time available for collecting data was insubstantial. It was necessary therefore that the kinematic model could be administered quickly. Lastly and most importantly, the model had to be valid, reliable and invoke minimal disturbance to the subject’s movement patterns. A 3 degree-of-freedom link segment model was ultimately deemed suitable.

The model selected was a derivative of the widely used Helen Hayes marker set (HH), which has been described and validated in previous papers (Davies et al., 1991; Kadaba et al., 1989; Kadaba et al., 1990). The main difference between the HH set and the HH derivative skin model selected for this study is the replacement of the thigh and shank wands with skin placed markers on the supra-patella and tibial tuberosity. A concern with the traditional HH marker set was the requirement for the shank wand to be co-linear to the knee joint line and lateral maleolus marker, to settle the knee and ankle joint centre, and the thigh wand to be co-linear to the greater trochanter and knee joint line marker, to settle
Chapter 5. Gait analysis method development

the knee flexion-extension axis of rotation. Marker application errors here will cause a knee flexion/extension offset (Kirtley, 2002) and abduction-adduction artefacts (Kadaba et al., 1990; Kirtley, 2002; Ramakrishnan et al., 1991; Zetterberg, 1999) due to the sequence dependency of the euler angle calculations and cross talk from the flexion to adduction axes. To ensure correct wand alignment, laboratories use mirrors and data verification checks prior to data collection which can be labour intensive (Kirtley, 2002). This problem is overcome with the modified skin based HH set because all the marker locations can be anatomically determined. The risk of the wands being struck by arm movement is also diminished. And although not quantified, the locations of the replacement markers for the wands and the stiffer attachment interface may make them less prone to artefacts caused by underlying soft tissue movement and high skeletal accelerations that occur from impact forces during running. Both models were used in pilot and familiarisation sessions before the main study and the skin system was considered easier to use and standardise. A comparison of the joint angles obtained with this model versus those reported in previous studies using other models and skeletal markers is contained in section 6.4.1.2.

5.2.2.1 Joint centre calculation
The marker set-up and application are described in figure 5.1. The hip joint centre is calculated using a technical coordinate system defined by the ASIS and sacrum marker, where the distance between the ASIS markers is used as a scaling parameter to adjust for individual anthropometric differences. The knee and ankle joint centres are calculated using a technical frame settled by the knee joint line, tibial tuberosity, and lateral malleolus marker. The distance between the knee joint line and lateral malleolus marker is used to scale the parameters. The regression equations to locate the joint centres have been described by Vaughan (1992).

5.2.2.2 Anatomical axis and euler angle calculations
The lower extremity is modelled as four segments, the pelvis, thigh, shank and foot. Each segment is described by an embedded right handed Cartesian coordinate system. These local coordinate systems are anatomically located to describe clinically meaningful joint rotations. The z-axis of the thigh segment is defined by the knee and hip joint centre. The x-z plane encompasses the knee and hip joint centres and the patella marker. The y-axis is orientated orthogonal to this plane creating a right-handed Cartesian coordinate system. The z-axis of the shank is defined by the knee and ankle joint centres, the tibial tuberosity
marker then settles the x-z plane, with the y-axis perpendicular to both the x and z axis. The x-axis of the foot segment is defined by the heel and toe marker, the x-z plane then encompasses the x axis and the ankle joint centre and the y-axis points to the right and perpendicular to this plane. These local segment definitions are illustrated and described in figure 5.2.

Sacrum: The centre of the sacrum at the height were it meets the iliac crest

ASIS: On the apex of the ASIS

Supra-patella: Quadriceps muscle relaxed. Mark the centre of the patella along a line connecting the most medial and lateral edge. The marker is placed 10mm above the superior border along a line connecting the centre of the patella with the ASIS.

Knee joint line: The middle point of the knee joint line

Tibial tuberosity: The centroid of tibial tuberosity

Lateral malleolus: Most prominent point at lateral malleolus

Calcaneal: 35mm* superior from the floor in the medio-lateral centre of the tuberosity

Metatarsal: 35mm* superior from floor Between the 2nd and 3rd metatarsal

*The calcaneal and toe marker were placed at a nominal height to standardise the foot segment parallel to the floor.

Figure 5.1. Description of marker placements for the modified Helen Hayes skin model.

The joint angles for the hip, knee and ankle are calculated using the Euler method, where the distal segment is referenced to the proximal segment (e.g. the thigh is referenced to the pelvis and so forth). The first rotation of the distal segment is about the reference y-axis, this creates a newly located coordinate system which can be denoted as X1, Y1 and Z1. The second rotation is about the newly orientated X1 axis, creating a new coordinate system for the moving segment denoted as X2, Y2, Z2. The final rotation then occurs about the Z2 axis. This gives a Y, X, Z rotation sequence, corresponding to flexion (+)/extension (-), adduction (+)/abduction (-) and internal rotation (+)/external rotation (-). This approach has been described by Kadaba et al. (1990) and Ramakrishnan et al. (1991)
and is widely used by the biomechanics community (Robertson and Caldwell, 2004) and is equivalent to the joint coordinate system (Cole et al., 1993; Grood and Suntay, 1983).

Since the foot is relatively fixed during stance until late propulsion, the ankle rotation in the transverse plane actually reflects the rotation of the tibia about the foot (McClay and Manal, 1997), accordingly this angle is subsequently referred to as tibial rotation.

![Diagram of anatomical coordinate systems](image)

**Figure 5.2.** Definitions for the local embedded anatomical coordinate systems of the modified HH skin model. RHCS = Right hand coordinate system.

### 5.2.2.3 Sensitivity to marker placement errors

Link segment models are not true 6 degree-of-freedom models, and are thus vulnerable to error propagation. This occurs because the calculation of an embedded distal segment coordinate system is dependent on the calculation of the proximal segment joint centre. This interdependency means that errors in locating the pelvic markers can cause errors in the hip and knee joint angles. These errors mostly affect the angles in the frontal and transverse planes. For example, a 10mm shift in the medio-lateral position of the ASIS marker can cause an offset of the knee abduction angle that is greater than 5% (Zetterberg, 1999). The rotations in the transverse plane are particularly prone to offsets caused by medio-lateral deviations in the supra-patella, tibial tuberosity, heel and toe marker. However, these offsets result in an approximate constant shift in the joint angle waveform.
(Zetterberg, 1999). Constant shifts can be adjusted for, although at the expense of some biomechanical information (this is discussed in section 5.6.4). Of most concern is the ‘cross talk’ effect caused by misaligned axes. The second Euler rotation (x-axis: abduction-adduction) is most sensitive to these effects because the majority of joint angle rotation occurs in the sagittal plane. This error is a function of the magnitude of y-axis rotation as well as the error in axis alignment due to incorrect model assumptions and marker placement error. Kadaba et al. (1990) illustrated the effect of medial and lateral deviations in the knee flexion axis to the knee abduction plot during walking (figure 5.3). This emphasises the need for standardisation and care in the application of markers, and caution in interpreting the results of abduction-adduction plots from kinematic models.

![Figure 5.3. The effect of incremental 5° medial and lateral shifts in the knee flexion axis of rotation on the knee abduction joint angle during walking (In Kadaba et al., 1990).](image)

5.3 Gait Protocol: Treadmill and unshod ambulation

5.3.1 Treadmill versus overground locomotion

5.3.1.1 General differences

Treadmills offer a controlled locomotor speed, which make them ideal for locomotion research. There has been a substantial quantity of research examining the parity between treadmill and overground locomotion. Despite this, the notion of whether treadmill gait is representative of overground gait is still a matter of contention.
There is general agreement that treadmills induce a systematic reduction in stride length and an increased cadence (Alton et al., 1998; Lemke et al., 1995; Nelson et al., 1972; Schache et al., 2001). There is also consensus that the shoe-sole angle at initial contact is less during treadmill ambulation (Nigg et al., 1995; Wank et al., 1998) causing a flatter foot contact pattern. Nigg et al. (1995) examined the effects of treadmill type and found a large mean difference in the shoe-surface angle (15.9° vs 8.0°). It is thought that subjects adopt a flatter touchdown because it is perceived as more stable.

There is conflicting evidence for the effect of treadmill ambulation on foot eversion. Nigg et al. (1995) found a small increase in rearfoot eversion during treadmill running (-20.4° vs -17.8°) while Lemke et al. (1995) found no difference in the magnitude of ankle eversion.

Results for knee joint movement have also been equivocal. Some studies have found no adaptations (Matsas et al., 2000; Nigg et al., 1995), while others have found increased knee flexion angles during treadmill walking (Alton et al., 1998, Wank et al., 1998) and reduced range of knee flexion during treadmill running (Savelberg et al., 1998). In studies that have found a difference in knee joint angle, these differences have been small e.g.; 2.4° (Wank et al., 1998). The clinical significance of differences of this size is unknown. No published studies on the effect of treadmill locomotion on the transverse and frontal plane knee movement could be found.

Hip ROM has been shown to be greater during treadmill running (Alton et al., 1998), this occurred predominantly due to greater hip extension at toe off (Schache et al., 2001). Only one published study has reported hip rotation and adduction adaptations to treadmill running, and no differences were found (Schache et al., 2001).

Two other aspects of gait analysis that have been measured for similarities between treadmill and overground running, are the vertical ground reaction force (GRF) (White et al., 1998) and the electromyographic (EMG) patterns of the lower limb muscles (Wank et al., 1998). The pattern of the vertical GRF was very similar between surfaces, although, the magnitude was slightly smaller on a treadmill. The EMG study showed similar waveforms and only very small differences in integrated EMG values.
5.3.1.2 Effect of treadmill experience

Nigg et al. (1995) demonstrated that the shoe-surface angle changed in a systematic manner from overground to treadmill running, whereas other measures of ankle movement such as eversion and tibial-floor angle showed random effects within subjects. However, when subjects with no treadmill running experience were removed from the analysis, the systematic effect of shoe-surface angle was removed. Further, when shoe type was controlled, there were still significant differences in the other angles measured but these were now small (Nigg et al., 1995). The removal of these systematic differences was possibly due to enhanced treadmill familiarisation of the experienced runners. This study provides useful information on potential reasons for the response to treadmill running between subjects, however, some of the differences should be interpreted with caution. The design of this study was complex, there were 22 subjects, one factor of running experience with 2 levels, one factor of treadmill with 4 levels, one factor of footwear with two levels and another factor of running speed with four different levels. And only one representative stride was used for each level of each factor. One stride may not be representative of a subjects gait, which may explain some of the random variability seen between surfaces.

5.3.1.3 Effect of familiarisation

Familiarisation to treadmill running may improve the validity of treadmill locomotion. Matsas et al. (2000) examined the differences between treadmill and overground walking in 16 subjects with no previous treadmill experience. Gait was analysed every 2 minutes of treadmill walking. Results showed no differences between the overground and treadmill condition in spatio-temporal variables after 6 minutes, and no differences in knee joint angles after 4 minutes. Another earlier study found slightly less conservative results, suggesting that 4 minutes was sufficient for familiarisation (Taylor et al., 1996). Matsas et al. (2000) suggest that some of the differences found between treadmill and overground gait may be due to lack of familiarisation.

5.3.1.4 Other causes of differences

Mechanical and perceptual differences have been attributed as reasons for treadmill adaptations. Mechanical effects are thought to occur due to energy transfer between the belt and subject. Theoretical studies have suggested that the mechanics of treadmill and overground gait are the same if the belt speed is constant (Ingen Schenau, 1980). Sensory stimuli, such as treadmill height and belt size, that are involved in perceptual processes and
influenced by treadmill design are thought to cause some adaptations to treadmill running (Nigg et al., 1995). One study suggested that belt slippage is responsible for certain treadmill adaptations. Specifically, intra-stride belt speed variation (ISBSV) was dependent on the motor size and mass of subject, and increased ISBSV was associated with greater kinematic differences (Savelberg et al., 1998).

5.3.2 Footwear

There is enormous scope for footwear modification and design. For example, the mechanical properties of the sole can differ in terms of friction, stiffness, and flexion point location. The material properties of the midsole can be altered to change the elastic modulus and load attenuation properties. Geometric aspects such as heel lift and flare can also be altered. Studies have shown that aspects of footwear design can affect running gait, for example, factors such as midsole hardness and heel flare have been shown to influence ankle eversion (Clarke et al., 1983; Hamill et al., 1992; Nigg and Morlock, 1987). The fit of the shoe and the type of insole could also affect gait. Current opinion also advocates that there may be a medium and long term adaptation to footwear modification that is effectively ignored in studies where subjects are first exposed to the footwear during the actual testing session (Mundermann, 2004). There are also technical problems associated with measuring foot movement in shod conditions (Stacoff et al., 2000c). Markers have to be placed on the shoe or the shoe has to be adapted to accommodate marker placement on the skin. If markers are placed on the shoe, then movement of the markers will reflect the movement of the shoe. Stacoff et al. (2000c) demonstrated that this overestimates foot eversion angles.

It has been demonstrated that subjects systematically adopt a flatter foot contact pattern when running unshod (De Wit et al., 2000). This adaptation is thought to be a protective mechanism to limit the pressure under the heel and is thought to affect ankle eversion. However, whilst skin/shoe marker based studies have shown reduced calcaneal eversion during barefoot running (e.g. Stacoff et al., 1991), a bone pin study found very small and insignificant differences in ankle eversion and tibial rotation angles between unshod running and running in a neutral shoe (Stacoff et al., 2000c).
5.3.3 Conclusions and recommendations

Despite the belief that the findings from treadmill locomotion cannot be translated to overground locomotion, there is little evidence to support this view. Most of the effects shown have been small and systematic. Further, any random effects were reduced in experienced runners and minimised following a short period of familiarisation. The use of a treadmill improves the efficiency of data collection and enables a larger sample size for the prospective study. It also controls the speed of locomotion, a factor which can affect kinematics (Milliron and Cavanagh, 1991). Whilst the use of a treadmill may be considered a delimitation, the advantages it offered were considered more critical to the outcome of the prospective study. Variables that showed a systematic adaptation to treadmill gait should not influence the outcome of the results. However, random effects could have an influence. To minimise these, only persons with prior experience of treadmill running were selected for the main study, and a 5 minute familiarisation session was included in the protocol. Further a Woodway PPS 55med treadmill was used. This treadmill has characteristics such as a toothed driving mechanism with zero belt slippage and a large 12 mm thick rubber running surface that should minimise any potential mechanical or perceptual adaptations. The relevant technical specifications are as follows; Motor power: 1kW; Mat size: 1560 x 550mm; Mat hardness: 40shoreD. It is argued that this set-up and procedure should provide findings that would be replicated in a similar study using overground locomotion.

It was decided to use a barefoot protocol to capture the kinematic data since it was reasoned that this would provide a more accurate technical assessment of foot kinematics. To discourage a subject-specific response to barefoot running, normal footfall patterns were emphasised during the familiarisation. Further, the treadmill mat was constructed of vulcanised rubber, meaning that it was softer than traditional treadmill mats and outdoor running surfaces (e.g. concrete and asphalt). It was expected that with familiarisation, if any adaptation to unshod locomotion occurred, this would predominantly be systematic and not affect the outcome of the study. Subjects that took part in pilot tests and the reliability study (section 5.6) commented that they felt comfortable walking and running unshod on the Woodway treadmill.
5.4 Signal processing

The aim of filtering or smoothing routines is to minimise artefact and random errors caused by electronic noise, marker flickering, marker shape distortion, wand vibration and digitising (Chiari et al., 2005). Most of these sources of noise occur at higher frequencies than that contained in human movement and so can be largely removed through signal conditioning.

5.4.1 Residual analysis method

A residual analysis, as described by Winter (1990), was undertaken to determine the cut-off frequency for filtering the raw kinematic data. This method is widely used and easy to interpret. Four subjects were randomly selected (3 controls, 1 PFPS) from the prospective cohort study sample (see section 6.2). One complete gait contact for walking (1.8 m s⁻¹) and running (2.78 m s⁻¹) with 5-6 extra frames at each end was filtered using a Butterworth low pass 4th order zero lag filter. This was done using cut off frequencies of 0.5 Hz, 1 Hz and each 1 Hz thereafter up to 20 Hz. The residual analysis applied to the tibial tuberosity, heel and toe marker in the x, y and z-axis, since the frequency component may be different on more distal locations and in different planes. The residual plots were inspected before choosing which points to take a regression line between. These points coincided with the linear portion of the curve plots (figure 5.4). The points taken for the regression line were all between 10 and 20 Hz. The cut-offs were selected based on where the intercept met the root mean square (RMS) residual line along the x-axis. This was calculated using the tabulated RMS values for each cut-off, finding the two cut-off frequencies where the intercept fell between and then calculating a ratio between the two corresponding integers.

5.4.2 Results and Discussion

There was a higher frequency component in the running signal compared to the walking, this is shown by the shift in the RMS curves to the right (figure 5.4) and the higher recommended cut off frequencies shown in Table 5.1. The residual analysis method gave suggested cut off frequencies of 10 Hz for walking and 12 Hz for running. The choice of 10 Hz for walking is in agreement and falls within the range of 6-10 Hz used in previous work (e.g. Ghoussayni et al., 2004; Holden et al., 1997; Reinschmidt et al., 1997ab; Scott
and Winter, 1991). Likewise, a 12Hz cut off for running is within the range of 8-22Hz used by previous studies (Hamill et al., 1992; Hamill et al., 1999; McClay and Manal, 1997; Stacoff et al., 2000abc; Savelberg et al., 1997; Stergiou and Bates, 1997; Stergiou et al., 2001ab, Van Woensel and Cavanagh, 1992).

An examination of the effect of different cut off frequencies on the calculated joint angle kinematics supports the choice of these cut off parameters. Figure 5.5 shows that when the cut off frequency is lower than 10Hz for walking and 12Hz for running, there is a decrease in the peak values. It is also clear that lower cut off frequencies result in altered joint angle coordination patterns. This is highlighted in the knee internal-external rotation curve for running (figure 5.5), where a lower cut-off frequency resulted in a unimodal rather than a bimodal curve. Lastly, it is evident that choosing a slightly higher cut off frequency (e.g. 15Hz) results in an irregular and noisy signal.

![Figure 5.4](image)

**Figure 5.4.** An exemplar residual plot (subject 4) for the tibial tuberosity marker along the x, y, z axis for walking (solid lines) and running (dashed lines). Also given is an example of the regression line used to calculate a cut off frequency, in this case for the y axis walking trial.

Although filtering has been widely discussed in the literature, there is no consensus on the optimal choice of filter or smoothing routine or on how to select the smoothing parameters. This is highlighted by previous work on running kinematics. Although most studies used a Butterworth 4th order zero lag filter, a wide range of different cut off frequencies were selected using a variety of methods for selecting the cut off frequency (Hamill et al., 1992;
Hamill et al., 1999; McClay and Manal, 1997; Stacoff et al., 2000abc; Savelberg et al., 1997; Stergiou and Bates, 1997; Stergiou et al., 2001ab, Van Woensel and Cavanagh, 1992). Algorithms are commonly used to optimise and automate the selection of cut off parameters, which has the advantage of standardising the cut-off selection between observers. However, most investigators agree that the parameters chosen should be based on careful inspection of the data and consideration for the problem to be addressed. In this manner, algorithms should not be used without also evaluating the output in the context of the protocol and the aims of the research.

There is a balance when choosing a cut off frequency, which has led many experts to advocate an interactive approach to signal processing. However, it is clear that for these data a higher cut off frequency introduces random irregularity to the signal, whilst a lower cut off distorts the magnitude, shape and timing of the signal. Thus, a 10 and 12Hz cut off seems to satisfy both the criteria of avoiding a violation of the validity of the study (over smoothing), and avoiding noisy and erroneous derivative data (under smoothing).

Table 5.1. Cut-off frequencies as determined by a residual analysis (Winter, 1990) for the walking and running trials for the 4 randomly selected subjects. KEY: Tib_tub = tibial tuberosity, C= control subject; P=PFPS subject.

<table>
<thead>
<tr>
<th>Marker and axis</th>
<th>Walking</th>
<th>Running</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 (C)</td>
<td>2 (C)</td>
</tr>
<tr>
<td>Tib_tub_x</td>
<td>8.0</td>
<td>9.7</td>
</tr>
<tr>
<td>Tib_tub_y</td>
<td>6.7</td>
<td>8.9</td>
</tr>
<tr>
<td>Tib_tub_z</td>
<td>6.7</td>
<td>9.6</td>
</tr>
<tr>
<td>Heel_x</td>
<td>8.5</td>
<td>8.0</td>
</tr>
<tr>
<td>Heel_y</td>
<td>8.4</td>
<td>8.3</td>
</tr>
<tr>
<td>Heel_z</td>
<td>8.7</td>
<td>9.5</td>
</tr>
<tr>
<td>Toe_x</td>
<td>8.3</td>
<td>8.0</td>
</tr>
<tr>
<td>Toe_y</td>
<td>8.6</td>
<td>9.6</td>
</tr>
<tr>
<td>Toe_z</td>
<td>9.0</td>
<td>9.8</td>
</tr>
</tbody>
</table>
5.5 Normalisation

5.5.1 Determination of initial contact and toe off

Four commonly used methods for determination of gait events have been described in the literature. These are: (i) force platform; (ii) foot switches (iii) kinematic algorithms and (iv) visual inspection. The force platform method is viewed as the gold standard. Kinematic algorithms have been described by Hreljac et al. (2000b) and are based on velocity thresholds for markers along the vertical axis. Wall and Crosbie (1996) showed
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the accuracy of temporal-spatial gait data to be within 0.02s when comparing visual detection with a force platform during barefoot walking. This method was more accurate and reliable than foot switches. Ghoussayni et al. (2004) showed zero mean difference between the visual inspection and force platform method for determination of heel contact during walking. For the toe off event / phase there was a marked difference between these two methods. However, this was predominantly systematic and a comparison between the visual inspection and kinematic algorithm technique showed no difference. Thus, kinematic algorithms seem to offer no advantages in terms of validity and reliability over visual inspection methods.

Kinematic algorithms have only been described for walking, no published algorithms for overground or treadmill running could be found. Further, on inspection of some sample data from treadmill running, there were differences in the x, y, z heel and toe signals between individuals that had different footfall patterns. Any algorithm would thus have to account for the range of subject foot contact strategies. Overall, the visual inspection method was deemed acceptable to determine initial contact and toe off events.

5.5.2 Interpolation

Gait data has commonly been normalised to percent of stride or to percent of foot contact. Since the kinematics during the stance or weight bearing phase of the gait cycle is the focal interest in this work program, all data were normalised to percent of the stance phase. This is common in other locomotion studies of lower limb overuse injury (Hamill et al., 1992; Stergiou and Bates, 1997) so will be useful for comparative purposes.

A Matlab script was written to normalise the displacement data to 100 points. A cubic spline interpolation was used as opposed to a Fourier transform. This is because the latter method causes 'end effects' when a matrix of less then 100 points is used and the stance portion of the run data consisted of approximately 60 frames or points.
5.6 Reliability study of the gait protocol and Helen Hayes skin model

5.6.1 Introduction

A limitation of some epidemiological studies is the lack of information regarding the reliability of the measurement variables. This is particularly relevant to gait analysis, where there are a number of sources of variability. Variability can be categorised into equipment error (e.g. accuracy, non-linearity, time dependency and quantisation error), marker application errors, kinematic model errors (e.g. scaling properties to calculate joint centres and axis alignment) and data reduction errors (i.e. information lost in smoothing/ filtering and normalisation errors). There is also some inherent biological variability (Newell et al., 2006), as such several strides are typically captured and a mean produced. Ultimately, the aim is to capture data that are representative of the individual’s gait.

Differences in observer experience, equipment configuration and protocol procedures can cause different results between gait laboratories, which is why most centres reference their own gait database. It is thus important to assess the between-day or intra-observer reliability of the gait analysis protocol.

The purpose of this study was to determine the between-day reliability of the kinematic model and gait protocol that was used for the studies contained in this thesis. This includes a reliability assessment of the temporal-spatial data, kinematic waveforms and joint angle summary statistics.

5.6.2 Method

Five healthy male subjects free of musculo-skeletal injury (mean age: 36yrs; mean height: 1.78m; mean weight: 79.6kg) volunteered and gave written informed consent to participate in the study. All subjects had experience of treadmill running.

Each subject was tested on two separate test days, 48 hours apart. On arrival, each subject undertook 5 minutes of treadmill walking and running for familiarisation purposes. Anatomical landmarks were located (Table 5.1) and 19mm diameter retro-reflective spheres were applied to the skin using double sided sticking tape.
Following preparation, each subject performed one minute of walking and one minute of running at 6.5km.h⁻¹ (1.81m.s⁻¹) and 10km.h⁻¹ (2.78m.s⁻¹) on a Woodway treadmill. Data were sampled at 240Hz for 15 seconds from 6 opto-electronic cameras (Qualisys Medical, AB). The cameras were calibrated before each testing session (calibration details are contained in section 5.2.1 and in ANNEX B). This gave approximately 15 full strides for walking and 20 full strides for the running speed.

For each subject and test day, six strides were selected for analysis. The strides were selected interactively based on those that had least marker occlusion and where the marker occlusion occurred only in one direction along the axes of the global coordinate system. A spline interpolation was used to fill missing coordinate data, although generally the data were of good quality. A few padding frames either side of the stride were exported so that end effects introduced from filtering did not affect the analysed data. The data were then filtered using a Butterworth 4th order zero lag digital filter with a 10Hz cut-off for walking and a 12Hz cut-off or running (see section 5.4). Data were normalised to percent of stance using a cubic spline interpolation (see section 5.5).

The following joint angles were decomposed and assessed for reliability: Hip adduction/abduction, hip internal/external rotation, knee flexion/extension, knee adduction/abduction, knee internal/external rotation, ankle dorsiflexion/plantar flexion, ankle inversion/eversion and tibial internal/external rotation. These 8 angles were assessed because they were analysed for an association with patellofemoral pain syndrome in the main clinical studies (Chapters 6-9), the justification of which is outlined in section 6.1.

5.6.2.1 Analysis
Temporal spatial data
Stride length, cadence (Hz) and stance phase proportion were calculated. Stride length (m) was determined as the ratio of speed (m.s⁻¹) divided by cadence. To allow for a comparison with previous studies, the coefficient of variation (CV) statistic was calculated, this was done for each subject and then averaged.

Entire waveform similarity
In describing the similarity between joint angle curves, it has been noted that simple summary statistics such as peaks are not satisfactory because they discard other data in the
waveform (Kadaba et al., 1989). Thus, to compare the overall between-day reliability of
the kinematic waveforms the adjusted coefficient of determination was used, this is given
by:

\[
R^2_a = 1 - \frac{\sum_{i=1}^{M} \sum_{j=1}^{N} \sum_{t=1}^{T} (Y_{ijt} - \mu_t)^2 / T(MN - 1)}{\sum_{i=1}^{M} \sum_{j=1}^{N} \sum_{t=1}^{T} (Y_{ijt} - \mu)^2 / (MNT - 1)}
\]  

(1)

where \(Y_{ijt}\) is the \(t\)th time point of the \(j\)th run on the \(i\)th test day. \(M\) is the \(n\) of test days (2),
\(N\) is the number of trials (6) and \(T\) is the number of samples (100). \(\mu_t\) is the average at time
point \(t\) over \(NM\) gait cycles (12) and is given by:

\[
\mu_t = \frac{1}{MN} \sum_{i=1}^{M} \sum_{j=1}^{N} Y_{ijt}
\]

(2)

\(\mu\) is the total mean over all time given by:

\[
\mu = \frac{1}{MNT} \sum_{i=1}^{M} \sum_{j=1}^{N} \sum_{t=1}^{T} Y_{ijt}
\]

(3)

Equation (1) shows that the numerator of the ratio represents the variance about the mean
at a particular time point over both test days. The denominator represents the total
variability about the grand mean of both test days. If the joint angle curves are similar over
the two test days this ratio will tend to zero and the coefficient of multiple determination
will tend to 1. The positive square root of \(R^2_a\) is termed the coefficient of multiple
 correlation (CMC). For each individual and each joint angle curve the between day CMC
was calculated. This was then expressed as a mean and standard deviation for all subjects.

On inspection of the subject mean curves for each test day, there was an offset in the
frontal and transverse plane joint angles from day 1 to day 2. The shape of the waveforms
remained similar but the entire curve had shifted up or down by a variable amount due to
marker placement error. In order to remove the effect of this offset, the subject mean value
for the joint angle on each test day was subtracted from each trial (daily mean subtraction – DMS). CMCs were then calculated to examine this offset effect. Group mean ensemble joint angles were also calculated for each test day and plotted. CMCs were also calculated to quantify the similarity of these curves.

**Summary statistics**

Peak values were obtained from each joint angle curve during stance. This was done for all angles except the dorsi-flexion waveform during walking, where a discernible peak could not be determined for all subjects. The time to peak was calculated for knee flexion and ankle eversion. These two temporal measurements were taken based on previous research into the timing disparity between these two events (Bates et al., 1979; Hamill et al., 1992; McClay and Manal, 1997). This relationship is hypothesised to be associated with anterior knee pain in accordance with the mechanism cited by Tiberio (1987) (see section 3.3.1). Other time to peak variables that were considered important in this relationship such as the timing of peak knee and tibial internal rotation were not calculated because of the tendency for a bimodal pattern in a proportion of trials and ultimately arbitrary decision in selecting which peak to use. To examine the reliability of these summary statistics, Bland and Altman's 95% limits of agreement (LOA) were calculated (Bland and Altman, 1986).

5.6.3 **Results and Discussion**

5.6.3.1 **Temporal-spatial data**

The temporal-spatial data showed excellent reliability, the average between day CV for all subjects was below 3.1% for all variables during walking and running (Table 5.2). These results were better than previously published values for treadmill running (Schache et al., 2002). Although this result doesn't validate the visual inspection procedure used for normalisation, it indicates that the method used to detect initial contact and toe off was well standardised.

5.6.3.2 **Sagittal plane kinematics**

As expected, movements in the sagittal plane showed excellent reliability (Table 5.3 and 5.4). All the CMC values exceeded .96 for walking and running, even without the DMS adjustment. Although the CMC is a dimensionless measure, Schache et al. (2002) consider values greater than .8 to indicate good reliability.
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Table 5.2. Mean, sd and between day coefficient of variation (CV) values for the temporal-spatial variables for walking and running

<table>
<thead>
<tr>
<th></th>
<th>Day 1 mean</th>
<th>Day 1 sd</th>
<th>Day 2 mean</th>
<th>Day 2 sd</th>
<th>CV</th>
<th>sd</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Walk</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cadence (Hz)</td>
<td>1.11</td>
<td>0.03</td>
<td>1.10</td>
<td>0.03</td>
<td>1.3</td>
<td>0.5</td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>1.63</td>
<td>0.04</td>
<td>1.64</td>
<td>0.04</td>
<td>1.3</td>
<td>0.5</td>
</tr>
<tr>
<td>Stance phase proportion (%)</td>
<td>54.4</td>
<td>0.89</td>
<td>54.7</td>
<td>0.77</td>
<td>1.2</td>
<td>0.6</td>
</tr>
<tr>
<td><strong>Run</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cadence (Hz)</td>
<td>1.37</td>
<td>0.06</td>
<td>1.37</td>
<td>0.05</td>
<td>1.5</td>
<td>0.1</td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>2.04</td>
<td>0.09</td>
<td>2.03</td>
<td>0.07</td>
<td>1.5</td>
<td>0.1</td>
</tr>
<tr>
<td>Stance phase proportion (%)</td>
<td>30.3</td>
<td>3.2</td>
<td>30.6</td>
<td>3.8</td>
<td>3.0</td>
<td>1.5</td>
</tr>
</tbody>
</table>

5.6.3.3 Frontal plane kinematics

Hip adduction showed good reliability with CMC values above .8 for walking (Table 5.3) and running (Table 5.4). However, for one subject the CMC during running was only .55, but with the DMS adjustment this improved to .7 and increased the group mean CMC to .88. Similar findings were found by Kadaba et al. (1989) for hip adduction. CMC values for knee abduction were also good, but improved by more than 10% to approximately .9 following a DMS adjustment. Ankle eversion waveforms showed poor reliability, although they were better than previously reported values (Kabada et al., 1989; Steinwender et al., 2000). Much of the reason for this poor reliability was due to marker offset errors, since with a DMS adjustment the CMCs approached .8 (Table 5.3-5.4). The higher CMC values reported in this study compared to earlier studies may be due to the speed standardisation that accompanies a treadmill protocol. Technological improvements in motion capture equipment may also be partly responsible.

5.6.3.4 Transverse plane kinematics

Although the hip and knee transverse rotation waveforms showed relatively poor CMC values, these were above .75 following the DMS adjustment. The CMC values for tibial rotation were excellent, the mean values for walking and running were greater than .93 after DMS adjustment (Table 5.3-5.4).
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Table 5.3. Between day mean adjusted CMCs (sd) for each joint angle for the walking trials with and without the mean offset adjustment (DMS). Also presented are the between day CMCs for the ensemble waveforms (Group).

<table>
<thead>
<tr>
<th>Joint</th>
<th>No DMS</th>
<th>DMS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>sd</td>
</tr>
<tr>
<td>Hip</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Add-abd</td>
<td>.90</td>
<td>.09</td>
</tr>
<tr>
<td>Int-ext. rot.</td>
<td>.62</td>
<td>.17</td>
</tr>
<tr>
<td>Knee</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flex-ext.</td>
<td>.99</td>
<td>.01</td>
</tr>
<tr>
<td>Add-abd</td>
<td>.81</td>
<td>.09</td>
</tr>
<tr>
<td>Int-ext. rot.</td>
<td>.62</td>
<td>.14</td>
</tr>
<tr>
<td>Ankle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dors-plant.</td>
<td>.96</td>
<td>.01</td>
</tr>
<tr>
<td>Inv-eve.</td>
<td>.65</td>
<td>.22</td>
</tr>
<tr>
<td>Tibia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Int-ext. rot.</td>
<td>.82</td>
<td>.06</td>
</tr>
</tbody>
</table>

Table 5.4. Between day mean adjusted CMCs (sd) for each joint angle for the running trials with and without the mean offset adjustment (DMS). Also presented are the between day CMCs for the ensemble waveforms (group).

<table>
<thead>
<tr>
<th>Joint</th>
<th>No DMS</th>
<th>DMS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>sd</td>
</tr>
<tr>
<td>Hip</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Add-abd</td>
<td>.83</td>
<td>.19</td>
</tr>
<tr>
<td>Int-ext. rot.</td>
<td>.66</td>
<td>.27</td>
</tr>
<tr>
<td>Knee</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flex-ext.</td>
<td>.97</td>
<td>.03</td>
</tr>
<tr>
<td>Add-abd</td>
<td>.79</td>
<td>.09</td>
</tr>
<tr>
<td>Int-ext. rot.</td>
<td>.60</td>
<td>.24</td>
</tr>
<tr>
<td>Ankle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dors-plant.</td>
<td>.98</td>
<td>.01</td>
</tr>
<tr>
<td>Inv-eve.</td>
<td>.69</td>
<td>.23</td>
</tr>
<tr>
<td>Tibia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Int-ext. rot.</td>
<td>.80</td>
<td>.09</td>
</tr>
</tbody>
</table>

5.6.3.5 Summary statistics

The group means for the peak values on day 1 and 2 were similar even without the DMS adjustment (Tables 5.5 and 5.6). However, without the DMS adjustment the limits of agreement were very wide (Tables 5.5 and 5.6). This is particularly evident for the movements in the transverse plane, for example, the tibial rotation LOA reduced from...
approximately ±14° to approximately ±1° following the offset adjustment in the walk data (Table 5.5). The LOA give an indication of the absolute within-subject agreement that can be expected between test days. The similar group mean values and wide LOA reported in Tables 5.5 & 5.6 demonstrate that marker placement errors were random rather than systematic. Further the mean bias and 95% CI show that the sum of all the individual differences between test days tended to zero (Tables 5.5 & 5.6) which further illustrates there was no systematic bias between test days.

 Whilst the reliability for the timing of peak knee flexion was good, the LOA values for the timing of peak ankle eversion were poor (Table 5.7). Inspection of the individual ensemble plots showed that the ankle eversion peak occurred over a large plateau where angular velocity is low. Since this peak is less defined, it is likely to have poorer reproducibility; this is in agreement with previous studies (Stergiou and Bates, 1997; Van Woensel and Cavanagh, 1992). Despite this, the variable has been widely used in studies of the function of the lower extremity.

5.6.3.6 Ensemble plots
The group ensemble curves were very similar between test days in terms of magnitude and shape (figure 5.6). The shaded areas of the graphs indicate that the between-subject variability was greater in relative terms (given the magnitude of the movement) for the hip rotation, knee rotation and ankle eversion joint angles. Although not reported, the within subject inter-stride variability was also greater for these curves. This may reflect some inherent biological variability and also a higher signal to noise ratio due to skin movement artefact and camera resolution. Model limitations are also likely to be a factor, for example, cross talk from the flexion to adduction axis (see section 5.2.2).

5.6.3.7 General comments
There was no trend of walking having better reliability than running or vice versa (Tables 5.3-5.7), a finding supported by a previous study of treadmill locomotion (Karamanidis et al., 2003). The reliability was generally worse for joint angles more distal from the hip (Table 5.3 & 5.4). This is possibly due to error propagation caused by small errors in marker placement, which was described in section 5.2.
Figure 5.6. Mean ensemble plots for day 1 (dashed line) and day 2 (solid line) during running, the shaded area represents the mean ± 1 standard deviation for the day 1 data.
Table 5.5. Mean peak values and between day 95% Limits of Agreement (LOA) with and without the offset adjustment (DMS) for the walk data.

<table>
<thead>
<tr>
<th>Peak angle (°)</th>
<th>No DMS</th>
<th>DMS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>µ day 1</td>
<td>sd</td>
</tr>
<tr>
<td>Hip Add.</td>
<td>3.50</td>
<td>1.27</td>
</tr>
<tr>
<td>Int Rot.</td>
<td>-7.79</td>
<td>5.01</td>
</tr>
<tr>
<td>Knee Flex.</td>
<td>26.30</td>
<td>3.95</td>
</tr>
<tr>
<td>Add.</td>
<td>0.18</td>
<td>2.51</td>
</tr>
<tr>
<td>Int Rot.</td>
<td>-8.28</td>
<td>7.80</td>
</tr>
<tr>
<td>Ank Eve.</td>
<td>-11.58</td>
<td>5.19</td>
</tr>
<tr>
<td>Tib Int rot.</td>
<td>10.81</td>
<td>9.84</td>
</tr>
</tbody>
</table>

Table 5.6. Mean peak values and between day 95% Limits of Agreement (LOA) with and without the offset adjustment (DMS) for the run data.

<table>
<thead>
<tr>
<th>Peak Angle (°)</th>
<th>No DMS</th>
<th>DMS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>µ day 1</td>
<td>sd</td>
</tr>
<tr>
<td>Hip Add.</td>
<td>3.97</td>
<td>3.08</td>
</tr>
<tr>
<td>Int Rot.</td>
<td>-8.92</td>
<td>8.18</td>
</tr>
<tr>
<td>Knee Flex.</td>
<td>7.89</td>
<td>6.71</td>
</tr>
<tr>
<td>Add.</td>
<td>-4.71</td>
<td>4.70</td>
</tr>
<tr>
<td>Int Rot.</td>
<td>-8.88</td>
<td>7.57</td>
</tr>
<tr>
<td>Ank Dors</td>
<td>25.87</td>
<td>5.42</td>
</tr>
<tr>
<td>Eve.</td>
<td>-12.52</td>
<td>5.82</td>
</tr>
<tr>
<td>Tib Int rot.</td>
<td>4.77</td>
<td>17.88</td>
</tr>
</tbody>
</table>
Table 5.7. Mean values for the timing of peak variables (% of stance) and 95% Limits of Agreement (LOA).

<table>
<thead>
<tr>
<th></th>
<th>Day 1 mean</th>
<th>Day 1 sd</th>
<th>Day 2 mean</th>
<th>Day 2 sd</th>
<th>Mean bias (CI)</th>
<th>95% LOA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walk</td>
<td>20.90</td>
<td>1.45</td>
<td>20.70</td>
<td>1.32</td>
<td>0.2 (-0.7 – 1.1)</td>
<td>-1.1 – 1.5</td>
</tr>
<tr>
<td>% Knee flex.</td>
<td>36.63</td>
<td>16.00</td>
<td>38.33</td>
<td>12.23</td>
<td>-1.7 (-9.8 – 6.4)</td>
<td>-14.5 – 11.1</td>
</tr>
<tr>
<td>% Ank eve.</td>
<td>29.63</td>
<td>16.90</td>
<td>34.47</td>
<td>19.57</td>
<td>-4.83 (-18.5 – 8.8)</td>
<td>-26.4 – 16.7</td>
</tr>
<tr>
<td>Run</td>
<td>35.43</td>
<td>2.63</td>
<td>36.43</td>
<td>3.51</td>
<td>-1.0 (2.2 – 0.2)</td>
<td>-3.0 – 1.0</td>
</tr>
<tr>
<td>% Knee flex.</td>
<td>29.63</td>
<td>16.90</td>
<td>34.47</td>
<td>19.57</td>
<td>-4.83 (-18.5 – 8.8)</td>
<td>-26.4 – 16.7</td>
</tr>
<tr>
<td>% Ank eve.</td>
<td>29.63</td>
<td>16.90</td>
<td>34.47</td>
<td>19.57</td>
<td>-4.83 (-18.5 – 8.8)</td>
<td>-26.4 – 16.7</td>
</tr>
</tbody>
</table>

5.6.4 Conclusions

Excellent reliability was reported for the sagittal plane movements, good reliability for the frontal plane movements, and acceptable reliability for the transverse plane movements after subtraction of the daily mean offset. Given the improvement in reliability following the DMS adjustment, this approach was adopted for all frontal and transverse plane data in the main study. There is a compromise when using this technique in that the origin of movement that describes the absolute anatomical position is lost. However, without the DMS, there is substantial random error, so there are no advantages to leaving these data without an offset adjustment. Most studies of transverse and frontal plane movements account for constant offset errors, some have subtracted the joint angles from a standing trial (e.g. McClay and Manal, 1997) while others have referenced the angle at initial contact as zero (e.g. Reischl et al., 1999). Knowledge of the variability contained within this model and protocol should enable a more informed interpretation of the results contained in the proceeding chapters.
Chapter 6

A prospective cohort study of gait kinematics and patellofemoral pain syndrome

6.1 Introduction

6.1.1 Background and rationale

Patellofemoral pain syndrome (PFPS) is universally elicited from activities that load the joint and patients frequently report running as the stimulus that precipitates symptoms. The mechanical model for the genesis of PFPS dominates clinical opinion and research in this area. The literature suggests that abnormal kinematic patterns during locomotion may be indicative of injurious loading acting on the body. The question of whether certain kinematic patterns predispose to injury is not a new one. However, at the time of this study there were no published prospective studies relating kinematic patterns during running with AKP or overuse injury.

Most of the case-control research has focused on the relationship between pronation and overuse injury (Callaghan and Baltzopoulos, 1994; Duffey et al., 2000; Hintermann and Nigg, 1998; Messier et al., 1991), and very few studies have correlated tibiofemoral movement with anterior knee pain (Cuddeford and Yack, 2000; Dillon, 1983; Powers et al., 2002). Despite this, the evidence suggests that the kinematic patterns of the segments that articulate with the patellofemoral joint may be better correlated to stress about the knee (Fuch et al., 1999; Huberti and Hayes, 1984; Lee et al., 1994; Mizuno et al., 2001) and as such, could be more sensitive indicators of patellofemoral pain. Since in-vitro studies have demonstrated the effect of tibiofemoral kinematics on patellofemoral kinematics and kinetics, it was considered that an analysis of the lower extremity kinematics alone would provide sufficient evidence of a predisposition for PFPS, and that this could be followed up with a further study on kinetics. Previous studies have shown how hip adduction (Huberti and Hayes, 1984; Mizuno et al., 2001), hip rotation (Lee et al.,
1994; Fuch et al., 1999; Powers, 2003a; Tiberio, 1987), knee flexion (Huberti and Hayes, 1984; Mizuno et al., 2001), knee abduction (Bailey et al., 2003; Huberti and Hayes, 1984; Mizuno et al., 2001), knee internal rotation (Powers, 2003a; Tiberio, 1987), ankle dorsiflexion (Hinterman et al., 1994), rearfoot movement/ foot pronation (Callaghan and Baltzopoulos, 1994; Powers, 2003a; Tiberio, 1987) and tibial rotation (Cudderford and Yack, 2000; Huberti and Hayes, 1984; Lee et al., 2001; Mizuno et al., 2001; Powers et al., 2002) may be associated with either altered kinematics and kinetics of the patellofemoral joint or anterior knee pain. Gait studies of patients with AKP have been confined to a few joint angles. Since the purpose of the phase I work was to generate hypotheses for future research, a less conservative approach was adopted where all the kinematic angles that have been shown to affect the patellofemoral joint were investigated. It was hypothesised that individuals who develop PFPS would display altered gait kinematics to those who remained injury-free.

6.1.2 Aims

The primary aim of this investigation was to:

(i) Determine whether there is an association between lower extremity kinematics during locomotion and the development of patellofemoral pain syndrome.

If an association was found, the secondary aim was to:

(ii) Explore the strength of the association between lower extremity kinematics and PFPS.

6.2 Methods

Ethical approval was obtained from the Defence Medical Services Clinical Research Committee, UK. All participants were verbally briefed about the methods and aims of the study and gave written informed consent.
6.2.1 Design

A prospective cohort study of military recruits enlisted onto the standardised 12 week common recruit syllabus (CMS(R)) military training program was undertaken (details of the program are contained in section 4.1.2 & 4.2.4). All subjects underwent gait analysis on the first day of training (baseline) and were followed up for occurrence of PFPS, lower extremity overuse injury and training outcome over the period of training.

6.2.2 Subject selection

136 male subjects participated in the study. All subjects were screened by a medical officer to ensure they were in good health, free of musculoskeletal injury and had no contraindications to exercise. Subjects with an injury or musculo-skeletal disorder that would affect their ability to undertake military training were excluded from the study. All subjects had prior experience of treadmill running.

6.2.3 Sample size

An ‘a priori’ power calculation was performed to determine the sample size. The type 1 error rate or alpha (α), which is the probability of rejecting the null hypothesis when it is actually true, was set at .05. Power (1-β: where beta is the probability of accepting the null hypothesis when it is actually false, also called the type 2 error rate), which is the ability of a study to detect a difference of a specified size when one actually exists, was set at .8. These levels are deemed sufficient for most medical research (Altman, 1992). The effect size or standardised difference (δ) between groups that the study was powered to detect was set at 1.27. This was based on previous studies into gait and anterior knee pain where the effect size ranged from 0.45 – 2.1 (Duffey et al., 2000; Nadeau et al., 1997). It is recognised that this choice was somewhat arbitrary, and a study can be powered to detect a difference of any size but it is important to separate statistical significance from clinical significance. In this case the choice was compounded by the study having no precedent in terms of design. As a compromise an effect size that lied between those given above (1.27) was used. The requisite sample size was adjusted to account for exclusions and the unequal sample size in each group. Previous research was used to estimate an exclusion rate of approximately 40% (Gemmell, 2002; Jones et al., 1993). To model the worst case scenario, the smallest reported incidence of PFPS in military recruits (5% - Cowan et al., 1996) was
chosen to calculate the ratio of the sample size in each group. Using these criteria, 69 subjects were required for the control group and 6 for the PFPS group (Altman, 1992; Mace, 1974). Accounting for exclusions, the minimum required cohort size was 125.

6.2.4 Kinematic data collection

The kinematic model, marker placement, testing protocol, filtering and normalisation procedures were described in detail in Chapter 5 and so will not be repeated here. The gait test speeds were 1.81m.s⁻¹ for walking and 2.78m.s⁻¹ for running. These speeds replicate those typically used in military training.

6.2.5 Independent variables

In accordance with the purpose of the study and results from previous studies, the following eight joint angles were generated: hip adduction, hip rotation, knee flexion, knee abduction, knee internal rotation, ankle dorsiflexion, foot eversion, and tibial rotation. Angular velocity was also calculated for these variables using the central difference technique where the forward-backward difference method was used to calculate the first and last data points (Robertson and Caldwell, 2004).

The time series joint angle data were parameterised to peak amplitude and excursion. Peak amplitude was also collected for the velocity data. To avoid discarding potentially important time-dependent information, the mean angle and velocity over mid-stance was extracted (figure 6.1). Midstance was defined as 17-50% of stance during walking (Perry, 1992; Whittle, 1996) and 35-45% of stance during running (Ounpuu, 1990). These phases were considered important because they encompass peak knee flexion and the peak patellofemoral joint reaction force (Heino and Powers, 2002; Scott and Winter, 1990). The acronyms for these variables are detailed in Table 6.1. The time to peak variables were not calculated because of questionable reliability. This was discussed in section 5.6.

Temporal-spatial data (cadence, stride length and stance phase proportion) were also calculated since they have been attributed to overuse injury in military training through unnatural stride lengths that a recruit may be forced to adopt in order to synchronise gait during group parade (Hill, 1996).
Figure 6.1. A graphical description of parameters extracted from the joint angle waveforms

Table 6.1. Nomenclature used to describe the kinematic joint angle variables

<table>
<thead>
<tr>
<th>Prefix</th>
<th>Suffix</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>HA-</td>
<td>PA</td>
<td>Peak angle</td>
</tr>
<tr>
<td>HR-</td>
<td>AMS</td>
<td>Angle over midstance (walking=17-50%; running=35-45%)</td>
</tr>
<tr>
<td>KF-</td>
<td>EXC</td>
<td>Angular excursion</td>
</tr>
<tr>
<td>KA-</td>
<td>PV</td>
<td>Peak velocity</td>
</tr>
<tr>
<td>AD-</td>
<td>VMS</td>
<td>Velocity over midstance (walking=17-50%; running=35-45%)</td>
</tr>
<tr>
<td>AE-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TR-</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Examples: HA-PA: Hip adduction peak angle; AD-EXC: Ankle dorsiflexion angular excursion

6.2.6 Case capture

6.2.6.1 Patellofemoral pain syndrome group

Subjects who complained of knee pain were referred to a research clinic. A single physician with an expertise in sports medicine made all diagnoses. The criteria used to diagnose a case of patellofemoral pain syndrome (PFPS) are given in Table 6.2. These criteria have been outlined by Thomee et al. (1999) and are essentially a diagnosis of exclusion. The criteria are more extensive than that used in the epidemiology study (Chapter 4) because the objectives and independent variables of this study are concerned with isolating dynamic mechanical risk factors for patellar pain. A copy of the clinical examination proforma is contained in ANNEX C.
To describe the symptoms of pain and disability in the PFPS cohort, a functional index questionnaire (FIQ) was administered (Kujala et al., 1993). This also included a visual analogue scale to record pain intensity (VAS).

### Table 6.2. Inclusion and exclusion criteria for a diagnosis of PFPS (CH = Clinical history).

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Indicator</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inclusion</td>
<td>✓ Anterior knee pain during or after 2/3 of the following: walking, running, ascending/descending stairs.</td>
</tr>
<tr>
<td>Exclusion</td>
<td>✓ Acute onset</td>
</tr>
<tr>
<td></td>
<td>✓ Signs of meniscal injury or intra articular pathology</td>
</tr>
<tr>
<td></td>
<td>✓ Ligament injury</td>
</tr>
<tr>
<td></td>
<td>✓ Surgery</td>
</tr>
<tr>
<td></td>
<td>✓ Neurological involvement or hip/lumbar referred pain</td>
</tr>
<tr>
<td></td>
<td>✓ PFP secondary to another overuse injury</td>
</tr>
<tr>
<td></td>
<td>✓ Tenderness over the patella tendon, pes anserine tendon or iliotibial band</td>
</tr>
<tr>
<td></td>
<td>✓ Plica syndrome</td>
</tr>
<tr>
<td></td>
<td>✓ Patella subluxation/dislocation</td>
</tr>
<tr>
<td>CH</td>
<td>CH (knock, twist, fall)</td>
</tr>
<tr>
<td>CH</td>
<td>CH, Joint swelling/effusion, McMurray's test.</td>
</tr>
<tr>
<td>CH</td>
<td>CH, Lachman test, Anterior draw</td>
</tr>
<tr>
<td>CH</td>
<td>CH, Straight leg raise</td>
</tr>
<tr>
<td>CH</td>
<td>CH, Palpation and stress tests, localised tendon inflammation.</td>
</tr>
<tr>
<td>CH</td>
<td>CH, Palpation and stress tests</td>
</tr>
<tr>
<td>CH</td>
<td>CH</td>
</tr>
</tbody>
</table>

### 6.2.6.2 Controls

The criteria to classify a control were: (i) Passed 12 weeks of Army training; (ii) No lower limb overuse injury; (iii) No symptoms of AKP for 3 years from the time of baseline data collection.

Criterion 1 was used to control the exposure 'dose time' to training. The second criterion was applied to minimise the possibility of other overuse injuries with similar aetiology masking true risk factors. Data relating to other overuse injuries were obtained by reviewing all medical notes and electronic hospital database records. The 3 year specification stated in criteria (iii) requires particular explanation. As part of a separate validation study, the cohort described in this study was followed up for anterior knee pain occurrence 3 years post enlistment to the Army. This validation study is detailed in Chapter 8, but it is important to mention here that 18 subjects who remained free of AKP during the 12 week Army training program subsequently developed AKP during the
proceeding 3 year follow up. This information was used to correct the control group in this study. One limitation of prospective studies with a short follow-up time is that results may be confounded by individuals originally classified as a ‘control’ subsequently developing the symptoms of interest after the surveillance period, this subsequent correction should minimise the risk of this potential flaw and provide a more robust control group.

6.2.7 Data reduction and statistical analysis

Six strides were processed for each subject. The dominant limb was used for the control group (for 84% this was the right limb) whilst the injured or most painful limb was used for the PFPS group. The mean was obtained from these six strides for each individual and used to calculate a group mean ensemble plot. The ensemble plots for each group were visually compared and used to interpret the results of the main statistical analysis.

The main analysis was conducted in two steps. First, the independent variables were reduced to a smaller set of factors using a factor analytic method. Second, the resulting factor scores were entered into a logistic regression procedure to explore the association between gait kinematics and PFPS. The rationale and procedure is explained below.

6.2.7.1 Principle components analysis

A limitation of describing waveforms based on summary statistics is the generation of a large set of variables, the walk and run dataset each contained 36. This has two key statistical implications; first, the reliability of logistic regression is impaired when there are too few cases in relation to the number of variables. Second, the parameterisation of the waveforms makes it likely that some of the variables are strongly correlated with each other, e.g. one may expect a correlation between peak velocity and angular excursion. If multicollinearity exists then the regression procedure will produce coefficients with poor reliability making it difficult to assess the relative importance of each of the independent variables. To address these limitations and ensure that the analysis considered the entire dataset and thus adhered to the multivariate and hypothesis generating purpose of this part of the PhD, a factor analytic method (principle components analysis (PCA)) was undertaken (SPSS, v10). PCA seeks to find the underlying structure in a dataset and determine subsets of variables that are measuring a similar construct. This is done through an examination of the correlation matrix of observed variables using eigendecomposition.
Ultimately the goal is to reduce a large number of observed variables into a smaller number of independent factors but preserve the maximum amount of variance contained in the original dataset (Tabachnick and Fiddell, 2001). The aims of this PCA were thus twofold, first, to find a more parsimonious representation of the data that reduced the number of variables but retained the maximum variance in the dataset, and second, to remove any collinearity from the dataset.

The principles, mathematics and method of PCA are explained in more detail in ANNEX D, however, for the purpose of continuity the following is a very brief outline of the four main steps. The first step was to remove variables with poor factorability. These were variables with little shared variance or an extreme correlation with another variable(s) (r>.9). The correlation matrix and Keyser Meyer Olkin (KMO) statistics were used to find these variables. The second step was to select the number of factors for extraction. This decision was based on the magnitude of the eigenvalues for each factor. The eigenvalues indicate the amount of variance from the original dataset explained by each factor. Kaisers criterion of selecting factors with eigenvalues in excess of one was used (Field 2005). This was cross referenced with Catell’s (1966, cited by Field 2005) scree plot method of extraction. The underlying variable composition of each factor was also scrutinised to ensure that each extracted factor had some theoretical relevance. The third step was to select a method of factor rotation. Factor rotation is an optimal solution that seeks to maximise the loadings of the important variables on a factor and minimise the loadings of the least important variables. An orthogonal (varimax and quartimax) and oblique rotation (directquartimin) was executed. The correlation between factor scores from the oblique rotation was used to assess whether the oblique rotation improved the fit of the factor model to the data. The final step was to label the factors by examining the rotated factor loading matrix. From the PCA, standardised factor scores were calculated (i.e. the mean equals zero and the standard deviation equals one) and used as the new independent variables. A factor score consists of a linear combination of the original variables where the variables that explain the most variance in a factor (i.e. are most important) have a heavier weighting.

6.2.7.2 Logistic Regression
The standardised factor scores and the variables with little shared variance were used as predictor variables for the regression analysis. A backwards stepwise (likelihood ratio)
logistic regression procedure was performed (SPSS v10). The criterion for removal of a variable was set at .05. The stepwise method was chosen because this was an exploratory analysis.

6.3 Results

The diagram in figure 6.2 describes the outcome of the 136 subjects who participated in the study. Eleven subjects developed PFPS, however, four had PFPS secondary to another overuse injury and were excluded from the analysis. Of the exclusions from the control group, 29 failed to complete training (criteria (i): incomplete training exposure), 17 sustained another overuse injury, 18 reported symptoms of anterior knee pain in the 3 year follow-up study, and 28 had no contact details or did not participate in the follow up study (see figure 8.1 in Chapter 8 for detailed breakdown of the follow up study). Thirty-seven subjects remained and formed the control group (figure 6.2).

There were no significant differences (p>.05) in the descriptive characteristics between groups (Table 6.3). Unfortunately, there were too few subjects to adjust for other possible covariates such as smoking and ankle injury in the main analysis.

6.3.1 Post hoc test of power

The power of the study to detect the ‘a priori’ specified effect size of 1.27 standard deviations between groups of n= 37 (control) and n=7 (PFPS) with alpha at .05 was 86.9%. Alternatively, the study had 80% power to detect differences of 1.18sd (figure 6.2b), (Mace, 1974). Due to the unequal sample sizes, the effect of the reduced control group size on the power of the study was small (figure 6.3 a). In contrast, an increase in the size of the PFPS cohort would have had a disproportionately greater effect on the study power (figure 6.3 a b). Thus despite the control group being smaller than predicted (section 6.2.3), the study still had >80% power to detect a difference of 1.27sd due to the extra subject in the PFPS group.
Chapter 6. Prospective cohort study of gait kinematics

Figure 6.2. Flow diagram describing the outcome of the original 136 cohort. The thick lines represent the PFPS and control group.

Table 6.3. Descriptive characteristics of the control and PFPS group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n=37)</th>
<th>PFPS (N=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>sd</td>
</tr>
<tr>
<td>Age</td>
<td>18.93</td>
<td>1.95</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.78</td>
<td>0.07</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>69.83</td>
<td>9.26</td>
</tr>
<tr>
<td>BMI (kg.m²)</td>
<td>21.88</td>
<td>2.49</td>
</tr>
<tr>
<td>Pre-enlistment running (miles.week⁻¹)</td>
<td>8.5</td>
<td>6.27</td>
</tr>
<tr>
<td>2.4 k run time (s)</td>
<td>605.7</td>
<td>37.11</td>
</tr>
</tbody>
</table>

Figure 6.3. The effect size (e) that the study could detect with at least 80% power (blue area) and a 5% type 1 error rate for predicted sample sizes in the PFPS group where n of controls = 37 (a); and predicted sample sizes in the control group where n of PFPS = 7 (b).
6.3.2 Pain and Symptoms

Six (86%) of the subjects developed PFPS by week 4 of training. The other subject developed pain during week eleven. The entire PFPS group reported pain when running and when either ascending or descending stairs. Three subjects also reported pain during walking. The PFPS group had a median score of 58/100 (100 represents perfect knee function) on the FIQ (IQR: 56-77; range: 54-88) and a median pain score (VAS) of 52 (IQR: 22-60; range: 10-77). Six subjects had unilateral pain (3 right, 3 left) and one subject had bilateral pain where the left limb was more painful than the right.

6.3.3 Temporal-spatial data

The temporal-spatial data were very similar between groups for both the walk and run data (Table 6.4) and as such were not further analysed using regression.

<table>
<thead>
<tr>
<th></th>
<th>Control mean</th>
<th>Control sd</th>
<th>PFPS mean</th>
<th>PFPS sd</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cadence (strides per second)</td>
<td>1.42</td>
<td>0.08</td>
<td>1.42</td>
<td>0.10</td>
<td>.853</td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>1.96</td>
<td>0.12</td>
<td>1.97</td>
<td>0.13</td>
<td>.802</td>
</tr>
<tr>
<td>Stance phase proportion (%)</td>
<td>32.5</td>
<td>3.4</td>
<td>31.8</td>
<td>3.1</td>
<td>.623</td>
</tr>
</tbody>
</table>

6.3.4 Joint angle ensemble plots

There were some general patterns of differences between groups in the walk ensemble plots. The peak and amplitude of hip adduction was greater in the PFPS group. Upon foot contact, the initial internal rotation movement of the hip and tibia was greater in the PFPS group. The knee also displayed less internal rotation excursion from 0-25% of stance. However, these differences were small and within one standard deviation (figure 6.4).

The same trends occurred in the run data ensemble plots. However, the pattern of increased hip internal rotation and greater excursion of tibial rotation from 0-40% of stance was more pronounced in the run data compared to the walk data (figure 6.5). The knee also showed a pattern of prolonged abduction during midstance in the PFPS group. The other mean joint angle plots were similar between groups.
Figure 6.4 Mean ensemble joint angle curves for the PFPS and control group during walking. KEY: Dashed line = control mean; Grey area = control ± 1SD; Red line = PFPS mean.
Figure 6.5 Mean ensemble joint angle curves for the PFPS and control group during running. KEY: Dashed line = control mean; Grey area = control ± 1SD; Red line = PFPS mean.
6.3.5 Principle components analysis

6.3.5.1 Choice of variables for factoring
All variables were normally distributed. The correlation matrix (R-matrix) and Kaiser-Meyer-Olkin (KMO) statistics were inspected to determine any variables with high collinearity or little shared variance. From the original 36 variables in the walk data set, 16 were removed leaving 20 variables for factoring. On similar principles, 10 variables were removed from the run data set, leaving 26 for factoring. Bartlett's test of sphericity was <.001 for both the walk and run data set, and the mean KMO statistic was .62 for the walk and .66 for the run data, thus, the data were deemed adequate for factoring.

6.3.5.2 Factor Extraction
Based on Kaisers criterion, six factors were extracted from the walk dataset, and eight from the run dataset. This very closely met the assumptions for Kaisers criterion which is valid when all communalities (h²) are >.7. All except one of the h² after extraction were >.66. Catell's (1966 cited by Field 2005) criteria for adequacy of factor extraction was also closely satisfied, here the inflexion point on the scree plot is used to select the number of factors (figure 6.6). Most importantly, the resulting factors were well defined by the variables (Tables 6.5 & 6.6).

![Figure 6.6. Scree plots for the walk (A) and run data (B). The arrows correspond to the number of factors that were extracted. Catell's (1966) criteria advocates factor extraction based on the inflexion point on the curve.](image-url)
Less than 26% and 35% of the residuals from the residual R-matrix were >.05 for the walk and run data respectively, indicating that the model based on this number of factors was a good fit.

6.3.5.3 Factor rotation
The orthogonal (Varimax & Quartimax) and oblique (directquartimin) rotations produced similar factors. However, the oblique rotation was chosen since the R-matrix for factor scores showed two pairs of factors that were slightly correlated. For the run data, factors 1 and 7 had an r of .33 and factors 1 and 8 had an r of -.25. This suggests that the oblique factor rotation was a better fit (Field, 2005). Previous studies have shown that some joint movements may be coupled (e.g. McClay and Manal, 1997), so from a theoretical basis some correlation among factors was expected, which reinforces the oblique rotation as the most sensible solution.

6.3.5.4 Factor Loadings and labels
The factor loadings contained in the pattern matrix represents the unique relationship and strength of relationship between each variable and the respective factor, where higher absolute loadings indicate a stronger relationship (Tables 6.5 & 6.6). The structure matrix gives the correlations between each variable and factor and so does not ignore the shared variance. This matrix also highlights the source of any correlations between factors. The pattern matrix was initially used to label the factors. Loadings greater than .63 have been classified as very good (Comrey and Lee, 1992) and were used to interpret factors. Where certain variables are loaded onto more than one factor, which can occur in an oblique rotation, then the pattern matrix which partials out shared variance may hide variables that still contribute some important variance for that factor. The structure matrix was used to find these variables, highlighted blue in Tables 6.5 & 6.6. With the exception of factor 7 (run data), the labels contained some theoretical and clinical relevance (Table 6.5 & 6.6).
Table 6.5. Factor loadings (pattern matrix) and communalities (h²) after factor extraction from the obliquart rotation for the walk data. Variables with loadings >.63 are in red in the pattern matrix and blue in the structure matrix (see section 6.3.5.4) and considered important to that respective factor. Loadings <.4 are suppressed for ease of interpretation.

<table>
<thead>
<tr>
<th>Walk</th>
<th>F1</th>
<th>F2</th>
<th>F3</th>
<th>F4</th>
<th>F5</th>
<th>F6</th>
<th>F1</th>
<th>F2</th>
<th>F3</th>
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Factor Labels
F1 Hip adduction
F2 Shank rotation
F3 Knee flexion
F4 Ankle eversion position
F5 Ankle dorsiflexion
F6 Hip rotation
Table 6.6. Factor loadings (pattern matrix) and communalities ($h^2$) after extraction from the obliquart rotation for the run data. Variables with loadings >.63 are considered important to that respective factor. These variables are in red in the pattern matrix and blue in the structure matrix (see section 6.3.5.4). Loadings <.4 are suppressed for ease of interpretation.

<table>
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<th>Oblique Factors (RUN)</th>
<th>Pattern Matrix</th>
<th>Structure Matrix</th>
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<td>HR-EXC .537</td>
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Factor Labels
F1  Leg rotation
F2  Knee abduction
F3  Hip adduction
F4  Knee flexion
F5  Ankle eversion
F6  Knee rotation
F7  Thigh/shank frontal and transverse plane velocity over midstance
F8  Hip rotation

6.3.6 Logistic regression

6.3.6.1 Walk data
For the walk data, factors 1-6, KF-PA, KR-AMS, AD-PA and AE-VMS were entered into the logistic regression model. The four additional variables were entered because they had
little shared variance and no collinearity with other variables in the dataset. None of the factors or variables significantly improved the fit of the model to predict PFPS above that offered by the constant (-2*log likelihood = 38.558). Factor 1 (hip adduction) was the last factor to be removed from the model (p-value of coefficient = .178).

6.3.6.2 Run data
Factors 1-8, KF-PA, KR-VMS, AD-PA and AE-PV were entered into the logistic regression model. A reduced score on factor 1 (leg rotation) was a significant predictor (p<.05) of PFPS (Table 6.7). This corresponds to increased hip and tibial internal rotation angles and higher tibial internal rotation peak velocity.

Whilst the regression coefficients for factor 3 (hip adduction) and factor 6 (knee rotation) were not significant at the 5% significance level, both factors did make a significant contribution to the explained variance in the predictive model as indicated by the change in the log likelihood (Table 6.7). Here, an increase in hip adduction angle, excursion and velocity increased the odds of developing PFPS, whilst a decrease in the excursion and velocity of knee internal rotation increased the odds of PFPS.

Factor 1 was an important and significant univariate risk factor (p=.036, p<.05). The other two factors from the final model were not significant when analysed alone but their p-values were still quite low at .097 (p >.05) and .082 (p >.05) for factor 3 and 6 respectively.

6.3.6.3 Predictive model
All standardised residuals were below 2.58, which indicate that the model was a good fit for most cases. The influence statistics were satisfactory, all Cook distances and standardised Cook distances (DFBeta) were below 1.0 and the leverage statistics suggested that none of the cases were having a disproportionate influence on the model.

The predictive model was able to separate most cases of PFPS from controls using factor 1 (leg rotation), factor 3 (hip adduction) and factor 6 (knee rotation) (figure 6.7a). The receiver operator characteristic (ROC) curve (figure 6.7b) shows that the model was able to predict PFPS with a sensitivity of 86% and specificity of 78%. Alternatively, by changing the probability classification, 100% sensitivity was possible with 76% specificity (figure
6.7b). The model explained 47% of the variance between a PFPS and control outcome, and was a significant predictor of PFPS ($\chi^2 = 13.98; p<.05$) (Table 6.7).

Table 6.7. Summary of the coefficients, significance tests and odds ratios for the final model produced from logistic regression for the run data. The final column reports the significance of the change in the model if the term is removed (LL = log likelihood).

<table>
<thead>
<tr>
<th>Factors (run)</th>
<th>$\beta$</th>
<th>se</th>
<th>p</th>
<th>Odds ratio</th>
<th>95% CI</th>
<th>sig. of change in LL</th>
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</thead>
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<td>4.487</td>
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<td>23.623</td>
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<td>F6 Knee rotation</td>
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</tr>
<tr>
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<td>.040</td>
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</tr>
</tbody>
</table>

$R^2 = .47$ (Nagelkerke)

Model $\chi^2 = 13.98$ (p=.003)

Hosmer and Lemeshow Test: $\chi^2=3.73$ (p=.881)

Figure 6.7. Frequency histogram of predicted probabilities for PFPS group membership (a); and resulting Receiver operator curve (ROC) showing the effect of different classification cut off probabilities on the sensitivity and specificity of the predictive model (b).
Chapter 6. Prospective cohort study of gait kinematics

6.4 Discussion

This was the first prospective cohort study to examine the association between gait kinematics and PFPS in a controlled training environment. The hypothesis that individuals who develop PFPS would display altered lower extremity kinematics at baseline can be accepted. The prospective design, standardised exposure to activity, homogeneity of age and case capture method make this a strong study from which to examine risk factors for PFPS. The study also had sufficient power to detect the specified 'a priori' effect size. It is clear from figure 6.3(a, b) that the most detrimental effect to the power was the size of the PFPS cohort and a larger control group would have had little effect on the study power (figure 6.3b).

6.4.1 Validity of the control group data

6.4.1.1 Exclusions

A large number were excluded from the control group due to no follow up data at the 3 year point, this could introduce some bias. However, there were no significant differences in age, height, weight, BMI, smoking, aerobic fitness, activity history or previous injuries (p>.05) between the group lost to follow up (n=28) and the control group (n=37) used in this study. In addition, the size of the control group was considerably larger than most previous studies that have quantified 3D gait kinematics.

6.4.1.2 Joint angle comparisons with previous studies

The control group ensemble plots for the sagittal plane joint kinematics were similar to previously presented data from skeletal pin (Lafortune et al., 1992) and skin marker studies (Novacheck, 1998; Ounpuu, 1990).

The amplitude and pattern of hip adduction also agreed with previous research in walking (Cho et al., 2004) and running (Schache et al., 2003). The magnitude of knee abduction was greater than that derived from bone pin studies that used RSA to determine the anatomical axis (La Fortune et al., 1992; McClay et al., 1990 in Ramsay and Wretenberg, 1999; Reinschmidt et al., 1997). The published data from skin based marker sets is highly variable with some showing knee adduction (Cho et al., 2004; Ferber et al., 2003; Ounpuu, 1990) and others knee abduction patterns (Chao et al., 1983). The constraints offered by
the lateral ligaments mean that very little abduction-adduction movement would be expected in a healthy knee joint (Nordin and Frankel, 2001). As discussed in section 5.2, the knee is particularly sensitive to errors from misaligned axis and cross talk because the movement predominantly occurs in one plane. Due to the uncertainty concerning the validity of this angle, the results for knee abduction will not be discussed further.

The ankle eversion plots displayed similar patterns of movement to previous studies but there was some disparity in the amplitude. Comparisons of foot eversion are complicated by the different approaches that have been used. However, studies where the movement was solely based on calcaneal markers, found eversion magnitudes from initial contact ranging from 4-18° (Callaghan and Baltzopoulos, 1994; Lemke et al., 1995; Liu et al., 1997; Soutas and Little, 1987; Scott and Winter, 1991; McClay and Manal, 1997; Duffey et al., 2000). Studies that quantified eversion based on markers placed on the fore and rearfoot found amplitudes of approximately 8° (Powers et al., 2000; Reischl et al., 1999). Goniometric studies of rearfoot movement found approximately 7° of eversion (Taunton et al., 1985). A mean eversion of approximately 4° was found in this study, which is low compared with previous reports. A discussion of the validity of the ankle eversion measurement in the context of the findings from this study is continued in section 6.4.4.

The mean hip rotation plot for walking was similar to previous data (Cho et al., 2004; Kadaba et al., 1989), and the mean running data displayed a similar bimodal pattern to previous studies that have used the HH wand marker set (Novacheck, 1998; Schache et al., 2003). The mean pattern for running also agreed with published data from a 6 DOF model up to 65% stance (Ferber et al., 2003). One author noted that there is a consensus that the hip internally rotates during load acceptance, but disagreement in the motion during the propulsion phase (Schache et al., 1999). However, previous studies have reported variability in the pattern of hip rotation during load acceptance, with some subjects showing internal and others external rotation patterns (Reischl et al., 1999; Powers et al., 2002). This variability was also present in the sample reported here.

The data for knee internal rotation was similar to other skin based kinematic models for walking (Chao et al, 1983; Kadaba et al., 1989) and running (Ferber et al., 2003). Skeletal pin studies have also found bimodal internal rotation patterns with modes at 0-25% and 70-100% of the stance phase of walking (Lafortune et al., 1994; Ramsey and Wretenberg,
1999), however, the 2nd mode was larger in these studies. The amplitude of knee internal rotation during initial stance of running agreed with skeletal pin studies (Reinschmidt et al., 1997).

The pattern of tibial rotation during walking (Lafortune et al., 1994) and running (Novacheck, 1998) agreed with previous studies. The data from walking showed approximately 3° less initial internal rotation than the skeletal pin study (Lafortune et al., 1994). These differences could be due to the different reference systems used (global vs foot), differences in walking speed or differences in the mode of ambulation since studies have generally shown good agreement if slightly overestimated tibial rotation from skin versus skeletal markers (Reinschmidt et al., 1997).

6.4.2 PCA

The statistical analysis of gait data has historically been problematic. This is due to its high dimensionality and the loss of information that occurs through the data reduction methods used to make the dataset more appropriate for conventional statistical analysis. The PCA adopted here was a compromise against these limitations. It fulfilled the statistical aims of removing redundancy and collinearity, and determining the underlying structure of the dataset. This ensured that the main exploratory analysis into the nature and strength of association of risk factors for PFPS was not restricted to a small number of joint angles and parameters.

The factor loadings for each variable were generally well partitioned on each component and the derived factor labels were theoretically explicable. This encouraging outcome minimises the potential for inherent subjective error that can occur from factor extraction when the variables share their variance on too many factors. It also provides a useful indication that the data were appropriate for factoring (Field, 2000).

Whilst the use of multivariate techniques and more complex analytical methods has expanded recently in gait analysis (Chau, 2001), there are still no published data to compare these factors with. However, it is interesting to note one previous study that did a PCA over the entire stance phase waveform of eight separate joint angles, reaction forces and moments. Here, each waveform was subsequently reduced to 2-4 components (Deluzio
et al., 1997). This approach has also been applied to EMG data (e.g. Davis and Vaughan, 1993). Future studies with sufficient power may wish to consider this approach to examine risk factors for injury.

One criticism of PCA is the assumption of a linear relationship between variables. However, the scatter plots among variables did not reveal any relationships that deviated from linear and without empirical evidence, the modelling of other relationships (e.g. quadratic) could result in an interminable analysis of the data structure and functional inter-relationships.

6.4.3 Risk factors

6.4.3.1 Walking

There were no significant factors in the walk data. That running showed greater discrepancy between groups for certain joint angles conforms to earlier work that showed larger differences at faster walking speeds (Heino and Powers, 2002). It is possibly also reflected in the clinical history of the PFPS group where all subjects reported pain from running but only three from walking. The majority of studies have only examined the walking gait of PFPS subjects (Heino and Powers, 2002; Callaghan and Baltzopoulos, 1994; Dillon et al., 1983; Nadeau et al., 1997; Powers et al., 1997b; Powers et al., 2002). Future studies on similar young populations with AKP may wish to examine running since this mode of ambulation may be more representative of the activity that caused symptoms and more sensitive in detecting differences.

6.4.3.2 Running

Factor 1 (leg rotation), a composite of hip and tibial transverse plane rotation, was a significant risk factor for PFPS. Some recently reported preliminary prospective data from 9 females with patellofemoral pain also support this finding (McClay Davis et al., 2004). McClay Davis et al. (2004) found the hip to remain in a more internally rotated position during stance in the group with pain. Tibial rotation was not reported. The direction of the association (internal rotation) for this factor also directly contradicts case-control studies that have measured these variables in patients with PFPS (Cuddeford and Yack, 2000; Dillon et al., 1983; Powers et al., 2002). It seems logical to suggest that the opposing findings in case-control studies may be a compensatory mechanism in persons with pain.
Certainly studies have shown other compensations due to pain, such as reduced knee flexion angle (Nadeau et al., 1997) and knee extensor moment (Powers et al., 2002).

Factor 3 (hip adduction) was an important predictor of PFPS. McClay Davis et al. (2004) also reported increased hip adduction in their prospective data. There is also tenuous agreement with studies that have found more valgus postures of the lower limb in the frontal plane during jump landing (Smith et al., 1991) and cycling (Bailey et al., 2003).

It is interesting that lower scores on factor 6 (knee rotation) increased the odds of PFPS. This indicates that the tibia and femur moved in a more inter-dependent manner in the transverse plane during stance in those that developed pain. This agrees with the hypothesis for AKP proposed by Tiberio (1987) (see section 3.3.1), although was not present in the preliminary data presented by McClay Davis et al. (2004).

Whilst one should be cautious about extrapolating these results to patellofemoral kinematics and kinetics, it is interesting to note some general findings from in-vitro studies. These studies have demonstrated internal femoral rotation to decrease the patella contact area (Fuch et al., 1999) and increase the contact pressure at 30-60° of knee flexion (Lee et al., 1994). Similar increased loading patterns have been reported from induced internal tibial rotation (Csintalan et al., 2002) and hip adduction (Huberti and Hayes, 1984). In further support of a mechanical mechanism for pain, Heino and Powers (2002) found increased patellofemoral stress during walking in a group of females with PFPS. This study was based on a two dimensional kinematic model, to date, there are no in-vivo studies that have examined the effect of transverse and frontal plane hip, knee and ankle joint motion on patellofemoral contact mechanics.

Although the cause of these significant gait factors cannot be determined from this study, it is interesting to speculate because it has implications for the management of PFPS. It is commonly assumed that internal tibial rotation is caused by the ground reaction force and foot eversion that occurs during stance, hence the use of foot orthoses to control foot and knee kinematics (e.g. Mundermann et al., 2003). However, the relationship between eversion and tibial rotation has been shown to be highly variable between subjects in both in-vivo (Nester, 2000; Reischl et al., 1999; Stacoff et al., 2000b) and in-vitro studies (Hintermann et al., 1995; Sommer et al., 1996). Further, a recent study found that the
forces at the proximal end of the tibia contributed more to internal tibial rotation than the forces acting at the distal end of the segment (Bellchamber and Bogert, 2000). These findings suggest that the transverse motions at the proximal joints are not predominantly dictated by ground reaction forces and distal segments. There is also some evidence that patients with PFPS have reduced hip abduction and hip external rotation strength (Ireland et al., 2003), however, there are no data relating these strength deficits to the kinematic alterations seen in the PFPS group of this study. It would be interesting to follow up this work and examine the relationship between muscle strength and gait, and the contribution of other variables to the significant findings from this study.

It is important to consider whether any of the gait differences between groups could be attributed to random sampling variation. One possibility could be a bias of subjects with a particular subject-specific response to barefoot locomotion in the PFPS group. Accordingly, the foot-fall contact patterns of all subjects were checked. Whilst it is acknowledged that the determination of foot contact patterns using kinematic data is not the gold standard, the proportion of midfoot contact strikers was similar in each group (control: 30%; PFPS: 29%). This was also supported by similar mean ensemble dorsiflexion angles at foot contact in each group (figure 6.5).

6.4.3.3 Strength of association and predictive model

It is difficult to assess the magnitude of the effect of each of these factors because the odds ratios are based on the standardised factor scores. Essentially, they represent the change in the odds of developing symptoms per one standard deviation change in the factor score. Nonetheless, the model suggests a fourfold increase in the odds of PFPS from a 1sd decrease in factor 1 (greater leg internal rotation) or 1sd increase in factor 3 (greater hip adduction), and a six-fold increase in the odds of PFPS for a 1sd decrease in factor 6 (less knee internal rotation). However, the confidence intervals for the odds ratios were wide, and given the sample size it is beyond the scope of this study to infer that one factor is more critical than another.

Despite the small sample size, the fit of the prediction model and the stability and influence statistics were excellent. The model was able to demarcate between a PFPS and control outcome with impressive overall prediction accuracy. As a comparison, two studies that attempted to predict AKP from patellofemoral alignment variables obtained from CT and
x-rays, found similar predictive efficacy (Pookarnjamorakot et al., 1998; Schutzer et al., 1986), and these studies were possibly more prone to selection bias influencing the type of cases within the AKP group (see section 3.1.4.4). However, it is also important to reiterate that the predictive model reported in this study was generated from a control group that excluded other overuse injuries, thus the model is only applicable to discriminating between PFPS subjects and those who remained free of other lower limb overuse injury. These other lower limb overuse injuries were excluded to avoid masking risk factors for AKP by including conditions such as tibial stress syndrome which may share similar aetiology. This concurred with the hypothesis generating purpose of the study and has been a common approach of many case-control studies. Despite this, it is not known what effect including other overuse injuries in the control group would have had on the predictive model and future work should attempt to establish this.

Whilst there was a reasonable spread in the predicted probabilities for PFPS group membership (figure 6.7a), it is likely that the model is over fitted to these data because of the small sample size. There were also two PFPS subjects that had predicted probabilities for the PFPS group below .25. It is likely that for these individuals there were factors other than gait kinematics that were more important in the aetiology of their pain. Future work should validate this model in other samples of anterior knee pain, for example, in different age and gender groups. The risk of injury could also be time-dependent. For example, the aetiology may be dominated by intrinsic factors such as gait in individuals who develop pain earlier, whereas, in individuals where the symptom onset is later, external factors may predominate. The small sample size prevented this type of time-based statistical modelling (for an example see Knapik et al., 2001).

There was still more than 50% of unexplained variance in the predictive model, clearly there are other salient factors for PFPS that were not measured in this study and it would be interesting to quantify these in conjunction with gait kinematics to see how they affect the explained variance. If PFPS is predominantly a mechanical phenomenon then including a measure of patellofemoral alignment or tracking could give a good indication of patellar contact mechanics (Fuchs et al., 1999; Huberti and Hayes, 1984; Lee et al., 1994; Mizuno et al., 2001) and risk of PFPS. Similarly one should also consider extrinsic covariates such as the exposure to activity and smoking. This type of study would require a substantially larger sample size.
6.4.4 Null findings

The absence of an association between PFPS and the sagittal plane factors may be a reflection of the sensitivity of these movements to disrupt the patellofemoral mechanism.

There was no relationship between factor 5 (ankle eversion (run)) and PFPS. With respect to the magnitude of eversion, this result agrees with all previous case-control studies (Callaghan and Baltzopoulos, 1994; Duffey et al., 2000; Messier et al., 1991; Powers et al., 2002) and the preliminary prospective data presented by McClay Davis et al. (2004). If it is the tibiofemoral movement that is most critical in PFPS, then these null findings may be a reflection of the high inter-subject variability in the movement transfer coefficient between the calcaneus and tibia (see section 3.3.1). One previous investigation did find a delayed timing in peak ankle eversion (Callaghan and Baltzopoulos, 1994) while another reported less eversion during the first 10% of stance (Duffey et al., 2000). It is possible that the results from these two studies were reflecting compensatory gait due to pain since eversion is considered to be a loading response to foot contact (Messier et al., 1991; Powers et al., 1999). Nonetheless, it is important to interpret the benign finding for eversion in this study with caution due to the limitations of the foot model used. Motion at the ankle is a complex movement resulting from rotation and translation of the talocrural and talocalcaneal (subtalar) joints (Liu et al., 1997). In this study the ankle was essentially modelled as a universal 3DOF ball and socket joint. Whilst it has been argued that this model reflects foot eversion as it is assessed clinically (Powers et al., 2002), the axis definition make it a poor representation of the functional anatomy of the foot (Lundberg et al., 1989). Numerous other methods have been used to calculate this angle, most can be traced chronologically. For example, studies have quantified the 2D projection of the calcaneal and tibia (Nigg and Morlock, 1987), modelled the foot as 2 monocentric hinge joints where one axis corresponds to the talocrural and another to the talocalcaneal joint (Scott and Winter, 1991), and quantified the hindfoot movement with respect to the shank in 3DOF (Soutas and Little, 1987) and 6DOF (Liu et al., 1997). The more complex and anatomically correct approaches were not feasible for this study given the objective to also explore other movements about the knee and hip, and the requirement for a large sample size. Nonetheless, there is a need for research that examines the association of foot eversion with AKP using more biomechanically correct models.
6.4.5 Limitations

The small sample size for the PFPS group should be taken into account when evaluating how representative these findings are. Importantly, given the poorly understood aetiology and varied criteria used for clinical and research purposes, the results should not be extended beyond the case group studied here. Further, the applications of these results only lend themselves to the population studied in terms of age and gender. A related concern is the problem of statistical over fitting due to the small sample size. Future work should thus examine these risk factors in larger populations.

The results for transverse plane hip and knee rotation should be interpreted with some caution due to the soft tissue artefact (STA) associated with these movements. Segmental error analysis has confirmed that the thigh is the main source of STA (Reinschmidt et al., 1997b). This is mainly due to muscle activation rather than skin movement, hence the benefit of markers placed away from the belly of the muscle. Unfortunately, these errors are predominantly random and share similar frequencies to the underlying bone so methods to remove them are impractical for a large clinical investigation (Leardini et al., 2004). As some consolation, the errors tend to be greatest during terminal stance and the swing phase of gait (Leardini et al., 2005), periods that were not parameterised in this study.

An additional problem with verifying the validity of the hip rotation result is that the only skeletal pin study to quantify this movement referenced the femur to the global coordinate system (Levens et al., 1948, cited by Ramsay and Wretenberg, 1999). It is only with recent advances in technology that the 3D movement of the reference segment for hip rotation (the pelvis) has been tracked and understood (Schache et al., 1999). More research is required to investigate the effect of different models and reference systems on the calculated hip rotation joint angle. This may aid our understanding of normal and pathological patterns of hip rotation.

The subtraction of the mean from each subject’s joint angle data (DMS adjustment) results in a loss of the potentially important origin of motion. Future research should attempt to address this issue, particularly with regard to the transverse plane rotations of the lower extremity.
Several strategies were adopted to minimise error, these were; the use of multiple cameras to ensure minimal missing raw data, the use of filtering to remove signal noise; the use of markers and marker placement less prone to vibration and STA, and the use of a barefoot protocol to estimate foot movement. Similarly, attempts to optimise reliability and minimise unwanted sources of between subject variance were made by; rigorous and standardised placement of markers, the use of a treadmill to standardise gait speed and a barefoot protocol to prevent random footwear adaptations; the use of subjects with experience of treadmill gait and a period of familiarisation to minimise learning effects and differences with overground locomotion. Ultimately, if the protocol is deemed reliable (see section 5.6) and valid, then other random errors and sources of between subject variance will reduce the sensitivity of the study to detect differences. Since some significant differences were found and acknowledging the possibility of type 1 statistical errors, it seems reasonable to conclude that the procedures adopted for this study were adequate for the joint angles where differences were found. Accordingly, one should be cautious about dismissing a possible relationship between variables such as knee abduction and ankle eversion given the validity of the method used to quantify these variables.

6.4.6 Conclusions

Based on the methods employed and the cohort studied, gait kinematics would appear to explain a significant amount of the variance in PFPS outcome. Internal rotation of the lower limb and adduction of the hip were important predictors of PFPS and it is suggested that these may be risk factors for some types of anterior knee pain. These findings could have applications for the treatment and prevention of PFPS. For example, research may wish to examine the effect of interventions such as orthoses, exercise and muscle training on these factors. Currently, some ongoing innovative research is also being undertaken into the possibility of gait retraining using online feedback, this could also have applications (Davis, 2005).

Finally, this was an explorative study to generate hypothesis for future work. Further work should thus attempt to validate these findings in other populations with AKP. Other studies may also wish to examine the effect of internal limb rotation on patellar biomechanics in-vivo using techniques such as MRI. Should such studies validate these results, this could lead to suitable intervention studies designed to prevent or treat anterior knee pain.
Chapter 7

Variability of joint coordination and PFPS

7.1 Introduction

7.1.1 Variability as a risk factor

Section 3.3.6 outlined some recent biomechanical work that used an approach developed from dynamical systems theory where movement variability is considered to have a functional role in maintaining a healthy musculo-skeletal system. From a biomechanical perspective, inter-stride coordination variability could offer protection from overuse injury through a number of mechanisms, for example, by distributing forces among broader areas of the same tissue, by loading the same tissue location but at different times, or by distributing cyclic forces to different tissues (James et al., 2000).

Previous studies showed that individuals with patellofemoral pain had reduced inter-stride joint coordination variability compared with healthy individuals (Hamill et al., 1999; Heiderscheit et al., 2002). Two alternative explanations were given for these findings. That they either reflect a protective mechanism where more constrained movement patterns are adopted to minimise pain or a risk factor where less flexible movement patterns localise loading on smaller tissue areas and cause injury. A prospective study is required to determine whether decreased variability is a cause or effect of PFPS.

This chapter reports the results from the analysis of the prospective cohort data presented in chapter 6 to examine the effect of variability on the development of PFPS. Given the findings from the previous case-control studies by Hamill et al. (1999) and Heiderscheit et al. (2002), it was hypothesised that individuals who developed PFPS will show reduced inter-joint coordination variability during gait.
7.1.2 Methods to quantify variability

Two techniques that provide a continuous measure of joint coordination variability have been described; these are the vector coding method (VC) (Heiderscheit et al., 2002) and the continuous relative phase (CRP) (Hamill et al., 1999). The VC method, which is calculated from angle-angle diagrams, provides information on the displacement ratio of two moving segments (see ANNEX E for a description) and has been criticised for not containing temporal information (Wheat and Glazier, 2006). The CRP approach has been more widely used and is derived from angular position and velocity data, thereby containing higher dimensional information on the movement state of a segment. Additionally, this approach has more parity with the biological system because the proprioceptive organs are sensitive to velocity as well as position (Burgess-Limerick et al., 1993). It is thus considered a suitable variable to represent the organisation of the neuromuscular system and compared to other approaches, has a stronger theoretical basis for quantifying movement variability (Wheat and Glazier, 2006).

7.1.3 Continuous relative phase

The rationale behind CRP was explained in sections 3.3.5 – 3.3.6, however, a number of different methods have been used to calculate this variable (Hamill et al., 2000; Kurz and Stergiou, 2002, Li et al., 1999). The methods differ in how the data are normalised and how the phase angle is calculated.

7.1.3.1 Normalisation of the phase plot

A normalisation procedure is typically applied to the angular displacement and velocity data so that they assume values of ±1. The purpose of normalisation is to prevent angular velocity dominating the calculation of the phase angle, but to retain the dynamic characteristics of the coordination of the segment. A few approaches to normalisation have been described (Hamill et al., 2000; Kurz and Stergiou, 2002), and recently there has been some debate concerning the validity and requirement for normalisation (Kurz and Stergiou, 2002). Kurz and Stergiou (2002) analysed the sagittal plane motion of the thigh and shank using 2 methods of normalisation and no normalisation. The normalisation routine that set the position data to a unit circle altered the CRP plots to an extent that it would change the interpretation of the coordination of the two segments. The authors argued that normalisation is actually not required because the arctangent function used to calculate the
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phase angle normalises the data. However, it is not known how normalisation affects the variability statistic and it is also not clear whether normalisation will affect the CRP for frontal and transverse plane movements. Hamill et al., (2000) also discussed whether the data should be normalised to the maximum of all trials or the maximum of each individual trial. This in particular may have an effect on the inter-stride coordination variability and requires consideration.

7.1.3.2 Calculation of the phase angle

Once a phase plot is generated it is necessary to extract the phase angle. Two different methods of calculation have been reported (Kurz and Stergiou, 2002), one results in a phase angle range of -90 to 90° and the other a range of 0 to 180°. The effect of these two methods on CRP variability is unknown.

The application of CRP to quantify variability in biomechanics is relatively new and so the nuances of its calculation and interpretation are still a little unclear. Before using the CRP then, it is important to gain an appreciation of how the different normalisation approaches and phase angle calculations affect the CRP variability variable so that a rationale for the choice of calculation is established. This is considered in section 7.2 below and should aid the interpretation of the results from the analysis on the PFPS prospective data.

7.1.4 Aims

The primary aim of this analysis was to:

(i) Determine whether there is an association between inter-stride joint coordination variability and the development of patellofemoral pain syndrome.

If an association was found, the secondary aim was to:

(ii) Explore the strength of the association between inter-stride joint coordination variability and PFPS.
7.2 Choice of CRP calculation method

7.2.1 Purpose

The purpose of this small analysis was to examine the effect of different methods of normalisation and different representations of the phase angle on the CRP variability calculations and gain an appreciation of the information contained in the CRP.

7.2.2 Method

Data were obtained from one randomly selected subject (age: 18 years; height: 1.84m; weight: 85kg) from the prospective cohort study in chapter 6. Six strides were used to calculate CRP variability.

7.2.2.1 Normalisation methods

Of the two normalisation methods presented by Kurz and Stergiou (2002), the method that normalises to the absolute maximum of displacement and velocity was used. This was chosen because the purpose of normalisation is to produce a scalar multiple of the original phase plot and maintain the dynamic qualities of the segment (Hamill et al., 1999; Li et al., 1999). The alternative method, which normalises to the range of the angular displacement data rather than the maximum, skews the displacement data to the boundaries of the phase plot. This distorts the dynamic aspects of the phase plots (Kurz and Stergiou, 2002).

The phase plots of angular displacement and velocity were normalised using the two methods detailed by Hamill et al. (2000). Method 1 normalises to the absolute maximum of all six strides. Method 2 normalises displacement and velocity to the absolute maximum of each individual trial (Table 7.1). Phase plots were also calculated without normalisation (Table 7.1).

Table 7.1. Methods used to normalise the angular displacement (\(\theta\)) and velocity data (\(\omega\)), where \(i\) represents each data point during stance and \(j\) represents each of six trials. Method 1 normalises to the maximum of all six trials and method 2 normalises to the maximum of each individual trial.

<table>
<thead>
<tr>
<th>Angular displacement</th>
<th>Angular velocity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Method 1</td>
<td></td>
</tr>
<tr>
<td>(\theta_i / \text{Max}({</td>
<td>\theta_{ij}}</td>
</tr>
<tr>
<td>Method 2</td>
<td></td>
</tr>
<tr>
<td>(\theta_i / \text{Max}({</td>
<td>\theta_i</td>
</tr>
<tr>
<td>No normalisation</td>
<td></td>
</tr>
<tr>
<td>(\theta_i)</td>
<td>(\omega_i)</td>
</tr>
</tbody>
</table>
7.2.2.2 Phase angle

The phase angle ($\phi$) for each segment was calculated from the phase plot using two methods. These were the reference phase angle and standard phase angle as described in figure 7.1 (Hamill et al., 1999; Kurtz and Stergiou, 2002).

![Phase angle calculation methods](image)

Figure 7.1. Phase angle calculation methods ($\phi = \text{phase angle}; \theta = \text{angular displacement}; \omega = \text{angular velocity}$), (a) Reference phase angle – this has a range of $-90^\circ$ to $+90^\circ$, where a positive $\phi$ represents positive angular velocities and negative $\phi$ represents negative velocities. (b) The standard phase angle has a range of 0-180° and doesn’t differentiate between the polarity of angular velocity.

7.2.2.3 Continuous relative phase

The continuous relative phase was calculated by subtracting the phase angle of one segment from another (e.g.; CRP _hiprot - tibrot_ = $\phi_{\text{hiprot}} - \phi_{\text{tibrot}}$). The following CRPs were calculated (justification provided in section 7.3.2): hip adduction - tibial rotation; hip rotation - tibial rotation; knee flexion - hip rotation; knee flexion - tibial rotation; knee adduction - ankle eversion, tibial rotation - ankle eversion.

CRP variability was calculated using the standard deviation (sd) at each time point over the stance phase of gait. Phase plots, phase angle plots, CRP plots and CRP variability plots were visually compared between the normalisation and phase angle calculation procedures. To assess the similarity between the CRP variability waveforms, coefficients of multiple correlations (CMC) were calculated between the two normalisation methods and between
the two methods of phase angle calculation, details of the CMC calculation were provided in section 5.6.2.1.

7.2.3 Results and Discussion

7.2.3.1 The effect of no normalisation

With no normalisation, the large velocity values caused abrupt shifts in the wave form when the phase plot crossed into a new quadrant (figure 7.2 a, b). At these points there was large inter-stride variability (figure 7.2c) compared to the normalised data (figure 7.4). During periods when the phase plots were in the same quadrant, the variability was low. This was also caused by the velocity data dominating the phase plots and is misleading. The results are in contrast to those reported by Kurz and Stergiou (2002). However, these authors analysed only sagittal plane movements of the thigh and shank, these segments go through a greater displacement and as such the velocity data may not dominate the phase angle to the same extent as joint angles that have smaller excursions. In fact, Heiderscheit et al. (2000) note that coordinates on the phase plot close to the origin will show more CRP variability than points further away even though they are the same distance apart and have the same absolute variability. Hence without normalisation two subjects with similar variability but different joint angles and velocities would have different computed CRP variability scores. A normalisation procedure is thus recommended.

7.2.3.2 The effect of normalisation method

Figure 7.3 shows some exemplar phase plots for data that have been normalised using methods 1 and 2. As expected, method 2 merged the phase plots at the end of the axes that corresponds to the absolute maximums for displacement and velocity. However, the normalisation method had little effect on the variability plot (figure 7.4 a, b) and the mean CMC between the two normalisation methods for all joint couples (using the reference phase angle) was .997 (sd: 0.002). Despite this, method 2, where data are normalised to each individual trial maximum, is recommended. This method removes the possibility of an outlying trial skewing the phase plots as could occur using method 1, and avoids the need to set criteria for removing outliers, which would ultimately be arbitrary (Hamill et al., 2000).
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Figure 7.2. No normalisation applied. (a) exemplar phase plot for tibial rotation (each line represents 1 trial (stance)); (b) phase angle plot of tibial rotation (each line represents 1 trial (stance)) and (c) CRP variability plot of hip adduction – tibial rotation. The phase angle and CRP variability plot use the reference phase angle.

Figure 7.3. Exemplar phase plots of 6 trials for tibial rotation, using normalisation method 1 (a) and normalisation method 2 (b).
7.2.3.3 The effect of phase angle calculation method

The method of phase angle calculation had deleterious effects on the CRP waveforms (figure 7.5 a, b). Clearly, the method of phase angle calculation affects the interpretation of whether a joint couple is considered in phase or out of phase. This is a fundamental issue that needs resolving if it is used as a tool to analyse coordination.

Whilst the phase angle calculation had an effect on the CRP variability plot (figure 7.6 a, b), the patterns were similar. This was reflected in the mean CMC for all joint couples using normalisation method 2 of .96 (sd: 0.02). Thus, the choice of whether to use the reference or standard phase angle does not appear critical for this analysis. However, the reference phase angle is recommended because it differentiates between velocity polarity thereby conforming more closely with the purpose of the CRP to represent the coordinative state of a segment.

7.2.4 Approach adopted

Based on the points raised from this small analysis, method 2 was selected to normalise the data, and the reference phase angle was chosen to calculate the phase angle.
7.3 Method

The walk and run kinematic data from the control and PFPS group of the prospective cohort study detailed in Chapter 6 were reanalysed. Information concerning the data collection protocols can be found in Chapters 5 and 6 and so will not be duplicated here.
7.3.1 Calculation of variability

CRP variability was calculated over the stance phase of gait using the calculation methods specified in section 7.2. To compare results with a previous study and cross reference findings from the CRP analysis, variability was also quantified for the run data using the vector coding method (Heiderscheit et al., 2002). The calculations for this method are contained in ANNEX E.

7.3.2 Independent variables

Variability was calculated for six joint couples, these are described in Table 7.2. These joint relationships have been studied in previous work into variability and PFPS (Hamill et al., 1999; Heiderscheit et al., 1999, 2002). Whilst these authors did not provide a rationale for the selection of each particular joint couple, all relate to the inter-joint relationships about the knee and it was thought that any dysfunction in these relationships may affect the knee extensor mechanism.

The variability plots over the stance phase of gait were parameterised to 3 variables for statistical analysis. The mean over the entire stance (Hamill et al., 1999) and the mean over midstance (Heiderscheit et al., 1999) were calculated. Additionally, the mean from 5-15% of stance and 10-20% of stance was calculated for the walk and run data respectively. These variables correspond to the period when the patella makes contact with the femur, which occurs at approximately 20° of knee flexion (Hungerford and Barry, 1979). This period was considered important because the patella has less stability when not congruently engaged with the trochlea, and it was thought altered variability of tibiofemoral movement here could affect the patellofemoral contact patterns. Further, a previous study also found a significant difference in variability between an injured and healthy group during early stance (Heidersheit et al., 2002).

7.3.3 Statistical analysis

Group ensemble plots for the PFPS group (n=7) and control group (n=37) were calculated over the stance phase of gait. These were used for a visual comparison and to aid the interpretation of the main analysis described below.
Similar to that described in section 6.2.7, the analysis was conducted in two steps. First a principle components analysis (PCA) was performed to reduce the number of variables and remove collinearity from the dataset. An overview of the fundamentals of PCA is contained in ANNEX D. Second, a backwards stepwise (likelihood ratio) logistic regression was performed on the resulting factor scores to explore the association between movement variability and PFPS. The removal criterion for a variable was set at .05 (change in log likelihood).

Table 7.2. Nomenclature used to describe variables/parameters extracted from CRP waveforms

<table>
<thead>
<tr>
<th>Prefix</th>
<th>Joint couple</th>
<th>Suffix Parameter</th>
<th>Parameter</th>
</tr>
</thead>
<tbody>
<tr>
<td>HATR-</td>
<td>Hip adduction – tibial rotation</td>
<td>-MEAN</td>
<td>Mean CRP over entire stance</td>
</tr>
<tr>
<td>HRTR-</td>
<td>Hip rotation – tibial rotation</td>
<td>-MS</td>
<td>Mean CRP over midstance</td>
</tr>
<tr>
<td>KFHR-</td>
<td>Knee flexion – hip rotation</td>
<td>-5</td>
<td>Mean CRP from 5-15% of stance (walk data only)</td>
</tr>
<tr>
<td>KFTR-</td>
<td>Knee flexion – tibial rotation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>KAAE-</td>
<td>Knee adduction – ankle eversion</td>
<td>-10</td>
<td>Mean CRP from 10-20% of stance (run data only)</td>
</tr>
<tr>
<td>TRAE-</td>
<td>Tibial rotation – ankle eversion</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Examples: HATR-MEAN = mean joint coordination variability over entire stance; HRTR-10 = mean joint coordination variability from 10-20% of stance

7.4 Results

7.4.1 Description of CRP variability ensemble plots
The walk data showed a relatively consistent magnitude of variability over the entire stance phase (figure 7.7). In contrast, with the exception of the joint couples involving ankle eversion, the run data showed slightly increased variability during initial stance (figure 7.8). For both the walk and run data the amount of variability was highest in the hip rotation – tibial rotation ensemble plot.

The variability was similar between the PFPS and control group over the stance phase of walking (figure 7.7). However, during running, the joint couples that contained tibial rotation tended to show reduced variability in the PFPS group shortly after initial stance (figure 7.8).
Figure 7.7. CRP inter-stride variability plots over the stance phase of walking for the six joint movement couples.

**KEY:**
- CONTROL mean
- CONTROL ± 1SD
- PFPS mean
- PFPS ± 1SD
Figure 7.8. CRP inter-stride variability plots over the stance phase of running for the six joint movement couples.

**KEY:**
- CONTROL mean
- CONTROL ± 1SD
- PFPS mean
- PFPS ± 1SD
Figure 7.9. Group ensemble vector coding variability plots over the stance phase of running for the six joint movement couples. These data were not normally distributed so the group median and interquartile range are given. $r$ represents the directional concentration and hence variability of the data.

**KEY:**
- CONTROL median
- CONTROL $\pm$ IQR
- PFPS median
- PFPS $\pm$ IQR
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7.4.2 Principle components analysis

From the original 18 variables in the walk data set, two were removed from the PCA due to high multicollinearity (KFHR-5, KFHR-MS). One variable was removed from the run data due to having little shared variance (KAAE-MS). Bartlett's test of sphericity was < .001 for both the walk and run data, and the mean KMO statistic was .64 for the walk and .62 for the run data, indicating the data were adequate for factoring. Based on Kaisers criterion, four factors were extracted for the walk data and 5 factors for the run data. The communalities ($h^2$) were all close to or greater than .7 (Table 7.3 & 7.4). This also corresponded well with the scree plot method of factor extraction (Catell, 1966) (figure 7.10). The five factors for the walk data explained 83% of the variance in the original dataset, and the four factors extracted from the run data explained 72% of the total variance in the run dataset. Thus the majority of the original variance was preserved in the extracted factors. Less than 37% and 44% of the residuals from the residual correlation matrix were > .05 for the walk and run data respectively.

![Figure 7.10. Scree plots for the walk (A) and run variability data (B). The arrows correspond to the number of factors that were extracted. Catell's (1966) criteria suggests factor extraction based on the inflexion point of the curve.](image)

The oblique rotation did not produce factors that were correlated with each other for either the walk or run data, therefore the orthogonal rotation was deemed suitable for these data. Both models were a good fit based on the residual correlation matrix.
Variables were deemed important to a factor if the factor loading was greater than or equal to .63 (Comrey and Lee, 1992). The variables were generally well partitioned on each factor and maintained some structural and theoretical relevance (Tables 7.3 & 7.4). The joint couples that contained tibial rotation over the early period of stance (5-15% for walk and 10-20% for the run data) explained the biggest proportion of variance in the data set. Two of the entire stance phase mean CRP variability statistics (HATR-MEAN; KFTR-MEAN) had low factor loadings on all the extracted factors, indicating they explained very little of the variance in the dataset.

Table 7.3. Factor loadings, communalities ($h^2$) and explained variance from the unrotated and orthogonally rotated PCA solution for the walk data. Variables with loadings >.63 are in red and considered important to that respective factor. Loadings <.4 are suppressed for ease of interpretation.
Table 7.4. Factor loadings, communalities ($h^2$) and explained variance from the unrotated and orthogonally rotated PCA solution for the run data. Variables with loadings >.63 are in red and considered important to that respective factor. Loadings <.4 are suppressed for ease of interpretation.

<table>
<thead>
<tr>
<th>Factors Matrices (RUN)</th>
<th>Unrotated</th>
<th>Orthogonal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Run Fl F2 F3 F4 Fl F2 F3 F4</td>
<td>F1 F2 F3 F4</td>
<td>$h^2$</td>
</tr>
<tr>
<td>KFTR-MEAN .840 .554 .421 .464</td>
<td>.740</td>
<td></td>
</tr>
<tr>
<td>HRTR-MEAN .750 .430</td>
<td>.658</td>
<td>.742</td>
</tr>
<tr>
<td>HATR-MEAN .739 .599</td>
<td>.568</td>
<td>.707</td>
</tr>
<tr>
<td>TRAE-10 .683 .493</td>
<td>.614</td>
<td>.644</td>
</tr>
<tr>
<td>HATR-10 .658</td>
<td>.433</td>
<td>.903</td>
</tr>
<tr>
<td>KFTR-10 .643</td>
<td>.483</td>
<td>.924</td>
</tr>
<tr>
<td>KFTR-MS .614</td>
<td>.572</td>
<td>.856</td>
</tr>
<tr>
<td>HRTR-10 .565</td>
<td>.444</td>
<td>.688</td>
</tr>
<tr>
<td>KFH-MS .814</td>
<td>.849</td>
<td>.749</td>
</tr>
<tr>
<td>KFHR-10 .711</td>
<td>.798</td>
<td>.661</td>
</tr>
<tr>
<td>KFHR-10 .642</td>
<td>.774</td>
<td>.626</td>
</tr>
<tr>
<td>HRTR-MS .546</td>
<td>.622</td>
<td>.795</td>
</tr>
<tr>
<td>KAAE-10 .642</td>
<td>.793</td>
<td>.657</td>
</tr>
<tr>
<td>KAAE-MEAN .631</td>
<td>.755</td>
<td>.584</td>
</tr>
<tr>
<td>TRAE-MEAN .491</td>
<td>.407</td>
<td>.587</td>
</tr>
<tr>
<td>TRAE-MS .664</td>
<td>.423</td>
<td>.604</td>
</tr>
<tr>
<td>HATR-MS .466</td>
<td>.517</td>
<td>.591</td>
</tr>
</tbody>
</table>

% total variance 31.1 16.8 12.8 10.8 20.2 19.3 17.0 15.1 71.5

Factor labels
F1 Tibial rotation 10%
F2 Hip rotation stance
F3 Ankle eversion stance
F4 Tibial rotation midstance

7.4.3 Logistic regression analysis

7.4.3.1 Walk
For the walk data factors 1-5 were entered into the logistic regression model. None of the factors were significant and a predictive model could not be generated. This supports the similarities seen in the variability ensemble plots (figure 7.7).

7.4.3.2 Run
Factors 1-4 and KAAE-MS were entered into the logistic regression model. A reduced score on factor 1 (tibial rotation 10%), which equates to reduced variability in the HATR10, KFTR-10 and HRTR-10 variables, was a significant predictor for PFPS (Table 7.5) and
also a significant univariate risk factor ($p = 0.047, <.05$). Factors 2 (hip rotation stance) and 3 (ankle eversion stance) made a significant contribution to the explained variance of the predictive model, as illustrated by the change in the log-likelihood when excluded from the model (Table 7.5). However, factors 2 and 3 were not significant univariate risk factors, the probability values were .215 ($p > .05$) for factor 2 and .404 ($p > .05$) for factor 3. This indicates that these were suppressor variables. A suppressor is a variable that is only significant when another factor is controlled or held constant. In this case, factors 2 (hip rotation stance) and 3 (ankle eversion stance) were only significant when adjusting for factor 1 (tibial rotation 10-20%). Here an increased score on factor 2 (hip rotation stance), which corresponds to increased variability in the HRTR-MEAN, KFHR-MS, KFHR-MEAN, KFHR-10 and HRTR-MS variables, increased the risk of PFPS. Likewise an increased score on factor 3 (ankle eversion stance), which corresponds to increased variability in the KAAE-10, KAAE-MEAN and TRAE-MEAN variables, also increased the risk of PFPS.

### 7.4.3.3 Predictive model

After backwards elimination, the final model contained factor 1 (tibial rotation 10-20%), factor 2 (hip rotation stance) and factor 3 (ankle eversion stance) (Table 7.5). The ROC curve (figure 7.11b) shows that the model was able to separate PFPS from control cases with a sensitivity of 86% and specificity of 89%. The model explained 46% of the variance in the two groups and was a significant predictor of PFPS ($\chi^2 = 13.74; p < .05$) (Table 7.5).

The leverage statistics for the model were satisfactory, suggesting that no single case exerted a significant influence on the model. One case had a high residual, this case belonged to the PFPS group but had a low predicted probability for PFPS membership (figure 7.11a). This case also had a DFBeta score greater than 1 for factor 1, suggesting it had an influence on the size of this coefficient. One other case in the control group also had a high DFBeta score. The analysis was repeated with these two cases removed and the same model was obtained but the coefficients were larger suggesting a bigger effect for the predictor factors. However, this new model also produced three other cases with DFBetas greater than 1 for factor 1. This is likely to be a symptom of the small sample size. If a model is based on a small number of cases, the removal of a case is likely to have a bigger effect on the model parameters than if a larger sample was used. Models derived from
smaller samples are naturally less stable, thus, the first model was presented here since fewer cases exerted an influence on the model parameters.

Table 7.5. Summary of the coefficients, significance tests and odds ratios for the final model produced from logistic regression for the run variability data. The final column reports the significance of the change in the model if the term is removed (LL = log likelihood).

<table>
<thead>
<tr>
<th>Run</th>
<th>β</th>
<th>se</th>
<th>p</th>
<th>Odds ratio</th>
<th>sig. of change in LL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>exp β</td>
<td>95% CI</td>
</tr>
<tr>
<td>F1 TR 10% Stance</td>
<td>-4.253</td>
<td>1.888</td>
<td>.024</td>
<td>0.014</td>
<td>.000</td>
</tr>
<tr>
<td>F2 HR All</td>
<td>1.521</td>
<td>0.737</td>
<td>.039</td>
<td>4.575</td>
<td>1.079</td>
</tr>
<tr>
<td>F3 AE All</td>
<td>1.102</td>
<td>0.586</td>
<td>.060</td>
<td>3.010</td>
<td>.955</td>
</tr>
<tr>
<td>Constant</td>
<td>-4.368</td>
<td>1.583</td>
<td>.006</td>
<td>0.013</td>
<td></td>
</tr>
</tbody>
</table>

$R^2 = .46$ (Nagelkerke)
Model $\chi^2 = 13.74$ (p=.003)
Hosmer and Lemeshow Test: $\chi^2 = 10.409$ (p=.238)

Figure 7.11. Frequency histogram of predicted probabilities for PFPS by group (a) and resulting ROC plot showing the effect of different classification cut off probabilities on the sensitivity and specificity of the predictive model (b).

7.4.4 Vector coding

The similarities and differences between groups noted for the CRP variability plots (figure 7.8) were generally also present in the vector coding plots for the run data (figure 7.9). A similar number of factors with similar factor labels were also extracted from the PCA on the vector coding data (Table E.1 in ANNEX E). Based on the logistic regression analysis,
factor 2 (tibial rotation 10%) was a significant univariate risk factor for PFPS (Table E.2 in ANNEX E), this agreed with the CRP data (Table 7.5). However, unlike factors 2 and 3 from the CRP data, the equivalent factors from the vector coding data did not improve the fit of the predictive model (Table E.2). The explained variance in PFPS outcome from the vector coding predictive model was 32% which was slightly less than the CRP model.

7.4.5 Combined variability and kinematic model (run data)

A secondary analysis on the run data was undertaken to examine how the variability data and conventional kinematic risk factors described in Chapter 6 combine to affect the risk of PFPS. The objective was to examine whether they explain similar variance in PFPS outcome or if they are independent.

Correlation analysis was performed to determine the level of association between factors. There was a mild but significant correlation between kinematic factor 1 (leg rotation) and variability factor 1 (tibial rotation 10%) (Table 7.6). All the other correlations were low and non-significant.

It was not possible to enter all the risk factors into a logistic regression model because there are too many significant predictors (n=6) for the number of cases (n of PFPS = 7). This can cause unstable regression coefficients and complete statistical separation (Tabachnick and Fidell, 2001). Thus to further examine whether the variability and kinematic data were explaining different variance and establish whether they are useful independent measures to include in a model, the most significant factor from the kinematic data was added to the variability model. Table 7.7 shows that the explained variance in PFPS outcome increased from 46% to 55% (Nagelkerke $R^2$) with the addition of the kinematic factor. However, this caused very unstable regression coefficients for each of the factors, and in particular for variability factor 1 as illustrated by the wide confidence intervals.

Table 7.8 shows that a low risk candidate based on the conventional kinematic predictive model was not necessarily a low risk candidate based on the inter-joint coordination variability model.
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Table 7.6. Correlation coefficients (r) between the kinematic and variability risk factors. The significance of each coefficient is given in brackets. Significant correlations are highlighted in red.

<table>
<thead>
<tr>
<th>KINEMATIC</th>
<th>VARIABILITY</th>
<th>F1 (leg rotation) (p-value)</th>
<th>F2 (hip rot. stance) (p-value)</th>
<th>F3 (ank. eve. stance) (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fl (tib. rot. 10%)</td>
<td>.330 (.029)</td>
<td>.049 (.753)</td>
<td>-.095 (.540)</td>
<td></td>
</tr>
<tr>
<td>F2 (hip rot. stance)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F3 (ank. eve. stance)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 7.7. Summary of the coefficients, significance tests and odds ratios when adding factor 1 from the conventional kinematic data to the variability regression predictive model. The final column reports the significance of the change in the model if the term is removed (LL = log likelihood).

<table>
<thead>
<tr>
<th>Run</th>
<th>β</th>
<th>se</th>
<th>p</th>
<th>Odds ratio</th>
<th>sig. of change in LL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>exp β</td>
<td>95% Cl</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Variability F1</td>
<td>-4.47</td>
<td>.201</td>
<td>.026</td>
<td>.01</td>
<td>.00</td>
</tr>
<tr>
<td>Variability F2</td>
<td>1.82</td>
<td>.83</td>
<td>.029</td>
<td>6.16</td>
<td>1.20</td>
</tr>
<tr>
<td>Variability F3</td>
<td>1.33</td>
<td>.67</td>
<td>.047</td>
<td>3.79</td>
<td>1.02</td>
</tr>
<tr>
<td>Kinematic F1</td>
<td>-.92</td>
<td>.55</td>
<td>.095</td>
<td>.40</td>
<td>.14</td>
</tr>
<tr>
<td>Constant</td>
<td>5.09</td>
<td>1.83</td>
<td>.005</td>
<td>.006</td>
<td></td>
</tr>
</tbody>
</table>

R² = .55 (Nagelkerke)
Model χ² = 17.00 (p=.002)
Hosmer and Lemeshow Test: χ²= (p=.855)

Table 7.8. The case-wise predicted probabilities for PFPS for each subject in the PFPS group based on the conventional kinematic logistic regression model and the CRP variability regression model.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Predicted probability for PFPS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Kinematic model</td>
</tr>
<tr>
<td>1</td>
<td>.93</td>
</tr>
<tr>
<td>2</td>
<td>.13</td>
</tr>
<tr>
<td>3</td>
<td>.67</td>
</tr>
<tr>
<td>4</td>
<td>.32</td>
</tr>
<tr>
<td>5</td>
<td>.47</td>
</tr>
<tr>
<td>6</td>
<td>.28</td>
</tr>
<tr>
<td>7</td>
<td>.20</td>
</tr>
</tbody>
</table>
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7.5 Discussion

This was the first prospective study to examine the association between inter-stride joint coordination variability and the development of PFPS. In light of reduced variability for factor 1 (tibial rotation 10%) and increased variability for factors 2 (hip rotation stance) and 3 (ankle eversion stance) being significant risk factors, the hypothesis of reduced variability in individuals who developed PFPS can only be partially accepted. The rationale for the study was concerned with the biomechanical implications of movement variability as opposed to the nature and causes of variability, as such, this discussion is focused on the biomechanical aspects of variability.

7.5.1 Comparison of ensemble plots with other studies

It is not possible to directly compare the ensemble plots with previous work because of the different methods used to calculate CRP variability and the different methods used to quantify and decompose joint angles (Hamill et al., 1999; Heiderscheit et al., 1999). Additionally, most studies have only presented summary statistics or an exemplar subject plot and omitted the mean ensemble plots (Selles et al., 2001; Heiderscheit et al., 2002), and no CRP variability plots in any format could be found for walking. However, similar to the trends demonstrated in Hamill et al. (1999) and Heiderscheit et al. (1999) during running, there was a subtle pattern of increased variability during initial stance in the joint couples about the knee. From a dynamical systems perspective it has been suggested that this may reflect the different functional demands over the gait cycle and allow the neuromuscular system the flexibility to adapt to environmental perturbations (Hamill et al., 1999).

The hip rotation - tibial rotation joint couple generally displayed the largest amount of variability. This was present to a lesser extent in the data presented by Hamill et al. (1999). It is possible that this additional variability was attributable to instrument or methodological error in the hip rotation joint angle as opposed to true biological variability. As discussed in chapter 6, more research is needed to validate the varied hip rotation patterns seen in the literature so that some of the uncertainty is removed from the interpretation of this variable.
Chapter 7. Joint coordination variability

7.5.2 PCA

The factor analytic method reduced the data from 18 variables into 5 factors for the walk and 4 factors for the run data. Similar to the kinematic data described in Chapter 6, the factor loadings were well partitioned and maintained the theoretical and structural relevance of the data in terms of containing variables from patterns of joint couples or from a particular period of stance. That the vector coding data produced exactly the same factors as the CRP data (ANNEX E) also gives some support to the structure of the variability dataset that was produced from the PCA. Unfortunately, there are no published studies that have used PCA on variability data from which to compare with the extracted factors here.

7.5.3 Variability risk factors

Similar to the results for the angular kinematic data (Chapter 6), there were no significant findings from the walk data, supporting the need for studies into running and anterior knee pain. The rest of this section will thus focus on the run data.

7.5.3.1 CRP run

Reduced CRP variability in factor 1 (tibial rotation 10-20%) during running was a significant independent risk factor. Heiderscheit et al. (2002) found significantly less vector coding variability in thigh–shank rotation from late swing to 10% of stance in a group of females with patellofemoral pain. Whilst this joint couple was an important constituent of factor 1 in our study, the period of stance was before that described by factor 1 and so cannot be used to support the results from this study. Further a visual inspection of the data presented by Hamill et al. (1999) also lends no support for this finding. It is important to reiterate that these were both case-control studies.

Paradoxical to the effect of factor 1 (tibial rotation 10-20%), increased variability in factors 2 (hip rotation stance) and 3 (ankle eversion stance) increased the risk of PFPS. However, these two factors were significant only after adjusting for the effects of factor 1, as independent risk factors they did not explain a significant proportion of the variance in PFPS outcome. Whilst Hamill et al’s (1999) study of individuals with patellofemoral pain included no statistical analysis, it is interesting to note that for two of the joint couples that were components of factors 2 and 3, hip adduction-tibial rotation and tibial rotation-foot eversion, the CRP data were visually similar between groups. Likewise, Heiderscheit et al.
(1999) found similar results using the vector coding method. It is possible that these factors could have been important in these studies if analysed using multivariate statistics.

The units of the CRP variability data (phase angle degrees) make it difficult to clinically interpret the magnitude of the difference between groups. However, figure 7.8 shows that for the joint couples that loaded heavily on factor 1 (Table 7.4), the mean difference between groups was slightly less than one standard deviation. It is also interesting that there was very little between-subject variability in the PFPS group over this time period as illustrated by the narrow standard deviation lines (figure 7.8). The odds ratio for factor 1 (Table 7.5) suggests that a decrease of one standard deviation in factor 1 increased the odds of PFPS by a factor of seven. For factors 2 and 3, inspection of the variability plots (figure 7.8) reveal very small differences between groups for the variables that define these factors. This is not surprising given that these factors were only important when adjusting for factor 1 (tibial rotation 10-20%). However, one should be cautious about interpreting the size of the effect by the regression coefficients in this study given the wide confidence intervals and small sample size.

7.5.3.2 Vector coding data
The logistic regression model for the vector coding data contained one significant factor, this factor was the equivalent of CRP factor 1 (tibial rotation 10-20%) (ANNEX E). Whilst this does not validate the result from the CRP method, it provides some reassurance that this finding wasn’t due to a quirk within the calculation of the CRP. The VC model was not improved by the addition of any of the other factors, and explained less of the variance in PFPS outcome compared to the CRP method. The VC method can be prone to artefacts when clusters of points are in close proximity on the angle-angle diagram as occurs when a joint angle changes direction. This may be responsible for the discontinuities that are present in the ensemble plots (figure 7.9). Heidersheit et al. (2002) suggested that during these time periods variability may be overestimated and prone to error. Accordingly, it has been suggested that this method may be less sensitive than the CRP method in quantifying subtle differences in variability (Wheat and Glazier, 2006).

7.5.3.3 Mechanisms for variability
Factor 1 (tibial rotation 10%) and 2 (hip rotation stance) both contain joint couples that include hip rotation and knee flexion over 10-20% of stance. Since reduced variability for
factor 1 was a risk factor and increased variability in factor 2 was a risk factor, this would suggest that the reduced variability in the variables for factor 1 was caused predominantly by less variability in the tibial rotation movement over 10-20% of stance. Although not proven, the possible biomechanical implications of reduced variability have been discussed. If reduced variability does localise loading onto more concentrated tissue areas (Hamill et al., 1999; Heidersheit et al., 1999, 2002, James, 2004), then this may be an explanation for why reduced variability in tibial rotation coordinative relationships over early stance increased the risk of PFPS in this study. Unfortunately, this explanation does not account for why the addition of increased variability in the coordinative structures containing hip rotation (factor 2) and ankle eversion (factor 3) enhanced the predictive model. However, excessive variability has been cited as detrimental to musculoskeletal health. For example, previous studies of balance in the elderly have shown that when the task constraints are not met, too much variability can indicate an unstable system (van Emmerik and van Wegen, 2000). Given the similarities in aerobic fitness and prior activity levels between groups, it is unlikely that the task constraints of running were not met in the PFPS group by inexperience or fitness. The muscles that invert the foot and externally rotate the hip are both active during the first half of stance (McClay et al., 1991). It is possible that deficits in strength (Ireland et al., 2003) or neuromuscular inhibition (Cesarelli et al., 1999; Voight et al., 1991), which have been reported in patients with patellofemoral pain, may be control mechanisms for variability. However, only one published study has examined any of the cited risk factors for AKP as a control mechanism for variability. This study examined the relationship between the Q-angle and variability and found no significant association (Heiderscheit et al., 2000). There is a need for more research into the control mechanisms of movement variability. One may speculate that muscular or neuromuscular deficits are associated with increased variability and structural abnormalities are associated with decreased variability.

However, it is also possible that the findings for factors 2 and 3 were due to chance, and given the absence of prospective studies and small sample size, it is important to validate these results in other samples. Issues surrounding the validity of the hip rotation variable were discussed in section 6.4, and despite the concerns about the validity of the frontal plane knee and ankle kinematics, these data were included in this analysis because it was not known how this would affect the validity of the variability data. Future research should
investigate the reliability and sensitivity of measures of variability to distortions from marker placement errors and model assumptions.

7.5.4 Predictive model

7.5.4.1 Variability

The final model explained a large proportion of the variance in PFPS outcome and was a good fit for all the data except one case. Similar to the predictive model based on the lower extremity joint angle kinematics, the model was able to separate PFPS and controls with impressive sensitivity and specificity. This justifies further investigation into the role of variability in the maintenance of joint health.

7.5.4.2 Combined variability and kinematic model

There was some shared variance between variability factor 1 and kinematic factor 1 as indicated by the correlations and reduced importance of the kinematic factor when added to the variability predictive model. It is not possible to partition the shared variance between factors to explain which factor is most important using logistic regression. This has to be done by experiment and study design. Related to the discussion in 7.5.3.3, future studies may wish to examine whether there is a relationship between the magnitude of certain angular kinematics and movement variability. However, there was some separate variance between these factors as illustrated by the correlations between factors. Further, it is clear from the case-wise predicted probabilities for PFPS based on the kinematic and variability model (Table 7.8), that a subject with high scores on the salient kinematic factors did not necessarily also have high scores on the salient variability factors. Unfortunately, there were too few cases per number of significant variables to validly explore the relationship between conventional kinematics and variability. Larger cohort studies are required if more salient variables are to be added to this predictive model.

7.5.5 Limitations

The CRP transforms intelligible angular kinematic data into a less intuitive format that is difficult to interpret. This is compounded by the different calculation methods that have been used for the CRP. In motor control a 0-360° phase angle representation has typically been used (Sholz, 1990). However, this representation has been discarded by biomechanists in favour of ranges such as 0-180° (Hamill et al., 1999) and -90 – 90°
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(Wheat and Glazier, 2006), possibly to avoid the discontinuities that occur at 0 and 360° (Wheat and Glazier, 2006). It is possible to retain the 0-360° representation and use circular statistics to calculate variability in a similar manner to the vector coding method (ANNEX E), however, there is no justification in the literature for why the new representations were chosen in favour of circular statistics.

The use of CRP to examine the synchronicity between two segments in the phase plane is based on the assumption that both segments have a sinusoidal time history (Deidrich and Warren, 1996; Peters et al., 2003). Many of the kinematic variables fail to meet this assumption. Despite this concern, the CRP has been used widely to examine coordination and coordination variability of the lower extremity (Hamill et al., 1999; Heiderscheit et al., 1999, Li Li et al., 1999) and trunk (van Emmerik et al., 1999; van Emmerik and Wagenaar, 1996; Selles et al., 2001) during walking and running. It should be emphasised that the technical papers commenting on the limitation of CRP for non sinusoidal data refer specifically to determining the degree of synchronicity in movement and not to movement variability. The effects on the calculation of variability are unknown. More work is required to establish the assumptions necessary for the application of CRP to study inter-joint coordination variability, and to understand the most appropriate normalisation method. An alternative method to the CRP that does not require the sinusoidal assumption (Deidrich and Warren, 1996; Peters et al., 2003) or contain issues surrounding normalisation (Kurz and Stergiou, 2002; Peters et al., 2003) is the vector coding approach (ANNEX E). And since similar results were obtained for factor 1 using the vector coding method, this supports the stability of this finding in the CRP data to sinusoidal violations and normalisation and phase angle calculation anomalies.

Despite the limitations of the CRP, it has the advantage of containing both spatial and temporal data. And since it contains higher dimensional information (Burgess Limerick et al., 1993), it is considered a more sensitive measure of joint coordination than the vector coding method (Wheat and Glazier, 2006). It is possible that this was subtly demonstrated in the data presented here. Other theoretical and philosophical merits have also been advocated. The traditional approach to biomechanical research into injury has been described as hierarchal and reductionist (Glazier et al., 2006). This is illustrated by many of the early studies that assessed the role of static biomechanical factors such as Q-angle and foot type, and studies that correlated discrete variables such as peak pronation with
injury. These studies ignored the interaction or coordination with other movements, and have been described as outcome driven. In contrast, the dynamical systems and the CRP approach establishes a continuous measure of inter-joint coordination and attempts to integrate the role of the underlying sensorimotor system dynamics, accordingly it has been described as process-driven (Glazier et al., 2006). Davids et al. (2006) suggested that this approach may be more productive and enable a greater understanding of the causes of injury rather than the more traditional descriptive approach. It is thought that future research in this area would benefit from collaborative ventures involving experts in biomechanics and motor control.

7.5.6 Applications

If variability is a risk factor it may have a number of applications. Aside from monitoring progress through rehabilitation and treatment efficacy, it possibly counters the common approach of removing variability from rehabilitation practice. In this sense, the effect of exercise therapeutics may be more beneficial when the sensorimotor system is in a transitional state. This could be achieved by varying known control parameters such as the speed of movement (Deidrich and Warren, 1995).

7.5.7 Conclusions

Based on the results from this study, inter-joint coordination variability during running would appear to be an important factor in why certain individuals develop PFPS. However, given the concerns over the calculation of CRP, further work should investigate the assumptions and boundaries of different approaches to quantifying variability. And since this is the first prospective study on a relatively small sample, attempts should also be made to cross-validate these findings in larger samples of anterior knee pain where data can be combined with conventional descriptors of gait.

Lastly, this work may be of use in stimulating hypotheses for future experimental work into the causes of variability. For example, one may hypothesise that particular strength deficits, lower limb morphological features, fatigue status, or footwear may be associated with variability. Knowledge of these factors would allow the design of suitable interventions to affect variability should it prove to be an important component of musculo-skeletal health.
Chapter 8

A 3-year longitudinal follow-up and validation study

8.1 Introduction

8.1.1 Rationale

The prospective cohort study reported in chapters 6 and 7 found three important risk factors for PFPS from the running joint angular kinematic data and three important risk factors from the movement variability data. Both these sets of factors had impressive predictive validity, explaining nearly 50% of the variance in PFPS outcome. Unfortunately the small sample size of the PFPS group (n=7) that was used to create the prediction models carries statistical and clinical limitations. A statistical model is prone to over fitting to the particular dataset that was used to construct it, this is a particularly important limitation when the sample size is small. It means that the predictive ability of a model will typically suffer shrinkage when applied to a different sample (Thomas et al., 2005). This was illustrated in the prospective data of chapters 6 and 7 by the wide confidence intervals for the regression coefficient estimates. The clinical limitations originate from the lack of agreement on the classification of anterior knee pain. This was highlighted in the literature review contained in Chapters 2 and 3 where numerous aetiological factors for the cause of pain, and pathophysiological explanations for the source of pain were discussed. As a result, it is not known whether the findings from the prospective study are applicable to other samples of anterior knee pain. There is thus a requirement to cross-validate the risk factors and predictive models found in the earlier prospective cohort study with another sample of PFPS subjects.

To cross-validate the findings from the previous study and minimise the possibility of erroneously interpreting inhibited movement as a risk factor, prospective rather than cross sectional data are required. Unfortunately, another prospective cohort study was not feasible given the requirement for such a large sample size. However, a solution was
apparent from a previous 6-year longitudinal follow-up study of anterior knee pain by Milgrom and colleagues (1996), where it was found that immunity from anterior knee pain in a 14 week military training program did not necessarily mean immunity thereafter. Specifically, 32% of Israeli Army conscripts who did not sustain AKP during basic training had some symptoms of anterior knee pain at a 6-year follow-up point. It was thus considered likely that a substantial proportion of the control group from the previous cohort may have since developed anterior knee pain, and that these individuals could be used to form another PFPS group to cross-validate the findings from the previous study. A longitudinal follow study of the non-injured controls from the cohort described in Chapter 6 was thus undertaken.

8.1.2 Aims and hypotheses

The aim of this study was to cross-validate the results reported from the earlier prospective study using a new group of PFPS subjects obtained from a 3 year follow up study. It was hypothesised that the joint angle kinematic factors and movement variability factors that were significant in the running data for the previously reported PFPS sample (Tables 6.7 and 7.5) would also be significant factors in a new PFPS group.

8.2 Method

The protocol was approved by the Royal Navy Personnel Research Ethics Committee of the Ministry of Defence. Written informed consent was obtained from all subjects who participated in the study.

8.2.1 Design

A 3-year longitudinal follow-up study was undertaken. The study followed up a control group that participated in a previous cohort study and remained free of lower limb overuse injury during a 12-week military training program. A combination of postal questionnaire, telephone interview and clinical appointment was used to ascertain cases of PFPS. The baseline gait data collected on day 1 of CMS(R) Army training 3 years prior to this follow up were examined to determine risk factors for PFPS.
8.2.2 Subject selection

The cohort was selected from the participants of the prospective study described in chapter 6. These subjects (n=83) had passed CMS(R) phase one training and entered the Army without sustaining PFPS or any other lower limb overuse injury.

8.2.3 Follow up

Given the high levels of operational deployment and the transient nature of life in the armed forces, the main obstacle to this study was ensuring an adequate response rate. To maximise participation, a three pronged approach of postal questionnaire, telephone interview and medical centre appointment was executed. The most recent postal address and telephone number of each subject was obtained from the Army personnel centre in Glasgow. A systematic approach to contacting potential participants was adopted. These procedures are described chronologically below:

(i) A letter, subject information sheet, and knee pain response form (ANNEX F) was initially sent to all subjects with a stamped return-addressed envelope. The information pack and questionnaire gave subjects the opportunity to object or consent to participation in the study. The questionnaire consisted of a number of screening questions to establish an episode of knee pain and assemble information on the nature of the pain in terms of precipitating events, date of onset and affected limb(s). A functional index questionnaire (FIQ) (Kujala et al., 1993) was included within the response form to further assess knee pain symptoms and morbidity (ANNEX F). Respondents were also requested to provide a convenient contact number.

(ii) At four weeks, the contact details of all non respondents were re-checked with the Army personnel centre. An attempt was then made to contact all non-respondents by telephone, and where contact was made, the knee pain response form was administered verbally. At the start of the telephone interview, participants were again given the opportunity to refuse participation. Where a contact telephone number could not be obtained, the subject's medical centre was contacted and an attempt was made to administer the questionnaire through the individual's medical centre.
(iii) At 8 weeks, all non-respondents and subjects who were unable to be contacted by telephone were sent a revised and shortened postal questionnaire. Again prior to this, last known addresses were re-checked. This process was repeated at 12 weeks.

(iv) Individuals who reported knee pain in either (i), (ii) or (iii) were contacted by telephone by a senior chartered physiotherapist. This phone call sought to verify their questionnaire responses and obtain a brief clinical history regarding their knee pain. If a subject was suspected of having overuse anterior knee pain, they were invited to attend a specially arranged research clinic at the Defence Medical Rehabilitation Centre (DMRC) as an outpatient. An outpatient appointment was subsequently sent to all individuals suspected of having had or currently suffering from anterior knee pain. The appointment also contained a letter to the patient’s GP explaining the patient’s participation in the study. At the same time a letter was sent to the participant providing a written explanation of the clinic.

8.2.4 Anterior knee pain research clinic

A single consultant physician with a background in sports medicine made all diagnoses. A clinical history and physical examination was undertaken and a diagnosis of PFPS was made according to the inclusion and exclusion criteria outlined in section 6.2.6.1 and Table 6.2. Any subjects with a diagnosis of PFPS were allocated to the PFPS(3year) group.

Given that subjects were exposed to different activity levels over the three year period, an attempt was made to quantify the activity levels of the PFPS group using the International Physical Activity Questionnaire (IPAQ) (short version). The IPAQ short questionnaire assesses the ‘last 7 day’ activity levels and has been validated and assessed for reliability in 12 international populations (Craig et al., 2003). The IPAQ was administered to all patients who attended the research clinic.

8.2.5 Analysis and statistics

The kinematic run data collected at baseline 3 years prior to this follow up study were filtered and normalised, and used to calculate joint angles as discussed in Chapter 5. The same eight joint angles and variables were decomposed as detailed in section 6.2.5.

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Similarly, CRP variability was calculated for the same inter-joint coordinative relationships as detailed in section 7.3.2.

Ensemble plots were calculated for the kinematic and variability data and used to make a visual comparison between groups. Factor scores were calculated for the kinematic and variability data using the PCA factor models explained in Tables 6.5 and 7.3. The standardised factor scores were used as variables for the regression analysis. The factors that were salient predictors in the final logistic regression models described for the joint angular kinematic and movement variability data in Tables 6.6 and 7.4 were entered into a regression model to predict PFPS(3year) group from control group membership. The control group was described in section 6.2.6.2. For the kinematic data this consisted of factors 1 (leg rotation), 3 (hip adduction) and 6 (knee rotation), and for the variability data this comprised factors 1 (tibial rotation 10%), 2 (hip rotation stance) and 3 (ankle eversion stance). A forced entry logistic regression method was used to calculate the odds ratios for each of these factors in the model. The Wald statistics for the factor beta coefficients, the odds ratios and the significance of the explained variance in the model were compared to the findings from the previous prospective study.

8.3 Results

8.3.1 Follow-up

Sixteen subjects were unable to be contacted due to no known address. Fifty-nine out of the remaining 67 responded to either the questionnaire or telephone follow-up, and there were four nil questionnaire returns. This gave a response rate of 71% (59/83). There were no significant differences in age, height, weight, BMI, pre-enlistment activity levels or aerobic fitness at baseline between the respondents and non-respondents (p>0.05). Eighteen subjects reported anterior knee pain and were invited to the research clinic. Five subjects did not attend, the reasons for non-attendance were not established. Four subjects were deployed overseas and unable to attend. Nine patients were ultimately seen in the clinic and eight were diagnosed with PFPS (figure 8.1)
8.3.2 Sample description

There were no significant differences in age, height, weight, BMI, pre-enlistment activity levels or aerobic fitness at baseline between the PFPS and control group (p>.05) (Table 8.1). At baseline 57% of the control group versus 38% of the PFPS(3year) group were smokers and 5% of the control group versus 13% of the PFPS(3year) group reported a previous ankle injury. Interestingly, the proportion that had suffered a previous ankle injury in the PFPS(3year) group increased to 63% at the 3 year follow up point. The proportion of smokers remained the same.

8.3.3 Pain and symptoms

Four subjects had unilateral pain (3 left, 1 right), and four subjects had bilateral pain where the left was more painful than the right in 3 subjects. The median duration of symptoms was 2.5 years (IQR: 2.1-2.5; range 0.5-2.5). The median score on the FIQ was 93/100 (IQR: 81-97; range: 43-100, where 100 equates to full function). The median pain (VAS) was 8 (IQR: 3-24; range: 0-57). None of the subjects were medically downgraded as a result of their AKP symptoms. The mean activity level (IPAQ) for the PFPS(3year) group
was 10,598 MET-min per week. Only 2/8 subjects reported a reduction in their typical activity due to their pain.

Table 8.1. Descriptive characteristics of the control and PFPS(3year) group at baseline.

<table>
<thead>
<tr>
<th>Variable</th>
<th>BASELINE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control (n=37)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>mean</td>
</tr>
<tr>
<td></td>
<td>18.93</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.78</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>69.83</td>
</tr>
<tr>
<td>BMI (kg.m²)</td>
<td>21.88</td>
</tr>
<tr>
<td>Pre-enlistment running (miles.week⁻¹)</td>
<td>8.5</td>
</tr>
<tr>
<td>2.4km run time (s)</td>
<td>605.7</td>
</tr>
</tbody>
</table>

8.3.4 Group ensemble plots and logistic regression

8.3.4.1 Joint angle kinematics

Inspection of the ensemble plots for the kinematic data revealed little dissimilitude between the PFPS(3year) and control group for any of the joint angles. Whilst the hip adduction and tibial rotation plot showed a similar direction of differences to that seen in the previous prospective study (figures 6.5 and 8.2), the differences were smaller and of questionable clinical relevance. Further, the trends of differences in the hip and knee rotation plots were opposite to those found in the earlier study (figure 6.5 and 8.2). There was a disparity between groups at terminal stance in the ankle eversion plots (figure 8.2), however, inspection of the individual data suggested that this was due to two outliers with prolonged eversion into late stance. As expected given the parity in the ensemble plots for the PFPS(3year) and control group, neither factor 1 (leg rotation), 3 (hip adduction) or 6 (knee rotation) were significant in the logistic regression model (Table 8.2).

A secondary analysis showed that none of the eight factors were significant when analysed independently. Finally, a similar approach to that used in Chapter 6 of backwards stepwise (likelihood ratio) logistic regression, also failed to produce a statistical model that was able to explain any of the variance in the PFPS(3year) group above that explained by the model constant.
Figure 8.2. Mean ensemble joint angle curves for the PFPS(3year) and control group during running. KEY: Grey area = control ± 1SD; Dashed line = control mean; Red line = PFPS(3year) mean.
### Chapter 8. Longitudinal follow up & validation study

**Table 8.2.** Odds ratios and coefficients from the logistic regression model to predict PFPS(3year) outcome using the salient **kinematic** factors from the study detailed in chapter 6.

<table>
<thead>
<tr>
<th>Factors (kinematics)</th>
<th>β</th>
<th>se</th>
<th>p</th>
<th>Odds ratio</th>
<th>exp β</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>F1 Leg rotation position</td>
<td>.384</td>
<td>.501</td>
<td>.444</td>
<td>1.47</td>
<td>.55</td>
<td>3.92</td>
</tr>
<tr>
<td>F3 Hip adduction</td>
<td>.437</td>
<td>.353</td>
<td>.215</td>
<td>1.55</td>
<td>.78</td>
<td>3.09</td>
</tr>
<tr>
<td>F6 Knee rotation</td>
<td>.376</td>
<td>.389</td>
<td>.335</td>
<td>1.47</td>
<td>.68</td>
<td>3.12</td>
</tr>
<tr>
<td>Constant</td>
<td>-1.74</td>
<td>.466</td>
<td>.000</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\[ R^2 = .11 \text{ (Nagelkerke)} \]

Model \( \chi^2 = 3.22 \text{ (p=360)} \)

Hosmer and Lemeshow Test: \( \chi^2=7.976 \text{ (p=.335)} \)

### 8.3.4.2 Inter-stride joint coordination variability

The mean plots for movement variability were similar between groups over the stance phase of running (figure 8.3). There was only a very slight pattern of reduced variability and reduced between subject variance in the PFPS(3year) group over the 10-20% period of stance for the hip adduction – tibial rotation and knee flexion – tibial rotation plots, similar to the previous PFPS group (figure 7.8 and 8.3). None of the factors that were significant predictors for the PFPS group were significant for the PFPS(3year) group, likewise the predictive model based on these factors also did not explain a significant amount of the variance in outcome (Table 8.3). A secondary analysis using a backwards stepwise (likelihood ratio) logistic regression procedure also failed to produce a statistical model.

**Table 8.3.** Odd ratios and coefficients from the logistics regression model to predict PFPS(3year) outcome based on the salient **variability** factors from the study detailed in chapter 7.

<table>
<thead>
<tr>
<th>Factors (variability)</th>
<th>β</th>
<th>se</th>
<th>p</th>
<th>Odds ratio</th>
<th>exp β</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>F1 Tibial rotation 10% stance</td>
<td>-.068</td>
<td>.435</td>
<td>.875</td>
<td>.93</td>
<td>.40</td>
<td>2.19</td>
</tr>
<tr>
<td>F2 Hip rotation all</td>
<td>-.542</td>
<td>.434</td>
<td>.211</td>
<td>1.72</td>
<td>.74</td>
<td>4.02</td>
</tr>
<tr>
<td>F3 Ankle eversion all</td>
<td>-.352</td>
<td>.455</td>
<td>.439</td>
<td>.70</td>
<td>.29</td>
<td>1.71</td>
</tr>
<tr>
<td>Constant</td>
<td>-1.602</td>
<td>.419</td>
<td>.000</td>
<td>.20</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\[ R^2 = .07 \text{ (Nagelkerke)} \]

Model \( \chi^2 = 1.993 \text{ (p=.574)} \)

Hosmer and Lemeshow Test: \( \chi^2=8.964 \text{ (p=.255)} \)
Figure 8.3. CRP variability plots over the stance phase of running for the six joint movement couples.

**KEY:**
- CONTROL mean
- CONTROL ± 1SD
- PFPS(3year) mean
- PFPS ± 1SD
8.4 Discussion

This 3 year follow up study sought to ascertain a new PFPS case group and cross-validate the results from a previous study. The response rate was acceptable and there was no evidence to suggest that the non respondents were biased by any demographic or physical characteristics. The gait characteristics of a group of subjects who developed PFPS after initially remaining free of anterior knee pain during a 12-week physically arduous training program were reported, and there was no evidence to suggest that there were any common gait characteristics in this group that were risk factors for PFPS. The hypothesis that similar risk factors to the previous PFPS group (Chapters 6 and 7) would be seen in this sample of PFPS subjects should thus be rejected.

8.4.1 Differences between PFPS samples

8.4.1.1 Pain and symptoms

There were a few differences in the characteristics between the PFPS(3year) group and PFPS group from the first prospective study that may have been responsible for the conflicting findings. The levels of pain and morbidity were noticeably different. The PFPS(3year) sample had very mild symptoms of pain, as indicated by the FIQ and VAS pain scores, compared to the previous pain group and also to other PFPS samples where reported in the literature (Nadeau et al., 1997; Salsich et al., 2001). Further, only 2/8 subjects reported that the pain caused a significant reduction in their normal activities.

8.4.1.2 Extrinsic factors

That the PFPS(3year) group did not contain any inherent risk factors in their gait, would suggest that there were other causes of their pain. The PFPS(3year) group were still very active, with an IPAQ activity score approximately 4 times greater than the European population average (Craig et al., 2003). The high ‘last 7 day’ activity levels in this group (IPAQ) hints that external factors may have predominated in their aetiology. Unfortunately, this interpretation cannot be confirmed because no activity data were collected for the control group and even the data for the PFPS(3year) group only refer to the recent exposure to activity (last 7 days). Quantifying activity levels validly is not trivial, and the IPAQ was administered to the PFPS(3year) group as a compromise in recognition of the potential importance of activity as a risk factor. Even the short version of
the IPAQ takes 10 minutes to complete and required assistance. For this reason it wasn’t included in the original postal questionnaire. The response rate for postal surveys such as these can be affected by the amount of information and time required from the participant (Silman and Macfarlane, 2002).

Subjects in this study could have been exposed to many other factors that may have caused their knee pain. For example, in addition to training load, there are factors such as footwear and type of training e.g. hill running, which were not controlled or adjusted for. This is the main disadvantage of this follow up study compared to the previous prospective study where subjects had a relatively homogenised exposure to risk factors over the injury surveillance period that included standardised activity, footwear, training surfaces, nutrition and sleeping patterns.

8.4.1.3 Time to injury onset
The PFPS(3year) group had a different time onset of injury. The difference in the time to symptom onset between groups could also be related to the factors that caused the injury. It seems reasonable to hypothesise that pain may occur earlier in subjects with characteristics that make them more predisposed to higher joint stress (e.g. gait kinematics). In this sense, time to symptom onset may also be an indicator of aetiology, and future studies may wish to model this using multivariable statistics such as Coxe’s regression (e.g. Knapik et al., 2001).

8.4.1.4 Diagnosis
The classification criteria for PFPS adopted from Thomee et al. (1999) for this study is a practical and pragmatic approach to diagnosis. As discussed in section 2.5, there are numerous other classification systems that use a combination of aetiological, pathological, surgical and practical approaches. Anterior knee pain has been differentiated into more than 40 sub classifications, many of which overlap (Holmes and Clancy, 1998). It is possible for a number of these subcategories to exist under the umbrella of Thomee et al.’s (1999) diagnosis. Further there are also different theories for the pathophysiology of anterior knee pain such as a neural and intra-osseous pressure model, where mechanical loading is not the predominating inciting mechanism. Given the different morbidity, levels of pain, and time of onset, it is possible that the source and pathophysiology of pain were different in the majority of the PFPS(3year) group compared to the previous case group. It
was also noted in the literature review that this could be one source of the variance in evidence for the risk factors for AKP (e.g. see Table 2.2).

8.4.2 Statistical errors and research design flaws

It is important to consider whether statistical errors and design flaws could have caused the conflicting findings.

8.4.2.1 Statistical errors

It is possible that the results of the first study were type 1 statistical errors and the null findings in this study actually reflect the true benign influence of gait kinematics. However, this seems unlikely given the control of the type 1 error rate (5%), the effect size (figure 6.3) and the effect of the small case group on reducing the statistical power for the first study.

Alternatively, the results from this follow up study may be type 2 statistical errors. There were four similar trends in the data set to the previous study, these were for the hip adduction and tibial rotation joint angles, and for hip adduction - tibial rotation and knee flexion - tibial rotation variability waveforms. However, the small magnitude of differences and the absence of agreement in other variables indicate that insufficient study power is an unlikely explanation for these findings. These trends can only be interpreted as random sampling differences.

8.4.2.2 Research design flaws

Another explanation for the different findings concerns the validity of the gait data as a true representation of each individual’s gait at the time of injury. For example an individual’s gait may have changed since baseline from a healthy to a risk pattern. If so this would mean the baseline measures of gait were invalid as exposure variables. Potential factors that may induce gait changes are age, physical stature/ body weight or exercise/ training effects. These are discussed below.

More than 75% of the pain group had suffered symptoms for at least 2 years, as such the interval from gait assessment to injury was less than 12 months for the majority of subjects. There is no evidence or theoretical basis to suggest that gait alterations related to
age could have occurred over such a short time period in this cohort. Unfortunately, previous studies have only focused on age related changes in the young (age 20-30) versus the elderly (age 70+) (DeVita and Hortobagyi, 2000; McGibbon and Krebs, 1999), or examined the development of paediatric gait through to adolescence (Cupp et al., 1999; Ganley and Powers, 2004).

Whilst each subject’s weight and BMI was not known at the onset of injury, at the 3 year point this had increased from 73.9 to 79.5kg and 22.6 to 24.8kg.m² respectively. However, this is still classified as healthy according to international guidelines (ACSM, 2005), and there is no evidence to suggest a change this small could have influenced gait.

The other possible explanation is that exercise or training may have induced gait changes. Very few studies have examined the effect of exercise on gait in a healthy population from an injury prevention or risk perspective. Most of the studies in this area have been on elderly populations (e.g. Kerrigan et al., 2003), in populations with neuromuscular disease (e.g. Morton et al., 2005) or populations with degenerative joint disease (e.g. Fisher et al, 1997). Only one study on the effect of exercise on gait in a healthy population could be found, this study reported no changes in any sagittal plane kinematic parameters following 6 weeks of running-based training (Lake and Cavanagh, 1996). There are no published studies on the effects of exercise on frontal or transverse plane kinematics.

Overall, it would seem that training effects are the only possible source of gait changes that could have invalidated the baseline measurements. And unfortunately, this explanation is purely speculative.

8.4.3 Conclusions

The most sensible and supported explanation for the null findings in this study is that the PFPS(3year) group were a different case group with a different aetiology to the PFPS group in chapter 6. Given the high activity levels and different time onset of symptoms, it is speculated that over activity may have been the prevalent aetiological feature that was the source of pain in this group. If this is true then it ascribes to the typical intrinsic and extrinsic risk factor model for overuse injury as illustrated in figure 8.4.
Figure 8.4. The typical fatigue model for overuse injury describing the interaction between intrinsic risk factors and extrinsic factors/ training load (e.g. Hreljac, 2000a; James et al., 2004; Messier and Pittala, 1988)

The importance of multivariate studies and quantifying activity levels has been discussed throughout preceding chapters, and it is possible that with accurate measurement of activity exposure and appropriate statistical adjustment, similar gait risk factors to the previous study could have been found.

However, the null findings in this study do not provide evidence to cross-validate the findings of the previous prospective study. Given the mild symptoms in the PFPS(3year) group, it is recommended that cross validation studies are continued but in groups with more severe symptoms of pain and using studies with appropriate control or adjustment for factors such as activity levels.
Chapter 9

The effect of PFPS on gait assessment:
A preliminary study

9.1 Introduction

9.1.1 Background

The prospective cohort study is the most advantageous design to study biomechanical risk factors for injury. However, the labour intensity of many biomechanical methods and the requirement for a large sample size to capture an adequate case group renders a prospective study impractical for many biomechanical investigations. Hence the almost universal use of the case-control design in this area.

The central problem with the case-control design lies with the interpretation and temporal attribution of a significant association. For example, the prospective study of gait detailed in this thesis found opposite findings for hip and tibial rotation to those reported in case-control studies (Cuderford and Yack, 2000; Dillon et al., 1983; Powers et al., 2002), indicating that results from case-control studies may reflect gait compensations or inhibited movement due to pain rather than risk factors for pain. Case-control studies of AKP have also reported reduced ankle eversion (Duffey et al., 2000) and decreased anterior-posterior propulsive ground reaction force (Duffey et al., 2000; Messier et al., 1991) during running, and reduced knee flexion (Nadeau et al., 1997; Powers et al., 1999) and knee extensor moment during walking (Heino and Powers, 2002; Powers et al., 1999) and stair ambulation (Salsich et al., 2001). Studies of isokinetic and isometric knee joint exercise using a similar design have also found inhibited quadriceps muscle activity (Grabiner et al., 1994). And in further support of the compensation interpretation, one study demonstrated increased loading response knee flexion during gait following temporary alleviation of symptoms using patellar taping (Powers et al., 1997a).
A few studies have reported reduced walking velocity, cadence and stride length in PFPS subjects (Powers et al., 1997a; 1999; Powers et al., 2002). Powers et al. (1997a) also found an increase in stride length during inclined walking in a group with PFPS following relief of symptoms with patellar taping. There are some conflicting findings, for example, two studies found no differences in the temporal-spatial characteristics during walking (Heino and Powers, 2002; Nadeau et al., 1997). There is little information on the temporal-spatial data during running in patients with AKP. One study reported a small increase of 6ms in support time in a group with AKP (Duffey et al., 2000), however, stride length and cadence were not analysed.

The temporal sequence or pathway of an association found between gait and AKP in a case-control study is a fundamental methodological issue that requires clarification in studies where the aim is to quantify exposure to risk factors. It has implications not just for interpreting the results of case-control studies but also for the use of gait analysis as an assessment tool and outcome measure in the treatment of PFPS. Despite this, very few studies have reported the pain experienced by subjects during testing (Nadeau et al., 1997; Salsich et al., 2001) and no studies have attempted to measure the effect of pain on gait in a within-subject repeated measures design.

9.1.2 Pilot data

9.1.2.1 Overview

The problem of capturing the representative gait of a subject with PFPS rather than pain-affected gait was highlighted in some pilot data. These data were collected from 3 male subjects with PFPS. The subjects were diagnosed with PFPS according to the criteria in Table 6.2 and were undergoing a 6-month home-based exercise rehabilitation program. All subjects had chronic recalcitrant pain and were medically downgraded from their military occupational role (Table 9.1). To investigate the validity of non-pain affected gait patterns in PFPS patients, subjects were instrumented with the HH model and underwent a 10 minute treadmill run to stimulate pain symptoms and assess gait alterations with increasing pain. At each two minute interval, 10 seconds of gait kinematic data were captured and subjects were asked to indicate their level of pain on a VAS scale.
9.1.2.2 Results and discussion

The injured limb of subjects with unilateral pain (subjects 2 and 3) showed less hip internal rotation, ankle eversion and tibial internal rotation (figure 9.1). Both limbs of the patient with bilateral pain (subject 1) also showed low amplitudes of these movements (for continuity these data are not presented). There was no systematic adaptation to pain as it increased during the 10 minute run (figure 9.2). These observations suggest that each subject’s gait may have been inhibited even when pain-free.

Whilst the results from this pilot work were quite notable, the data are from only 3 subjects and it is possible that subjects had these characteristics before they developed PFPS. A comparison of gait before and after the onset of PFPS is thus required to verify these adaptations. Such a pre-post pain study would provide information critical to the interpretation of findings from case-control studies of gait biomechanics in patients with PFPS.

Table 9.1. Description of each subject that participated in the pilot study of gait and PFPS.

<table>
<thead>
<tr>
<th>Subject</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>33</td>
<td>26</td>
<td>19</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.80</td>
<td>1.61</td>
<td>1.70</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>85</td>
<td>67</td>
<td>78</td>
</tr>
<tr>
<td>BMI (kg.m²)</td>
<td>26.2</td>
<td>25.8</td>
<td>27.0</td>
</tr>
<tr>
<td>Injured limb</td>
<td>Bilateral</td>
<td>Right</td>
<td>Right</td>
</tr>
<tr>
<td>Duration of Symptoms (years)</td>
<td>9</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>FIQ (Kujala et al., 1993)</td>
<td>91</td>
<td>63</td>
<td>66</td>
</tr>
<tr>
<td>Most Pain in last week (VAS)</td>
<td>25</td>
<td>50</td>
<td>37</td>
</tr>
<tr>
<td>Mean pain during test (VAS)</td>
<td>0</td>
<td>31</td>
<td>10</td>
</tr>
</tbody>
</table>
Chapter 9. Effect of PFPS on gait

Figure 9.1. Ensemble joint angle plots (°) in the injured (red) and uninjured limb (black) from the 2 subjects with unilateral pain (subjects 2 and 3). (a) hip rotation; (b) ankle eversion and (c) tibial rotation. The lines represent the mean over the entire 10 minute run (n of cycles = 18).

Figure 9.2. An exemplar plot (subject 3) of tibial rotation (°) taken at 2 minute intervals over the 10 minute run, the corresponding pain intensity is detailed in the legend. Each line represents the mean from 3 strides.
A pre-post study of treadmill running was thus set up in conjunction with the longitudinal follow up study described in Chapter 8. The baseline pre-PFPS data existed from the cohort identified in Chapter 6 and the post onset of PFPS gait data were collected when the subjects were followed up 3 years later (Chapter 8). The study was restricted to running kinematics because the earlier prospective study did not show any significant association between walking kinematics and PFPS. It was hypothesised that subjects would display modifications in the joint angle kinematics that were shown to be risk factors in the prospective study described in Chapter 6. Accordingly, reduced hip adduction, hip internal rotation and tibial internal rotation, and increased knee internal rotation was expected in the post onset of PFPS data. Given the findings from the pilot work and previous studies, it was also hypothesised that subjects would display modified ankle eversion and reduced knee flexion during stance.

9.1.3 Aim

The aim of this preliminary study was to:

- Examine the effect of PFPS on gait kinematic patterns.

9.2 Method

9.2.1 Design

This was a preliminary investigation. Kinematic data were collected at baseline on a cohort of healthy individuals and subjects were followed up at 3 years to determine occurrence of PFPS. Participants who were diagnosed with PFPS underwent a repeat gait assessment at this timepoint. The within-subject differences before and after onset of PFPS were compared to gain an indication of the effect of PFPS on gait kinematics.

9.2.2 Subject selection

Subjects were recruited into the study based on participation in a 3 year longitudinal follow-up study (Chapter 8) and a diagnosis of PFPS. Subjects were excluded if they had
any other co-morbidity that could affect their gait patterns. Eight subjects met the inclusion criteria but two were ultimately excluded due to chronic back pain and achilles tendinosis.

9.2.3 Data verification

Given the 3 year interval between the two repeat gait assessments, two data verification studies were performed to check that the gait analysis methodology still produced similar and reliable data. Two problems were envisaged, firstly, if a systematic shift occurred between the two sets of measurements over the 3 year period as a result of intra-observer measurement drift then this would erroneously be interpreted as a pain effect. Secondly, if the between-day reliability had deteriorated, incorrect conclusions could also be drawn. Thus, the verification checks were done to ensure that no intra-observer systematic bias existed between the pre and post onset of PFPS gait assessment and that the between day reliability was still acceptable. For the purpose of clarity and continuity, the data from these two studies are contained in ANNEX G, however, the following sub-sections briefly outline the two studies and main findings.

9.2.3.1 Check for systematic bias in protocol

To check for systematic bias over the data collection period, a new control group was formed at the 3-year timepoint (2005) and compared to the gait kinematics of the original control group data collected in 2002. The 2005 control group consisted of 7 healthy individuals (Table G.1, ANNEX G). These individuals were screened by a senior chartered physiotherapist for any present or previous musculo-skeletal abnormality or pathology that may have affected their normal gait patterns (Table G.2). Given the matched health status of the 2002 and 2005 control groups, the kinematic data should be similar in both groups if no systematic drift in measurement technique over the 3 year period had occurred.

The 36 variables that were generated from the 8 joint angles for the 2002 prospective data (section 6.2.5) were also created for the 2005 data and compared using independent t-tests. Even without adjusting for the inflated type one error rate caused by multiple comparisons, there were no significant differences in any of the variables between groups (Table G.3-G.5, ANNEX G). This suggests that no systematic bias had been introduced to the protocol over the time frame of the study. The results from this study are reported in ANNEX G.
9.2.3.2 *Check for reliability*

To ensure the between day reliability of the protocol was satisfactory for the post-pain data collection a reliability study was performed on 5 subjects. These subjects were selected from the 2005 control group described in ANNEX G.

Gait assessment was undertaken on two separate test days 48 hours apart. CMCs were calculated (see equations (1), (2) and (3) in section 5.6.2.1) to quantify the reliability of the waveforms, and LOA (Bland and Altman, 1986) were calculated to assess the agreement between the peak angles from each waveform. The between day reliability was deemed good and the results were generally better than the 2002 reliability study (Table G.6-G.7, ANNEX G). The full results are also provided in ANNEX G.

9.2.4 *Kinematic data collection*

The protocol and procedures used to obtain gait kinematics were identical to those used for the baseline data described in Chapters 5 and 6, and so will not be repeated here.

9.2.5 *Data reduction and analysis*

Hip adduction, hip rotation, knee flexion, knee abduction, knee internal rotation, ankle dorsiflexion, foot eversion, and tibial rotation angles were calculated over the stance phase of gait from the baseline and post injury data. The relevance of these joint angles was discussed in section 9.1 and they have been used and reported consistently in the preceding studies. For each subject the mean at baseline (pre-PFPS) was subtracted from the mean at the 3 year point (PFPS). This was used to calculate a mean ensemble difference plot which provided a continuous measure of the mean change in a joint angle over the stance phase of gait since developing PFPS. These plots were visually examined.

The joint angles where differences were hypothesised (hip adduction, hip rotation, knee rotation, ankle eversion, tibial rotation) were parameterised to five variables. The five parameters were the peak angle, angular excursion, peak velocity, angle over mid stance (35-45% stance) and velocity over midstance. These variables were described and justified in section 6.2.5. A series of paired t-tests were performed on each of the 25 variables to assess the within-subject difference since the onset of PFPS. Given the preliminary nature of this study, alpha was set at .05 and not corrected for multiple comparisons.
9.3 Results

9.3.1 Sample description

At 3 years, the mean weight of the subjects had significantly increased by approximately 5kg from baseline (p=.007, <.05), this was also reflected in the BMI (p=.004, <.05) (Table 9.2).

9.3.2 Pain and symptoms

Subjects had suffered symptoms of anterior knee pain for a mean of 2.5 years. The mean score on the FIQ was high and the mean pain on the VAS was low (Table 9.2), indicating that the symptoms of PFPS in this group were generally very mild and less severe than the PFPS group reported in Chapter 6. This was also reflected in their occupational grade, where a consultant rheumatologist deemed all subjects fully fit for military and trade duties. Most subjects were pain-free during testing and where pain was experienced it was very mild (Table 9.2).

Table 9.2. Descriptive characteristics of the study sample at baseline (pre-PFPS) and at 3 years (PFPS). Data represent the mean and standard deviation (brackets) unless stated.

<table>
<thead>
<tr>
<th></th>
<th>Pre-PFPS</th>
<th>PFPS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>19.4 (2.8)</td>
<td>22.6 (2.5)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.78 (0.1)</td>
<td>1.78 (0.1)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>73.67 (11.6)</td>
<td>78.70 (11.9)</td>
</tr>
<tr>
<td>BMI (kg.m²)</td>
<td>22.79 (2.0)</td>
<td>24.81 (2.3)</td>
</tr>
<tr>
<td>Duration of Symptoms (years)*</td>
<td>-</td>
<td>2.5 (1-2.5)</td>
</tr>
<tr>
<td>FIQ (Kujala et al., 1993)</td>
<td>-</td>
<td>90 (10)</td>
</tr>
<tr>
<td>Most Pain in last week (VAS / 100)$</td>
<td>0</td>
<td>8 (4-13)</td>
</tr>
<tr>
<td>Mean pain during test (VAS / 100)$</td>
<td>0</td>
<td>1 (0-4)</td>
</tr>
</tbody>
</table>

*median (range); $median (inter-quartile range)

9.3.3 Temporal-spatial data

There were no significant differences (p>.05) in any of the temporal-spatial data from baseline to PFPS (Table 9.3).
Table 9.3. Mean temporal-spatial data and p-values from paired t-test at baseline (pre-PFPS) and at 3 years (PFPS).

<table>
<thead>
<tr>
<th></th>
<th>pre-PFPS</th>
<th></th>
<th>PFPS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>sd</td>
<td>mean</td>
<td>sd</td>
</tr>
<tr>
<td>Cadence (Hz)</td>
<td>1.46</td>
<td>0.07</td>
<td>1.45</td>
<td>0.04</td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>1.91</td>
<td>0.09</td>
<td>1.92</td>
<td>0.06</td>
</tr>
<tr>
<td>Stance phase proportion (%)</td>
<td>35.5</td>
<td>4.6</td>
<td>33.4</td>
<td>3.5</td>
</tr>
<tr>
<td>Stance phase time (ms)</td>
<td>243</td>
<td>29</td>
<td>231</td>
<td>27</td>
</tr>
</tbody>
</table>

9.3.4 Joint angle data

Generally the data were very similar in pattern and magnitude from baseline to PFPS (figure 9.3). However, there were a few significant differences in the paired t-tests of the parameterised joint angle variables. There was significantly less hip internal rotation during midstance after PFPS onset (p<.05), however, the mean difference was only 1.4° (Table 9.4). Tibial internal rotation was also significantly less during midstance (p<.05), similarly the mean difference was small at 2° (Table 9.4). The ankle eversion velocity over midstance was significantly less after developing PFPS (figure 9.3, Table 9.4). There were no significant differences in the knee flexion or knee rotation discrete variables (Table 9.4).

Interestingly, although not statistically analysed, there was less knee extension and ankle plantarflexion during the final push off phase of stance (75-100%) after the onset of PFPS (figure 9.3).
Chapter 9. Effect of PFPS on gait

Figure 9.3. Group mean ensemble plots (n=6) for the change in joint angle kinematics over the stance phase of running after the development of PFPS (PFPS - pre PFPS). Key: Solid line: mean; broken line: ± 1 SD.
### Table 9.4. Mean differences and p-values from the paired t-tests for the gait variables. AMS = angle over midstance, VMS = velocity over midstance. The blue font indicates variables that were components of kinematic factor 1 (leg rotation), the green font represents kinematic factor 3 (hip adduction) and the red font represents kinematic factor 6 (knee rotation).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Peak Angle (°)</th>
<th>Excursion (°)</th>
<th>Peak Velocity (deg.sec⁻¹)</th>
<th>AMS (°)</th>
<th>VMS (deg.sec⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip Adduction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hip Adduction</td>
<td>-0.3</td>
<td>0.4</td>
<td>-3.8</td>
<td>-0.4</td>
<td>-0.9</td>
</tr>
<tr>
<td>AMS (°)</td>
<td>0.3</td>
<td>0.6</td>
<td>14.9</td>
<td>0.3</td>
<td>11.6</td>
</tr>
<tr>
<td>VMS (deg.sec⁻¹)</td>
<td>-1.2</td>
<td>-1.1</td>
<td>-41.0</td>
<td>-1.0</td>
<td>-30.8</td>
</tr>
<tr>
<td>Hip Rotation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hip Rotation</td>
<td>-0.6</td>
<td>-1.5</td>
<td>-30.4</td>
<td>-1.4</td>
<td>-5.7</td>
</tr>
<tr>
<td>AMS (°)</td>
<td>0.8</td>
<td>1.3</td>
<td>35.5</td>
<td>0.5</td>
<td>34.6</td>
</tr>
<tr>
<td>VMS (deg.sec⁻¹)</td>
<td>-2.5</td>
<td>-4.9</td>
<td>-121.7</td>
<td>-2.8</td>
<td>-94.6</td>
</tr>
<tr>
<td>Knee flexion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee flexion</td>
<td>0.3</td>
<td>0.3</td>
<td>-2.4</td>
<td>-2.4</td>
<td>-4.9</td>
</tr>
<tr>
<td>AMS (°)</td>
<td>1.1</td>
<td>1.8</td>
<td>23.8</td>
<td>4.3</td>
<td>58.8</td>
</tr>
<tr>
<td>VMS (deg.sec⁻¹)</td>
<td>-2.3</td>
<td>-4.3</td>
<td>-63.5</td>
<td>4.8</td>
<td>58.8</td>
</tr>
<tr>
<td>Knee Rotation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee Rotation</td>
<td>-0.4</td>
<td>-0.4</td>
<td>-24.9</td>
<td>-1.0</td>
<td>-49.8</td>
</tr>
<tr>
<td>AMS (°)</td>
<td>0.6</td>
<td>0.9</td>
<td>25.2</td>
<td>0.5</td>
<td>48.6</td>
</tr>
<tr>
<td>VMS (deg.sec⁻¹)</td>
<td>-1.1</td>
<td>-2.8</td>
<td>-89.7</td>
<td>-2.2</td>
<td>-174.8</td>
</tr>
<tr>
<td>Ankle Eversion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ankle Eversion</td>
<td>0.2</td>
<td>-0.3</td>
<td>-1.4</td>
<td>0.7</td>
<td>60.7</td>
</tr>
<tr>
<td>AMS (°)</td>
<td>0.5</td>
<td>1.0</td>
<td>18.8</td>
<td>0.8</td>
<td>17.1</td>
</tr>
<tr>
<td>VMS (deg.sec⁻¹)</td>
<td>-1.0</td>
<td>-2.8</td>
<td>-49.8</td>
<td>-1.3</td>
<td>16.7</td>
</tr>
<tr>
<td>Tibial Rotation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tibial Rotation</td>
<td>1.2</td>
<td>23.0</td>
<td>60.7</td>
<td>1.2</td>
<td>47.6</td>
</tr>
<tr>
<td>AMS (°)</td>
<td>1.2</td>
<td>93.8</td>
<td>23.0</td>
<td>1.2</td>
<td>37.1</td>
</tr>
<tr>
<td>VMS (deg.sec⁻¹)</td>
<td>-1.8</td>
<td>-218.0</td>
<td>60.7</td>
<td>-1.8</td>
<td>-47.8</td>
</tr>
</tbody>
</table>

*P < .05
9.4 Discussion

Whilst this was a preliminary study, these are the first gait data to be presented from a repeated measures study on a sample of subjects before and after onset of PFPS. In experimental studies the dose of the factor of interest is typically exaggerated to allow the effects of the factor to be detected, in this regard it was unfortunate for this study that the cohort had very mild symptoms of pain. However, in observational studies such as this, it is not possible to control for this outcome. Despite this there were some small but significant differences.

9.4.1 Temporal-spatial data

There were no significant changes in the temporal-spatial characteristics following onset of PFPS. This result agrees with two previous walking studies (Heino and Powers, 2002; Nadeau et al., 1997). A few studies did report reduced cadence and stride length (Powers et al., 1997a; 1999; Powers et al., 2002), however, these differences only occurred when subjects were allowed to adopt a preferred walking speed. Two running studies have compared the stance phase duration during running in an AKP and control group (Messier et al., 1991; Duffey et al., 2000). Results were equivocal, one study found no difference (Messier et al., 1991), which agrees with this study, while the other found a slightly longer stance phase duration in the AKP group (Duffey et al., 2000), although this difference was only 6ms. Overall, the findings from studies concerning temporal-spatial differences have been heterogeneous, this is possibly a reflection of differences in the symptom severity of the case groups and the different protocols adopted regarding speed standardisation.

9.4.2 Joint angle data

The finding of reduced hip and tibial internal rotation at midstance after developing PFPS was hypothesised. However, there is suspicion over the validity of these findings for two reasons. Firstly, the magnitude of difference was small. Secondly, whilst the data verification check for systematic bias did not show any significant differences (ANNEX G), the 2005 control cohort showed 1.5° less hip internal rotation and 1.7° less tibial internal rotation compared to the 2002 cohort (Table G.3, G.5). These differences were similar to those found in the pre-post pain data. Within subject statistical tests such as the
Chapter 9. Effect of PFPS on gait

paired t-test that was used for the pre-post data, are more powerful than between subject tests, which may explain why these two variables were not significant in the data verification check (ANNEX G). Most importantly however, is whether one can infer with confidence that these two results were true compensations due to PFPS, and unfortunately the results from the systematic bias check prevent this.

Despite this, it is still possible that these small differences were true effects. The directly conflicting findings from case-control and prospective studies, and the notable results from the pilot data described in section 9.1.2 show some consistency and plausibility for a compensation effect for these gait associations. As such, it is recommended that future work investigates the effect of PFPS on these transverse rotations of the leg using samples with a more severe symptom complex.

The finding of reduced ankle eversion velocity over midstance was more convincing. Whilst not as pronounced, this pattern agrees with the data from the pilot work, and also tentatively with one study that found reduced ankle eversion velocity over the 1st 10% of stance (Duffey et al., 2000). Since the foot acts as the interface between the lower extremity and the ground, foot movement during contact may offer some control or perceived control over the rotations of the proximal segments. It is possible that subjects adopted this strategy to limit the transverse rotations about the knee. However, as discussed further work is required to investigate this.

9.4.3 Inhibition in the absence of pain

Given that some compensation occurred (i.e. foot eversion) even though pain was virtually absent in subjects during testing, suggests that some movements may be inhibited beforehand regardless of the presence of pain. This implies that it may not be possible to capture normal kinematics in persons with PFPS. This is particularly notable given the mildness of symptoms in this cohort. The absence of symptoms during testing might also suggest that inhibition was to some extent centrally controlled which is in agreement with experimental studies that have artificially induced muscle pain with a hypertonic saline solution (Graven-Nielsen et al., 2002).
This may have implications for corrective treatment and rehabilitation, for example, if movement is inhibited despite the absence of pain, then this could hinder the restoration of normal muscle function through rehabilitation. In this sense, strategies such as biofeedback (Reid, 1992) and patellar taping (Powers et al., 1997a) may be beneficial to discourage inhibited patterns. However, further research is needed to examine the neuromuscular nature of any altered kinematics that occur as a consequence of PFPS.

These findings illustrate the problem of establishing the temporal sequence or pathway of association between a variable and an outcome using a case-control study. The results suggest that some of the significant findings from case-control designs may originate after the onset of symptoms, which clearly rules out a causative interpretation. Further, the assumption that normal movement function can be measured when pain is absent, as has been alluded to in a few articles on AKP (e.g. Duffey et al., 2000; Messier et al., 1991), may also be invalid. From a methodological perspective these findings do not lend support for the use of case-control studies to examine inherent movement characteristics as risk factors for anterior knee pain. This is a critical methodological issue because it concerns causation, yet many authors of case-control studies have not attempted to determine whether the associated variable was a precursor or successor to pain (Callaghan and Baltzopoulas, 1994; Dillon et al., 1983; Livingston and Mandigo, 2003; Powers et al., 2002) or discussed the possibility of the variable being a compensation effect (Callaghan and Baltzopoulas, 1994; Livingston and Mandigo, 2003). The literature also contains studies where the aims have differed in that they were to examine either risk factors for AKP (Callaghan and Baltzopoulas, 1994; Dillon et al., 1983; Duffey et al., 2000; Heino and Powers, 2002; Livingston and Mandigo, 2003; Messier et al., 1991; Powers et al., 2002) or compensations of AKP (Nadeau et al., 1997; Powers et al., 1997b; Powers et al., 1999) but have used the same case-control design and measured similar gait variables.

It is likely that some individuals with PFPS don’t exhibit compensatory gait strategies. Unfortunately the statistical analysis used in this study only examines for overall group differences. However, even if some subjects are able to run without pain inhibition, in a case control study the problem of classifying a subject’s gait as typical still remains.
9.4.4 Limitations

As was noted, despite the diagnosis of PFPS, this group had a high level of function and a very mild symptom complex, which may have affected the sensitivity of the study to detect differences. Further, the six subjects used in this study were recruited from the longitudinal follow up study described in Chapter 8, and as reported, there were no risk factors in their gait at baseline. Given the absence of risk factors at baseline, one may question whether any compensations in the hypothesised joint angles would be expected. Both these limitations are likely to have had an influence on the findings of this study, and as such, the sample was not ideal for studying the effects of PFPS on gait. Despite this, the study was undertaken because it was thought that if differences were found in this mild sample, then this would lend further support for the findings of the earlier reported studies into gait and PFPS.

Future research may also wish to examine the effect of PFPS on joint coordination movement variability. Case-control studies have suggested the presence of more constrained coordination patterns (Hamill et al., 1999; Heiderscheit et al., 2002). Variability was not further assessed in this study because the PFPS sample was highly functional and only minor differences were found in the joint angle data. Further, the between day reliability of capturing gait kinematics was slightly better at the 3 year data collection point (ANNEX G) and it was thought that this might have an influence on the variability data.

9.4.5 Conclusion

This study has demonstrated that even in subjects with very mild symptoms of PFPS and no presence of pain, there may be some subtle gait compensations in the movement of the foot. Additionally, one should not rule out the possibility that the transverse rotations of the hip and tibia can also be affected by subtle compensatory behaviour. However, given the limitations of these latter two findings, more work is needed to assess the magnitude and nature of movement inhibition in subjects with more severe symptoms of anterior knee pain.

It is considered that this work has applications for the design and interpretation of research into dynamic risk factors for injury, the treatment of anterior knee pain with exercise
rehabilitation and the assessment of movement dysfunction as a cause of pain in individuals with PFPS.
Chapter 10

Summary, relevance, further study and conclusions

10.1 Summary of findings

The incidence of overuse anterior knee pain over a brief 12 week exposure to a military training program was high (8.75%). Heavy smokers and individuals with a history of ankle injury were at greater risk of AKP. These associations, while explaining only a small proportion of the variance (6%) in individuals who developed AKP, were independent of lifestyle factors such as prior activity levels. Gait showed a greater association with PFPS. Factor analytic and multivariate logistic regression found 3 factors from treadmill running that explained approximately 45% of the variance in PFPS outcome. The factors comprised increased hip and tibial internal rotation, increased hip adduction and decreased knee internal rotation during stance. Inter-stride movement coordination variability also showed an association with PFPS. The group that developed PFPS had reduced movement variability in the inter-joint relationships that contained tibial rotation (factor 1). Increased variability in the inter-joint relationships that contained hip rotation and ankle eversion were also risk factors when adjusting for the first factor. Some of this variance was separate to that explained by the angular kinematic model. However, a cross-validation study in a new sample of subjects with PFPS did not produce results to support these risk factors. The null findings from this study were attributed to the mild symptoms of the PFPS group and possible case group differences.

Findings from a preliminary repeated measures study of gait kinematics before and after the development of PFPS showed some subtle alterations in gait despite the absence of symptoms during testing and the very mild case group.
Chapter 10. Conclusions and future work

10.2 The nature of the findings

Given the objective of this thesis to study risk factors, it is important to discuss the nature of the findings from this work. Establishing causation has been at the root of many scientific and medical investigations, however, statistical association does not imply causation, a point outlined and discussed in Austin Bradford-Hill’s landmark paper on epidemiology (Hill, 1965). There is no straightforward approach for establishing the nature and importance of an association, but some key philosophical points iterated in epidemiological position papers (Greenland et al., 1999; Hill, 1965; Phillips et al., 2004) are considered here.

The prospective design allowed the sequence of association to be established in that the exposure (i.e. gait kinematics) preceded the occurrence of injury, which addresses the limitations of other studies in this area. The strength of association and effect size of the gait variables were not insubstantial, and the factors had good sensitivity and specificity in predicting PFPS from individuals who remained free of lower limb overuse injury. That the gait data fitted a linear model of risk reasonably well, suggests a possible dose-response relationship. The findings were also plausible and coherent in relation to expert opinion (Tiberio, 1987; Powers, 2003a), experimental work (e.g. in-vitro studies: Lee et al., 1994; Fuchs et al., 1999) and the stress hypothesis for AKP (Heino and Powers, 2002). These points add to the strength of gait kinematics as an important risk factor, and hint to some proximity to the inciting mechanism for PFPS. However, the results were not reproduced in another sample of PFPS and so the main limitation is the consistency of the findings. Whilst this was attributed to case group differences, further cross validation work is required on different and larger samples to evaluate the role and importance of gait on AKP. Such research should also examine whether gait characteristics can distinguish AKP from other lower limb overuse injuries.

Other research is also required before a causative role of gait can be attributed. In-vivo experimental ‘cause-effect’ research is needed to examine the effect of these findings on the hypothesised stress mechanism for PFPS. Intervention studies using the randomised controlled trial are required to examine the effect of altering these risk factors on prevention and treatment. If the kinematics of gait is a causative mechanism, then in an
experimental study one would expect these risk factors to be related to increased stress, and in a randomised controlled trial one would expect relief of symptoms or prevention of AKP by altering the gait risk factors.

One should also be cautious about overemphasising results from statistical significance testing and the dichotomous evaluation of p-values (Phillips et al., 2004), effect sizes and confidence intervals are more informative. Accordingly, some of the non-significant factors found in this PhD could be important, for example, previous AKP and relative training load (aerobic fitness) showed trends in the confidence intervals, but methodological flaws such as response bias and missing data may have reduced the statistical significance.

It is recognised that this work is a first step and that causation cannot be inferred. These studies were a starting place for investigating the role of gait on anterior knee pain. Further work is required to refute or support these results and gain a greater understanding of the nature of these findings.

10.3 A possible mechanism for gait related overuse anterior knee pain

The mechanical model for AKP has been well discussed and the findings from this program of work would appear to support a mechanical contribution to certain types of AKP. In-vitro studies have demonstrated that internal rotation of the tibia (Csintalan et al., 2002) and femur (Fuchs et al., 1999; Lee et al., 1994) and an increased dynamic Q-angle (Mizuno et al., 2001) can increase the patellofemoral contact pressure through a decrease in the patellofemoral contact area. An increase in retro-patellar pressure was also observed in an in-vivo study of walking in patients with AKP, and MRI scans showed that this was due to a decrease in the contact area (Heino and Powers, 2002). However, this study was not designed to determine whether tibiofemoral movement or another contributory factor such as patellar congruency caused the decrease in contact area. Given the findings from the gait kinematic study, one may speculate that the significant factors relating to internal rotation of the lower limb may cause a decrease in patellofemoral contact area that increases the pressure behind the patella stimulating pain in a pathomechanical manner as
described in section 2.5.1. Likewise the finding of reduced inter-stride variability in tibial movement reported in Chapter 7 may also intervene in the normal mechanical functioning of the patellofemoral joint through affecting the distribution of stress through the patellofemoral joint. It is obvious from this model that any other factor that increases the contact pressure of the patellofemoral joint through either contact area or force may also be a risk factor for AKP. As such there are likely to be other biomechanical factors such as patellofemoral congruency that are important determinants of retro-patellar stress and that may also account for some of the unexplained variance in why certain individuals develop AKP.

10.4 Relevance, contribution and applications of the work

10.4.1 Clinical

Whilst this thesis provides no direct evidence to support a particular treatment approach, there are one or two wider clinical applications, and also some results that may provide a rationale for future rehabilitation research.

10.4.1.1 Joint angle kinematics

The findings from this thesis lend tentative support for correcting movement dysfunction in the treatment of PFPS. Interventions that help control hip and tibial internal rotation and hip adduction may be beneficial (e.g. exercise rehabilitation and orthoses). However, it is important to understand the cause of these movement differences since this will ultimately determine the most effective treatment strategy. A movement dysfunction could originate from a number or combination of sources. For example, strength deficits, structural abnormalities and neuromuscular control are possibilities, and all have been implicated in AKP. Whilst some research has quantified the relationship between foot morphology and foot and shank kinematics, further work is needed to examine other factors that may be associated with these gait kinematics. Gait retraining may also have applications, and is now feasible with the advances in motion analysis technology and online gait analysis (e.g. Davis, 2005).
10.4.1.2 Movement variability

The results for movement variability, while requiring further validation, may ultimately have clinical applications. Aside from the obvious transfer such as the assessment of rehabilitation progress and treatment efficacy, another hypothesis concerns the effect of variability on exercise therapeutics. For example, rehabilitation may be more beneficial when the sensorimotor system is in a more variable or transitional state. This could be achieved by varying known control parameters for variability such as the speed of movement (Deidrich and Warren, 1995).

10.4.2 Methodological and research

The phase 1 studies were developed based on the limitations in the literature. It is thought that future biomechanics research sharing similar objectives may benefit from the prospective and multivariate approach used here.

10.4.2.1 Prospective cohort studies

These were the first prospective cohort studies into lower extremity joint kinematics, inter-stride joint coordination variability and AKP. The conflicting findings from the prospective gait study reported here and published case-control studies, and the findings from the preliminary study into the effect of PFPS on gait (Chapter 9) may have implications for the use and interpretation of case-control studies in this area.

10.4.2.2 Multivariate and integrative approach

The biomechanics of anterior knee pain was the principle focus of this work. However, in recognition of the multi-factorial and interdisciplinary nature of musculoskeletal injury and the aim to explore risk factors, an attempt was made to integrate concepts outside the traditional realm of biomechanics. For example, attention was given to the clinical aspects of AKP such as the diagnosis and pathophysiology of pain. An attempt was also made to adhere to rigorous epidemiological principles in the appraisal of the literature, research design, statistical analysis and the interpretation of results. It was argued that many previous biomechanical studies of injury suffer from being overly descriptive with little consideration given to other covariates that may confound or mask true risk factors. It is felt that future research into risk factors for AKP may benefit from greater recognition of the multi-factorial nature of injury and a multivariate and evidence-based approach (Bahr
and Holm, 2003). In fact, a similar model was described by Winston et al. (1996) with reference to risk factors for pediatric trauma. The authors emphasized that the integration of biomechanics into epidemiological research is more likely to aid our understanding of the control mechanisms of injury, and have termed this approach ‘biomechanical epidemiology’. A simplified and adapted schematic of this approach is shown in figure 10.1.

Given the limited understanding gained by case-control studies, there is a requirement for sufficiently powered multivariate prospective studies into the biomechanics of injury. Whilst these can pose logistical problems and be cost prohibitive, they may ultimately enhance our understanding of injury. This may be best executed through collaborative ventures.

10.4.2.3 Covariates

The epidemiology study of potential covariates produced two variables (smoking and previous ankle injury) that may be worthwhile factors to control or adjust for in large clinical trials. The importance of controlling for exposure to activity in the design of studies into risk factors for AKP was also illustrated in this work. For example, the high incidence of new onsets of AKP from a short 12-week training program shows how AKP can be unmasked upon exposure to activity, while the interpretation of the validation study in chapter 8 was limited by the failure to control for activity levels in the design or analysis. Some cross-sectional data on activity levels in this group hinted that this may have been important but it was unable to be confirmed due to the study design.

10.4.2.4 Data reduction and factor analytic methods

The factor analytic methods may be a useful tool for future research into gait and injury, and also have other applications in gait analysis (Chau, 2001). A concern with the treatment of gait data is the slightly arbitrary disposal of large amounts data. Methods such as the PCA may reveal the underlying structure of gait and enable a more informed choice of data disposal by removing the shared or common variance in the dataset. With the unique data retained, the data can be further analysed and may have greater sensitivity for detecting abnormal gait. PCA was used in this project to reduce the number of discrete variables into their main components, but it could also be applied to partition components within the entire gait waveform (e.g. Deluzio et al., 1997). To date very few biomechanical
or gait studies have used factor analytic methods, future work may wish to examine the use of such methods in gait and injury research.

![Diagram of approach adopted for this work](image)

**Figure 10.1.** Schematic diagram of the approach adopted for this work. Adapted from and termed Biomechanical epidemiology by Winston et al. (1996)

### 10.4.2.5 Analysis
This project has emphasised and demonstrated the importance of multivariable analytical techniques in injury research. The multi-factorial nature of injury was clearly evident from the literature review. Further, the limited interpretation of the cross-validation study in chapter 8, illustrated the usefulness of adjusting for uncontrolled factors that may not be the principle focus of a study e.g. activity levels.

### 10.4.2.6 Other issues
**Gender**
The findings from this thesis are only applicable to males. Incidence rates for AKP in females are more than twice that of males (Lichota, 2003). Numerous explanations have been given for the high incidence in females but they are poorly understood. In addition to the physical and structural differences between the average male and female, there are documented differences in gait (Ferber et al., 2003; Schache et al., 2003). Females are also
exposed to postural differences caused by wearing high-heeled shoes and an adducted seating position. Hormonal fluctuations have also been associated with injury. Where feasible, future research should include male and female subjects and be sufficiently powered to control for a gender interaction.

Running versus walking
The results contained in this thesis suggest that running was a more sensitive indicator of risk of AKP than walking. To date most studies have only used walking protocols. Future work into similar young cohorts may benefit from using running as the mode of ambulation.

10.5 A future prospective cohort study into biomechanical risk factors

The requirement for a larger prospective study that includes the salient variables found in this work with additional measures of patellofemoral alignment/ tracking, was discussed in the previous chapters. Prior to such a study, it is recommended that two method development work topics are undertaken, the output from which could be implemented into the methods of a prospective trial. The rationale and suggested outline for the method development work and prospective study are briefly detailed below. It is hypothesised that a combination of patellar alignment/ kinematics and locomotion kinematics may provide a better description of the knee extensor mechanism and thus be a powerful tool for the prediction of AKP.

10.5.1 Patellofemoral motion / alignment
Patellar alignment and kinematics appear to be an important factor to consider in the development of PFPS. For example; Heino and Powers (2002) showed that persons with PFPS had a decreased contact area and higher patellofemoral contact pressure during gait while Pookarnjamorakot et al. (1998) found a mean difference in the patellar tilt angle of 6.5° between 50 AKP patients and 78 controls. To date, our knowledge of patellofemoral movement, alignment and contact patterns comes from bone pin studies, MRI scans and
studies on cadavers. The in-vivo methods are too impractical, invasive or expensive for a clinical trial. However, a non-invasive and harmless technique to measure patellar tracking over the final 20° are of knee extension has recently been reported (Lin et al., 2003). It involves the use of a patella clamp and traditional motion analysis techniques. This technique could be developed to provide an indication of patellofemoral motion and alignment and be of use for a large prospective study.

10.5.2 Measurement of hip and tibial rotation

Results from studies presented in this thesis (Chapters 6, 7, 9), in-vitro studies (Fuchs et al., 1999; Lee et al., 1994), empirical studies on hip rotation strength (Ireland et al., 2003), gait studies on AKP, (Cudderford and Yack, 2000; Dillon et al., 1983) and MRI studies on femoral rotation (Sikorski, 1979; Powers et al., 2003b) demonstrate that hip and tibial rotation are critical variables for normal patellofemoral function. Unfortunately, these joint angles are also those with the most concern regarding validity and reliability.

Six DOF kinematic models are now widely used in gait analysis. Capozzo et al. (1995) described a technique involving clusters of markers termed the CAST (Calibrated Anatomical Systems Technique) method, which offers a more mathematically complete description of movement and does not suffer from the error propagation problem that afflicts the 3 DOF HH model used in this project. Functional approaches to determining the hip joint centre have also been published (Schwartz and Rozumalski, 2005), which may minimise the between-day offset errors. These approaches may be more sensitive and powerful to use in a clinical trial than the 3DOF method. Some experimental work is recommended to verify and implement an appropriate marker set-up and compare with the 3DOF data.

10.5.3 A future prospective cohort study into risk factors

The output from the work strands outlined in 10.4.1 and 10.4.2 could be implemented into the methodology of a clinical prospective cohort study to examine risk factors for PFPS. The significant variables from this PhD could also be included as predictors. It is also recommended that such a study use a military training population because of the high incidence of AKP and homogeneity of exposure to extrinsic factors. Given the large number of predictor variables and the requirement for a mixed gender study, it is important
to ensure that such a study is sufficiently powered to examine these factors using multivariate analysis, in addition to obtaining a large case group that is more clinically representative of the PFPS population.

10.5.3.1 Aims of prospective study

The aims of such a study would be to:

- Examine the association between patellofemoral alignment and AKP
- Cross-validate findings on lower extremity kinematics and movement variability
- Develop a predictive model for anterior knee pain

10.6 Conclusions

This project has provided some evidence to suggest that lower extremity kinematics may be an important risk factor for certain types of anterior knee pain. A large epidemiological study also produced two variables that may be worth considering as covariates in future clinical studies into AKP. The contrasting findings with case-control studies and the subtle compensation effects that were found in a preliminary repeated measures study in a group with mild symptoms of PFPS also suggests that one should be cautious about inferring risk factors from case-control studies of gait and PFPS. The phase I studies contained in this thesis were explorative and hypothesis generating. Future research should attempt to cross-validate these findings in larger samples of PFPS, combine other potentially salient factors such as patellofemoral alignment and explore the nature of these factors. It is hoped that this will increase our understanding of the risk factors for overuse anterior knee pain and ultimately benefit the prevention, treatment and assessment of this condition.
ANNEX A. Epidemiology Questionnaire

AKP STUDY ENTRY QUESTIONNAIRE

Please complete Sections A, B and C and give to the Doctor when you are seen. The answers that you give will be used solely by the researcher undertaking this study and will not affect any part of your training or treatment at ATR Pirbright, please be honest with your answers.

A. YOUR DETAILS

<table>
<thead>
<tr>
<th>Full Name</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Service no</td>
<td></td>
</tr>
<tr>
<td>Date of Birth</td>
<td></td>
</tr>
<tr>
<td>Sex (circle)</td>
<td>M F</td>
</tr>
<tr>
<td>Ethnic Origin (circle)</td>
<td>Afro-Caribbean White Asian Chinese African Fijian Other</td>
</tr>
<tr>
<td>Cap Badge</td>
<td>Guards REME RLC RA</td>
</tr>
</tbody>
</table>

B. SOCIAL HISTORY

1. Do you smoke or did you previously smoke? (circle) YES NO

If yes - how many cigarettes per day? .......... per day

And how long have you been smoking? .......... years

When did you give up smoking (if applicable)? ........

2. Do you drink alcohol? (circle) YES NO

If yes - how many per week

- Pints ..........
- Shorts ..........
- Glasses of wine ..........

C. TRAINING HISTORY

3. Before you joined the army...

(a) Did you do any strength training? YES NO

If yes - how many sessions per week .......... sessions

how long was each session .......... hrs .......... mins

how long have you been doing this training .......... months

(b) Did you do any running? (circle) YES NO

If yes - how many miles at any one time .......... miles

how many times per week ..........

how long have you been doing this training .......... months

THANK YOU FOR TAKING TIME TO COMPLETE THIS QUESTIONNAIRE.
THE NEXT SECTION IS FOR YOUR DOCTOR TO FILL IN.
## Medical History:

### Previous Knee Injuries (please circle)

<table>
<thead>
<tr>
<th>ACL</th>
<th>OTHER LIGAMENTS</th>
<th>PATELLA</th>
<th>ANTERIOR KNEE PAIN</th>
<th>OTHER</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Knee Operations  
Y  N

Please specify.............................................................

### Previous Ankle Injuries (please circle)

<table>
<thead>
<tr>
<th>ACHILLES TENDON</th>
<th>RECURRENT INVERSION SPRAINS</th>
<th>OTHER</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Ankle Operations  
Y  N

### Previous lower leg fractures – (please circle)

<table>
<thead>
<tr>
<th>TIBIA</th>
<th>FIBULA</th>
<th>FEMUR</th>
<th>PATELLAR</th>
<th>OTHER</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Previous stress fractures - (please circle)

<table>
<thead>
<tr>
<th>FOOT</th>
<th>TIBIA</th>
<th>FEMUR</th>
<th>PELVIS</th>
<th>OTHER</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Annex A. Epidemiology questionnaire
ANNEX B. Motion capture system check

B.1 Introduction

A small analysis was done to determine the accuracy of the motion capture system in determining the distance between two points of a known linear distance when moved randomly around the measurement volume. The aim of this system check was to assess the accuracy of the 3D motion capture system (Qualisys Medical, AB) over the measurement volume used for the studies described in Chapters 5-9.

B.2 Method

The system was calibrated using the dynamic wand technique and direct linear transformation. The wand consists of two markers mounted on a rigid rod, where the distance between the centroids of the two markers is 301.4mm (provided by Qualisys Medical, AB).

The wand was moved around the measurement volume and the data captured. The three-dimensional coordinates of this trial were calculated using the earlier calibration sequence. The measurement volume shown in figure 1 approximates the boundaries of coordinates that were taken during the gait studies (Chapters 5-9).

The 3D coordinates were used to calculate the linear distance between the two points at three approximate locations within the measurement volume (figure D.1). Two locations were at the perimeter of the measurement volume and one at the centre. For each location, the distance was calculated for 5 frames of data. This was compared to the known actual distance of the wand to gain an appreciation for how the camera residuals given in the calibration report translate to actual measurement accuracy throughout the measurement volume. As was stated in section 5.2.1, only calibration sequences that had average camera residuals less than 1.5mm were used for the main study.

Figure B.1. A schematic view of the approximate boundaries that encompassed a subject’s gait, and the three approximate areas where the data were checked for accuracy.
Annex B. Motion capture system check

The following equation was used to calculate the wand length from the measured coordinates:

\[ L = \sqrt{(x_1-x_2)^2 + (y_1-y_2)^2 + (z_1 - z_2)^2} \]

Where \( L \) is the length of the wand, \([x_1, y_1, z_1]\) are the three dimensional co-ordinates of wand marker 1 and \([x_2, y_2, z_2]\) are the three dimensional co-ordinates of marker 2.

B.3 Results and conclusion

The absolute error of the system in determining the known length of the wand (301.4mm) over different areas of the measurement volume ranged from 0.09 to 1.91mm (0.02 to 0.65%) (Table D.1). Whilst there was slight variation in the accuracy over the 3 different areas, the errors in each area were small and considered excellent. Instrumental errors are thus unlikely to have an effect on the outcome of the studies contained in this PhD.

### Table B.1. Wand location and estimated length vs actual length (mm).

<table>
<thead>
<tr>
<th>Area</th>
<th>Marker 1 ([X_1,Y_1,Z_1]) (mm)</th>
<th>Marker 2 ([X_2,Y_2,Z_2]) (mm)</th>
<th>Estimated wand length (mm)</th>
<th>Diff. from actual (301.4mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>[-108.34, -15.95, 993.46]</td>
<td>[-342.42, 93.15, 836.62]</td>
<td>302.16</td>
<td>0.76</td>
</tr>
<tr>
<td></td>
<td>[-91.91, -16.87, 999.20]</td>
<td>[-334.90, 84.94, 855.86]</td>
<td>301.31</td>
<td>-0.09</td>
</tr>
<tr>
<td></td>
<td>[-74.92, -17.64, 1004.32]</td>
<td>[326.24, 84.63, 875.42]</td>
<td>300.39</td>
<td>-1.01</td>
</tr>
<tr>
<td>2</td>
<td>[960.59, 29.14, 382.75]</td>
<td>[830.98, 241.01, 211.88]</td>
<td>301.21</td>
<td>-0.19</td>
</tr>
<tr>
<td></td>
<td>[951.93, 26.31, 379.71]</td>
<td>[824.22, 241.07, 211.08]</td>
<td>301.21</td>
<td>-0.19</td>
</tr>
<tr>
<td></td>
<td>[943.08, 23.65, 376.82]</td>
<td>[817.13, 241.10, 210.39]</td>
<td>301.22</td>
<td>-0.18</td>
</tr>
<tr>
<td></td>
<td>[934.05, 21.14, 374.11]</td>
<td>[809.72, 241.09, 209.82]</td>
<td>301.22</td>
<td>-0.18</td>
</tr>
<tr>
<td></td>
<td>[924.86, 18.77, 371.59]</td>
<td>[802.02, 241.03, 209.38]</td>
<td>301.22</td>
<td>-0.18</td>
</tr>
<tr>
<td>3</td>
<td>[444.81, 199.79, 268.97]</td>
<td>[408.82, 451.78, 429.38]</td>
<td>300.88</td>
<td>-0.52</td>
</tr>
<tr>
<td>(middle)</td>
<td>[435.76, 192.70, 261.99]</td>
<td>[401.75, 447.06, 418.89]</td>
<td>300.79</td>
<td>-0.61</td>
</tr>
<tr>
<td></td>
<td>[426.39, 185.64, 255.07]</td>
<td>[394.55, 442.22, 408.57]</td>
<td>300.69</td>
<td>-0.71</td>
</tr>
<tr>
<td></td>
<td>[416.64, 178.56, 248.25]</td>
<td>[387.23, 437.27, 398.42]</td>
<td>300.58</td>
<td>-0.82</td>
</tr>
<tr>
<td></td>
<td>[406.49, 171.44, 241.56]</td>
<td>[379.80, 432.20, 388.46]</td>
<td>300.47</td>
<td>-0.93</td>
</tr>
</tbody>
</table>

*see figure B.1
ANNEX C. AKP Clinical Examination Proforma

Name: ___________________________  Service No: ___________________________
Date of Referral: ___________________________  Days since onset of pain: ___________

History:
- Precipitating event: .............................................................................................................
- Involved Knees: Left / Right / Both ..................................................................................
- Duration of Symptoms: ..........................................................................................................
- Function: Pain on running? Yes / No....How long for? .........................................................
  Pain walking? Yes / No....How long for? ..............................................................................
  Pain ascending and descending stairs? Yes / No.................................................................
- Knee swelling? Yes / No........................................................................................................
- True locking? Yes / No...........................................................................................................
- Giving way? Yes / No............................................................................................................
- Present activity level? .............................................................................................................
- Imaging? X ray / MRI / Arthroscopy ....................................................................................
- Previous surgery/injury to knee? ............................................................................................

Past Medical History:

Examination:

<table>
<thead>
<tr>
<th>Knee</th>
<th>Left</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>ROM</td>
<td>Flex:</td>
<td>Ext:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Effusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patella apprehension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patella tenderness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patella Igt tenderness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lachman test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior draw</td>
<td></td>
<td></td>
</tr>
<tr>
<td>McMurray's test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Collateral ligaments</td>
<td>Med 0: 20:</td>
<td>Med 0: 20:</td>
</tr>
<tr>
<td>Lat 0: 20:</td>
<td>Lat 0: 20:</td>
<td></td>
</tr>
<tr>
<td>Joint line tenderness</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

DIAGNOSIS: 
KNEE: BOTH / LEFT / RIGHT

SIGNATURE: ___________________________
DATE: ___________________________
ANNEX D. Fundamentals of principle components analysis

The purpose of this annex is to provide some background information for a practical understanding of principle components analysis (PCA) and outline the key points in the execution of a PCA. It is hoped that this will give the non-user of PCA the information to understand and interpret the results contained in Chapters 6-8. However, this is not a definitive reference to PCA and where further detail is required, referral to the three main sources that were used to compile this ANNEX is recommended (Chau, 2001; Field, 2005; Tabachnick and Fiddell, 2001).

D.1 PCA versus factor analysis

A quick comment should be made on the distinction between factor analysis and principle components analysis because the term factor analysis has unhelpfully and incorrectly been used synonymously with PCA in many research papers and textbooks. Essentially factor analysis derives a mathematical model and extracts the factors from the model, whereas a PCA merely determines the linear variates in the entire observed data. Philosophically, factor analysis thus assumes that the factors cause the observed variables whereas the PCA is based on the variables causing the factors. For this reason factor analysis is typically used to assess, confirm or develop a theory, and PCA is used and recommended when the structure of the data is unknown and the analysis is exploratory. However, there should be little difference in the solutions generated from a factor analysis or PCA as was demonstrated in an extensive literature review (Guadagnoli and Velicer, 1988, cited by Field 2005). Lastly, some authors have preferred to term the extracted factors from a PCA as ‘components’ rather than ‘factors’, the term ‘factor’ has been used in this thesis.

D.2 Overview and Applications

PCA is statistical method that explains the variability in a number of measured variables in terms of a fewer number of factors or components. The factors reflect the patterns of correlations among the measured variables (e.g. see Table D.1). Scores for each of these factors can be calculated as a linear combination of the original variables, where variables that are influential on a particular factor have a heavier weighting. A factor (F_i) can thus be represented by the equation:

\[ F_i = b_{i1} X_1 + b_{i2} X_2 + \ldots + b_{in} X_n \]  

(1)

where \( b \) are the factor loading coefficients for each of the observed variables \( X_1, \ldots, X_n \). The loading coefficient is indicative of the amount of variance in variable \( X \) that is captured by factor \( F_i \).

The main applications of PCA are to examine the underlying structure of a dataset and to reduce the number of variables. PCA is thus primarily an explorative tool. Despite its limited use in biomechanics, it has direct relevance to the analysis of human movement data where time series data are typically parameterised and represented by summary statistics. In explorative studies such as those contained in this thesis, this creates a large number of variables which can violate the use of more powerful and more appropriate multivariate statistical techniques such as logistic regression (see section 6.2.7.1 for examples of the limitations for the use of regression). In the absence of any theoretical justification for the removal variables from the data set (which should be made when designing the research), the analysis would likely consist of a series of univariate hypothesis tests. This has statistical limitations such as an inflated type 1 error rate, and provides little insight into the structure of the data and how this may affect the relationship of interest. Hence the use of factor analysis as a means of establishing the data structure and reducing the number of variables prior to a multivariate analysis.
Annex D. Principle components analysis

D.3 Theoretical basis

PCA consists of a number of mathematical steps. The main steps are briefly outlined below.

The correlation matrix \((R)\) of the measured variables is the primary matrix which is manipulated to undertake a PCA. The final PCA model, in terms of the number of factors extracted and the variables that load heavily on each factor, should relate back to the pattern of correlations in the original \(R\). This is illustrated in Table D.1 which shows the correlation matrix for the variables that had high loadings on factor 1 (leg rotation) and 3 (hip adduction) of the run kinematic data presented in Chapter 6. The highlighted areas clearly distinguish these two factors by the size of the correlation coefficients.

<table>
<thead>
<tr>
<th></th>
<th>HAPA</th>
<th>HAEXC</th>
<th>HAPV</th>
<th>HRAMS</th>
<th>TRPA</th>
<th>TREXC</th>
<th>TRPV</th>
<th>TRAMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>HAPA</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>HAEXC</td>
<td>0.70</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>HAPV</td>
<td>0.70</td>
<td>0.90</td>
<td>1.00</td>
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<tr>
<td>HRAMS</td>
<td>0.06</td>
<td>0.09</td>
<td>0.14</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TRPA</td>
<td>0.03</td>
<td>0.10</td>
<td>-0.02</td>
<td>-0.60</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TREXC</td>
<td>0.14</td>
<td>0.00</td>
<td>0.11</td>
<td>0.50</td>
<td>-0.82</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TRPV</td>
<td>-0.17</td>
<td>0.02</td>
<td>-0.10</td>
<td>-0.48</td>
<td>0.83</td>
<td>-0.99</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>TRAMS</td>
<td>0.18</td>
<td>0.21</td>
<td>0.13</td>
<td>-0.61</td>
<td>0.83</td>
<td>-0.68</td>
<td>0.63</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Mathematically, PCA is based on finding the eigenvectors of the correlation matrix of the observed variables and calculating the respective eigenvalues. The process (eigendecomposition) effectively reorganises the variance in \(R\) into \(n\) factors, where \(n\) equals the number of measured variables. The mathematics of calculating the eigenvectors mean that most of the variance in the original variables is typically contained in the first few factors, with the largest variance in factor 1 descending to the \(n\)th factor. The eigenvalues indicate the amount of variance contained within each eigenvector, and are used to determine how many factors to extract. Because most of the variance in the data set is typically partitioned onto the first few factors, it is typically only these that are considered important and interpreted.

The statistical relevance of eigenvectors and eigenvalues is easier to conceptualise geometrically using a bivariate example. Taking the most important two variables of factor 4 (knee flexion) from the kinematic run data (see Table 6.1 and 6.6), the red lines in figure D.1 can be seen as a reorganised representation of the variance contained in these two variables. These two lines actually represent the eigenvectors. The eigenvectors are used to calculate the factor loading matrix and hence can be seen as the factors of these two variables. The first factor (F1) contains nearly 70% of the variance of the original dataset. The eigenvalues can be visualised as the length of the red lines, as such a comparison of the eigenvalues provides an indication of the amount of variance captured in each factor. It is also worth noting that the eigenvectors are orthogonal and independent. This is because factor 2 was calculated on the basis of explaining the maximum variance from the residual variance matrix that remained after factor 1 had been calculated, in this way the factors are ordered (in terms of the amount of explained variance) and orthogonal. In this very simple example, it is clear that one factor, containing approximately 70% of the variance in
the observed data, could be used as a more parsimonious representation of the two knee flexion variables.

![Image](image.png)

**Figure D.1.** A geometric illustration of eigenvectors and eigenvalues. The red lines represent the eigenvectors of the variables KF-PV and KF-EXC (see Table 6.1 and 6.6). The length of the red lines represent the eigenvalues and hence amount of observed variance explained by each eigenvector.

The next step is to calculate the factor loading matrix. This represents the correlations between variables and factors and is found by multiplying the eigenvector matrix by the root of the eigenvalue matrix. The factor loading matrix and corresponding eigenvalues are used to determine how many factors to extract.

The factor loading matrix is then rotated through multiplication of a transformation matrix consisting of a matrix of sines and cosines of an optimum angle ($\Psi$). The aim is to find an optimal solution where the factor loadings are maximised on the important variables of a factor (i.e. variables with a high loading in the factor loading matrix) and minimised on the least important variables. This is illustrated graphically in the factor plot in figure D.2 which shows a schematic example of an orthogonal rotation using a hypothetical example from the gait data in Chapter 6. Here the factor loadings for the variables are plotted on the factor classification axes. By rotating the factor axes through ($\Psi$), the loadings for the variables are maximised on one factor and minimised on the other, thus improving the fit of the factor model to the data.

This was a very brief overview of the key matrices and principles behind the mathematics of PCA. Further details on the mathematics underpinning PCA, including a description of all the matrices and the matrix algebra, are contained in Tabachnick and Fiddell (2001).
Annex D. Principle components analysis

Figure D.2. Geometric representation of an orthogonal factor rotation, the horizontal axis can be seen as factor 1 (leg rotation) and the vertical axis factor 3 (hip adduction) for the run data from the PCA described in Table 6.6. The factor loadings for each variable are plotted. The orthogonal rotation of the factor classification axis through $\Psi$ maximises the loadings onto the factor to which the variables relate to most.

D.4 PCA procedure

D.4.1 Factorability

Before undertaking a PCA it is necessary to ensure that the data are suitable for factoring and meet a few statistical assumptions. The key points regarding the factorability of the data are outlined below.

D.4.1.1 Normality

The statistical criteria which are used to determine how many factors to extract are based on the assumption of normality, therefore PCA is enhanced when all the data follow a normal distribution. The kolmogorov-smirnov test can be used to test this.

D.4.1.2 Linearity

Because the PCA is based on the correlation matrix, it models a linear relationship among the observed variables. Therefore the relationships between variables should also be linear. This can be assessed using scatter plots.

D.4.1.3 Shared variance

Given that the aim of PCA is to find clusters of correlations or variables measuring similar constructs, there is a requirement for some shared variance in the correlation matrix in order for a PCA to be appropriate. If all the variables are unique with no shared variance then the data are unsuitable for PCA and reduction. If this is the case the correlation matrix will resemble an identity matrix i.e. all variables correlating only with themselves. Bartlett’s test of sphericity tests the null hypothesis that $R$ is an identity matrix, and so should be <.05 to indicate some shared variance.
The Kaiser-Meyer-Olkin statistic (KMO), which represents the spread of patterns of correlations in R for a variable, where 1 equals a compact distribution and 0 equates to a diffuse distribution, is an important measure to check the suitability of the variables for factoring. Values >.7 are considered good, consideration should be given to discarding variables with values <.5. One should also examine the average KMO, here a value >.6 is considered satisfactory.

D.4.1.4 Multicollinearity
Multicollinearity can be problematic to a PCA because it makes it impossible to determine the unique contribution of a variable to a factor. Therefore, variables that correlate very highly (r > .9) should be eliminated.

D.4.1.5 Sample size
The stability and reliability of a PCA model is dependent on the sample size. The communalities ($h^2$) of factors after extraction represent the amount of variance explained by the extracted factors for each of the variables, and can be used to assess sampling adequacy. These take values between 0 and 1, where 1 equates to the entire variance being explained by the factors. When the sample size is <100, it is recommended that these values are >.6 for all variables.

D.4.2 Factor extraction
An important decision in PCA is the choice of how many factors to extract. There are a few statistical criteria that can be used to make this decision and ensure adequate factor extraction, these are explained below.

D.4.2.1 Eigenvalues and scree plots
As mentioned earlier, the eigenvalues indicate the amount of explained variance from the original data that is contained in each factor. As such, they can be used to determine how many factors to extract from the PCA. Kaiser (1960, cited by Field 2000) recommends extracting all factors with eigenvalues >1. This rule is valid when the communalities are >.7. It is also recommended that the scree plot is checked as a guide. The scree plot consists of the factors ordered along the x-axis and their corresponding eigenvalues plotted on the y-axis (e.g. see figure 6.6). The results should closely correspond with Catell's (1966 cited by Field 2005) criteria where the number of factors extracted is taken from the point of inflexion on the scree plot. The logic behind this is that the factors on the flat part of the curve with similar eigenvalues are likely to be explaining random variance and thus are not real factors. However, the scree plot tends to only be accurate to within one or two factors.

D.4.2.2 Total explained variance from factors
If an orthogonal rotation has been used to optimise the factor loadings (there are two main types of factor rotation - orthogonal and oblique, these are explained below) then it is possible to examine the total amount of original variance explained by the extracted factors. Since the intention is to retain as much of the variance from the original dataset, one should look for values greater than 70%.

D.4.2.3 Reproduced correlation matrix
The correlation matrix can be reproduced using the new factor model and rotated factor loadings. This can be compared with the original R and residuals calculated to determine the adequacy of fit of the model. It is recommended that the differences between the observed and model correlation coefficients are small and that more than 50% of the residuals are less than .05.
D.4.3 Choice of rotation

D.4.3.1 Orthogonal versus oblique
The premise behind factor rotation was shown in figure D.2. Figure D.2 actually demonstrates an orthogonal rotation meaning that the two factors are independent and unrelated. This type of rotation ensures that the factors stay uncorrelated. However, it is also possible to perform an oblique rotation. An oblique rotation allows any of the factors to correlate and is used if the factor structure is suspected to be related in any way, for example for theoretical reasons.

To determine whether the oblique rotation fits the observed data better than an orthogonal rotation, it is necessary to undertake a PCA using an oblique rotation, calculate the factor scores for each subject on each factor and assess whether these factors are correlated using the factor correlation matrix. If any of the factors show a correlation (values of $r > .3$ are considered important), it indicates that the oblique rotation may be a better fit of the data. This should also be supported by an underlying theoretical explanation for why the factors could be related.

D.4.3.2 Rotation technique
There are some different techniques for rotating the factors that depend on the type of rotation performed (i.e. orthogonal or oblique). For an orthogonal rotation the most common technique is the varimax method. This attempts to load the maximum variance on each factor. However, most statisticians also check the quartimax rotation, unlike the varimax rotation this works across the rows of the factor loading matrix and attempts to maximise the variance on each variable. The results of both rotations normally closely agree as was demonstrated in section 6.3.5.3.

For oblique rotations the main group of techniques for rotation is called direct oblimin. However, the analyst sets the degree of correlation permitted between factors by specifying a value called delta ($\delta$). Delta values less than zero allow less correlated solutions while values greater than zero permit more correlation between factors. The degree of correlation that results for a given $\delta$ ultimately depends on the dataset. The default value for $\delta$ of zero is recommended (when delta is zero it is termed a directquartimin rotation), however, if the calculated factors are highly correlated and barely distinguishable then this suggests that too many factors have been extracted and thus one should revisit the factor extraction.

Whilst the orthogonal rotation is typically used by researchers because it relies on less decision making by the operator thus removing some of the potential observer bias, it has been argued that truly orthogonal rotations rarely occur in human sciences and as such the oblique rotation should always be chosen (Field, 2005)

D.4.4 Interpretation of factors

Whilst the factor scores are a linear composite of all the observed variables, in reality, it is often a small cluster of variables that contribute most to that score. As such it is a useful process to label these factors based on the variables with the greatest weighting. This also serves as a check of the suitability and appropriateness of the PCA, since the factor labels should maintain some theoretical relevance. The main matrices used to interpret the factors depend on the type of rotation that was performed, these are described below.

D.4.4.1 Rotated factor loading matrix
This matrix is interpreted if the orthogonal rotation was chosen. The squared factor loadings contained in this matrix represent the variance of the observed variable explained by each factor. Whilst previous research has used a variety of cut off criteria for rating the importance of the contribution of a variable, Comrey and Lee (1992) state that factor loadings in excess of .63 can be considered as good. Additionally, it is also recommended that loadings $<.4$ are suppressed in the matrix to ease interpretation.
D.4.4.2 Pattern and structure matrix
These are the key matrices from an oblique rotation. The pattern matrix is similar to the orthogonally rotated factor loading matrix, it represents the unique correlation between a factor and a variable. The structure matrix is a product of the pattern matrix and the correlations amongst factors, so it represents the relationship between a factor and a variable plus the overlapping variance amongst factors (i.e. shared variance). The pattern matrix is thus the first matrix to use to interpret and label factors. However, it is recommended that the structure matrix is also reported and examined for any variables that may have been hidden in the pattern matrix due to correlations between factors. This masking of variables due to factor correlation is illustrated in the structure matrix contained in Table 6.5 & 6.6 of Chapter 6.

D.4.5 Calculation of factor scores
Before factor scores are calculated, the measured data are standardised to a z-score. This means that they have a mean of zero and a standard deviation of one. Equation (1) is then applied to the standardised data, where the factor score coefficients are found by a product of the inverse of the original correlation matrix and the rotated factor loading matrix. This results in a standardised factor score with a mean of zero and standard deviation of one.

The reduced data, comprised of a smaller number of factors represented by standardised factor scores, can then be used for subsequent analysis.
ANNEX E. Description of the vector coding method

E.1 Vector coding description and calculation

Vector coding provides a continuous measure of joint coordination. It is based on the orientation of the vector between two points on an angle-angle diagram. This orientation (\(\gamma\)) is measured in degrees from the right horizontal and takes values of 0 to 360° (figure E.1). Values of 45 and 225° indicate that the two segments are moving synchronously, values of 0 and 180° indicate that the proximal segment is moving and the distal segment is fixed and values of 90 and 270° indicate that the distal segment is moving and the proximal segment is fixed (figure E.1).

![Angle-angle diagram of knee flexion (x-axis) and tibial rotation (y-axis) defining the vector coding method. FC = foot contact.](image)

The relative motion angle (\(\gamma\)) is a circular variable, which renders the typical standard deviation statistic inappropriate for determining the variability of these data. For example, the standard deviation of 0°, 1°, 359°, 358° is 206.7° which is misleading because these relative motion values actually indicate that there is very little coordination variability. This problem is circumvented by the use of circular statistics (Batschelet, 1981). Using circular statistics, variability is determined by first calculating the mean cosine and sine of the relative motion angle (\(\gamma\)) over a number of trials (n), this is given by (1) and (2):

\[
x = \frac{1}{n} \sum_{i=1}^{n} \cos \gamma_i \\
y = \frac{1}{n} \sum_{i=1}^{n} \sin \gamma_i
\]

From this the length of the mean vector (r) is calculated and reflects the directional concentration and variability of the data (3):

\[r = \sqrt{x^2 + y^2}\]

The information detailed here has been described by Hamill et al. (2000) and Heiderscheit et al. (2002).
E.2 Results from the PCA and logistic regression of the vector coding variability data

Table E.1. Factor loadings, communalities ($h^2$) and explained variance from the unrotated and orthogonally rotated PCA solution for the vector coding run data. Variables with loadings $>.63$ are in red and considered important to that respective factor. Loadings $<.4$ are suppressed for ease of interpretation.

<table>
<thead>
<tr>
<th>Run</th>
<th>F1</th>
<th>F2</th>
<th>F3</th>
<th>F4</th>
<th>F1</th>
<th>F2</th>
<th>F3</th>
<th>F4</th>
<th>$h^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>HRTRMEAN</td>
<td>.785</td>
<td>.648</td>
<td>.504</td>
<td>.753</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HRTRMS</td>
<td>.745</td>
<td>.485</td>
<td>.763</td>
<td>.832</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>KFTRMS</td>
<td>.697</td>
<td>.719</td>
<td>.647</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>KFHRMEAN</td>
<td>.608</td>
<td>.472</td>
<td>.734</td>
<td>.714</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>KFHRMS</td>
<td>.577</td>
<td>.479</td>
<td>.526</td>
<td>.563</td>
<td>.643</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>KFHTEN</td>
<td>.551</td>
<td>.486</td>
<td>.816</td>
<td>.686</td>
<td></td>
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<td></td>
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<tr>
<td>HATR10</td>
<td>.801</td>
<td>.863</td>
<td>.789</td>
<td></td>
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<tr>
<td>KFTR10</td>
<td>.763</td>
<td>.926</td>
<td>.865</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>HATRMEAN</td>
<td>.402</td>
<td>.576</td>
<td>.512</td>
<td>.513</td>
<td>.679</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>TRAE10</td>
<td>.471</td>
<td>.483</td>
<td>.645</td>
<td>.634</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HRTR10</td>
<td>.659</td>
<td>.664</td>
<td>.786</td>
<td>.792</td>
<td>.647</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>KAAEMEAN</td>
<td>.562</td>
<td>.503</td>
<td>.792</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>HATRMS</td>
<td>.462</td>
<td>.586</td>
<td>.828</td>
<td>.726</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>TRAEMEAN</td>
<td>.465</td>
<td>.586</td>
<td>.854</td>
<td>.807</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Factors
- F1: Hip rotation stance
- F2: Tibial rotation 10%
- F3: Tibial rotation midstance
- F4: Ankle eversion stance

% total variance: 30.0 19.2 12.8 11.7 21.6 19.5 17.9 14.5 73.5

Table E.2. Summary of the coefficients, significance tests and odds ratios for the final model produced from logistic regression for the run vector coding variability data. The final column reports the significance of the change in the model if the term is removed (LL = log likelihood).

<table>
<thead>
<tr>
<th>Run</th>
<th>$\beta$</th>
<th>se</th>
<th>p</th>
<th>exp $\beta$</th>
<th>95% CI</th>
<th>sig. of change in LL</th>
</tr>
</thead>
<tbody>
<tr>
<td>F2 Tibial rotation 10-20%</td>
<td>-2.607</td>
<td>1.326</td>
<td>.049</td>
<td>.074</td>
<td>.005</td>
<td>.991</td>
</tr>
<tr>
<td>Constant</td>
<td>-2.803</td>
<td>.992</td>
<td>.005</td>
<td>.061</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$R^2 = .32$ (Nagelkerke)
Model $\chi^2 = 8.98$ (p=.003)
Hosmer and Lemeshow Test: $\chi^2=6.95$ (p=.542)
ANNEX F. Knee pain response form

KNEE PAIN REPLY FORM

A. The following questions are to determine whether you have had any knee pain and how your knee pain occurred. Please TICK the appropriate box that corresponds to you.

A1. Have you had any knee pain since you completed Phase one of Army recruit training?  
Yes  No

If you answered Yes to the previous question, please complete the rest of the questionnaire and return using the stamped addressed envelope. If you answered No, please ignore the questions below and go to the consent and contact details part in section (C) on page 5 of this form.

A2. Did the pain occur gradually during exercise e.g., running / walking?  
Yes  No

A3. Did the pain occur after a knock/ twist or fall  
Yes  No

A4. Do you still have pain?  
Yes  No

A5. Which knee has/ had pain?  
Right  Left  Both

A6. If both which knee is causing you most pain?  
Left  Right

A7. When did the pain first occur?  
M M Y Y Y Y

B. The following questions are to find out about the level of pain that you are experiencing and how this pain affects your daily activities. For each statement, please tick the option which most corresponds to you for your present condition.

B1. Limp  
None  Slight or periodic  Constant

B2. Supporting your weight  
Full support without pain  Painful  Weight bearing impossible

B3. Walking  
Unlimited  Able to do more than 2 km (1.25 miles)  
1-2 km (0.6 - 1.25 miles)  Unable to do

B4. Stairs  
No Difficulty  Slight pain going down  Pain going up and down  Unable to do

B5. Squatting  
No difficulty  Repeated squatting painful  Painful each time  Possible when partially supporting weight  Unable to do
### Annex F. Knee pain reply form

<table>
<thead>
<tr>
<th>B6. Running</th>
<th>No difficulty</th>
<th>Pain after more than 2km (1.25 miles)</th>
<th>Slight pain from the start</th>
<th>Severe pain</th>
<th>Unable to do</th>
</tr>
</thead>
<tbody>
<tr>
<td>B7. Jumping</td>
<td>No difficulty</td>
<td>Slight difficulty</td>
<td>Constant pain</td>
<td>Unable to do</td>
<td></td>
</tr>
<tr>
<td>B8. Sitting with knees bent for a long time</td>
<td>No difficulty</td>
<td>Pain after exercise</td>
<td>Pain forces you to straighten legs</td>
<td>Constant pain</td>
<td>Unable to do</td>
</tr>
<tr>
<td>B9. Pain</td>
<td>None</td>
<td>Slight and occasional</td>
<td>Interferes with sleep</td>
<td>Occasionally severe</td>
<td>Constant and severe</td>
</tr>
<tr>
<td>B10. Swelling</td>
<td>None</td>
<td>After severe exertion</td>
<td>After daily activities</td>
<td>Every evening</td>
<td>Constant</td>
</tr>
<tr>
<td>B11. Abnormal painful kneecap movements</td>
<td>None</td>
<td>Occasionally in sports activities</td>
<td>Occasionally in daily activities</td>
<td>At least one dislocation</td>
<td>More than two dislocations</td>
</tr>
<tr>
<td>B12. Loss of muscle bulk in the thigh</td>
<td>None</td>
<td>Slight</td>
<td>Severe</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B13. Decrease in the amount you can bend your knees</td>
<td>None</td>
<td>Slight</td>
<td>Severe</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Pain - indicate your greatest level of discomfort with your knees during the past week by placing a mark on the line below.

- **No pain at all**
- **Pain as bad as it possibly could be**
C. Consent and Contact Details

I have read the subject information sheet and understand the general nature, potential risks, duration of the study and what is expected of me. I have read and understand the no fault compensation scheme provided by the Ministry of Defence, and understand that in the event of sustaining an injury, illness or death as a result of participating in this research, that I, or my dependants may enter a claim. This consent is specific to the study described in the information sheet and does not imply consent in subsequent studies. I reserve the right to withdraw from this study at any time and this will not be held against me in subsequent dealings with the Ministry of Defence.

Contact Details

If you have agreed to participate, we may wish to contact you in the next four weeks. If the contact details printed on this letter were incorrect, or you will be moving shortly, can you amend or advise below. It is particularly important that we have your correct phone number.

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Print Name:

Sign:

Date:

Tel no Work: ................................................................. Evening:

Mobile: .................................................................

Please return this form using the stamped addressed envelope, thank you for your time and cooperation.
ANNEX G. Systematic bias and reliability check: pre-post study

G.1 Background

Two studies were undertaken to ensure that valid interpretations could be made should any differences arise from the pre-post study to assess the effect of PFPS on gait patterns (see Chapter 9). The first study checked for systematic bias in the measurements over the period of the repeated pre-PFPS and PFPS gait measurements and the second study checked that the reliability was still acceptable at the time of the PFPS gait assessment. A brief description of each of these studies along with the results follows below.

G.2 Systematic bias

It was important to exclude or account for the effect of measurement bias or systematic drift over the 3-year time period of the study as a potential source of difference between the pre-PFPS and PFPS data. This was done by forming a new control group and comparing the kinematic data of this group with the control group of the original prospective cohort collected in 2002. The time points of data collection for these two control groups corresponded to the time points when the pre-PFPS and PFPS data were collected (2002 and 2005).

G.2.1 Method

G.2.1.1 Subject selection (Control group 2005)

Seven healthy subjects formed the new 2005 control group, these individuals were screened by a senior chartered physiotherapist for any present or previous musculo-skeletal abnormalities or pathologies that could affect their normal gait patterns. The screening criteria are detailed in Table G.1. These criteria were applied to ensure a robust control group with a healthy musculoskeletal status similar to the 2002 group.

Table G.1. Exclusion criteria for selecting the control group where CH = clinical history; PE = physical examination; ASIS = anterior superior iliac spine.

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous anterior knee pain</td>
<td>CH</td>
</tr>
<tr>
<td>Previous lower limb surgery</td>
<td>CH, PE</td>
</tr>
<tr>
<td>Joint effusion at the ankle or knee</td>
<td>Clarke sign</td>
</tr>
<tr>
<td>Abnormal or injured medial/ lateral collateral ligaments</td>
<td>Valgus/varus stress at 15° knee flexion</td>
</tr>
<tr>
<td>Abnormal or injured anterior/posterior cruciate ligaments</td>
<td>Lachmans, pivot shift, PCL droop</td>
</tr>
<tr>
<td>Meniscus injury</td>
<td>McMurray</td>
</tr>
<tr>
<td>Leg length discrepancy &gt;1.5cm</td>
<td>PE, ASIS to medial maleolus</td>
</tr>
<tr>
<td>Femoral anteversion</td>
<td>PE</td>
</tr>
<tr>
<td>Restricted hip, knee or ankle sagittal plane movement</td>
<td>PE</td>
</tr>
<tr>
<td>Hypermobility</td>
<td>Hypermobility in lower limb (Beighton test)</td>
</tr>
<tr>
<td>Neural pathology</td>
<td>(single leg raise)</td>
</tr>
<tr>
<td>Poor alignment and stability</td>
<td>(single leg squat)</td>
</tr>
</tbody>
</table>

G.2.1.2 Kinematic data collection

Kinematic data during treadmill running were collected and processed using the same protocol as the 2002 cohort. These are described in detail in Chapter 5.
G.2.1.3 Variables and analysis
The 36 variables that were generated from the 8 joint angles for the 2002 prospective data were also created for the 2005 data. These are detailed in Table 6.1 and figure 6.1. Independent t-tests were used to compare the 2 control groups for each variable (SPSS Inc, v10). All variables were normally distributed. The degrees of freedom were adjusted for variables with unequal variances in each group (SPSS, 1999).

G.2.2 Results and discussion
G.2.2.1 Sample description
The 2005 control group was older and slightly heavier than the 2002 group (Table G.2). However, there is no evidence or theoretical reason to suggest that gait matures from the age of 19 to 28 years. And despite the heavier body weight, the mean BMI still falls within the ideal range of 20 – 25 kg/m² (ACSM, 2005). Likewise there is no evidence to suggest that a BMI difference of this magnitude will affect gait. The mean present activity levels of the 2005 group was 10,941 MET-min per week, which suggests this was a highly active group. This provides reassurance that individuals were not asymptomatic from AKP due to low activity levels.

<table>
<thead>
<tr>
<th>Table G.2. Descriptive statistics for the 2002 and 2005 control group. Standard deviations are in brackets.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cohort Mean (sd)</td>
</tr>
<tr>
<td>N</td>
</tr>
<tr>
<td>Age (years)</td>
</tr>
<tr>
<td>Height (m)</td>
</tr>
<tr>
<td>Weight (kg)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
</tr>
</tbody>
</table>

G.2.2.2 Differences between 2002 and 2005 control group
There were no significant differences between the 2002 and 2005 control group for any of the discrete variables from the eight joint angles (Table G3-G5). This suggests that the protocol and methods were consistent over the 3 year time-period of the study and that there is no significant systematic bias that may influence the results of the main pre-post PFPS study (Chapter 9).

<table>
<thead>
<tr>
<th>Table G.3. Hip joint group mean discrete variables, difference, 95% CI for the difference and result from the independent samples t-test comparing the 2002 and 2005 control group.</th>
</tr>
</thead>
<tbody>
<tr>
<td>---</td>
</tr>
<tr>
<td>Cohort mean (sd)</td>
</tr>
<tr>
<td>Peak Angle (°)</td>
</tr>
<tr>
<td>Excursion (°)</td>
</tr>
<tr>
<td>Peak Velocity (deg/sec⁻¹)</td>
</tr>
<tr>
<td>AMS (°)</td>
</tr>
<tr>
<td>VMS (deg/sec⁻¹)</td>
</tr>
<tr>
<td>Peak Rotation</td>
</tr>
<tr>
<td>Excursion (°)</td>
</tr>
<tr>
<td>Peak Velocity (deg/sec⁻¹)</td>
</tr>
<tr>
<td>AMS (°)</td>
</tr>
<tr>
<td>VMS (deg/sec⁻¹)</td>
</tr>
</tbody>
</table>
### Table G.4. Knee joint group mean discrete variables, difference, 95% CI for the difference and result from the independent samples t-test comparing the 2002 and 2005 control group.

<table>
<thead>
<tr>
<th></th>
<th>Cohort mean (sd)</th>
<th>2002</th>
<th>2005</th>
<th>mean diff</th>
<th>95% CI diff</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td></td>
<td></td>
<td></td>
<td>lower</td>
<td>Upper</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak Angle (°)</td>
<td>40.62 (3.64)</td>
<td>39.52 (4.10)</td>
<td>1.11</td>
<td>-1.98</td>
<td>4.19</td>
<td>.47</td>
</tr>
<tr>
<td>Excursion (°)</td>
<td>21.98 (5.37)</td>
<td>24.99 (4.59)</td>
<td>-3.01</td>
<td>-7.39</td>
<td>1.37</td>
<td>.17</td>
</tr>
<tr>
<td>Peak Velocity (deg.sec⁻¹)</td>
<td>408.99 (74.93)</td>
<td>433.85 (68.01)</td>
<td>-24.86</td>
<td>-86.40</td>
<td>36.67</td>
<td>.42</td>
</tr>
<tr>
<td>Knee Abduction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak Angle (°)</td>
<td>-3.61 (1.42)</td>
<td>-3.62 (1.54)</td>
<td>0.01</td>
<td>-1.19</td>
<td>1.21</td>
<td>.98</td>
</tr>
<tr>
<td>Excursion (°)</td>
<td>8.15 (3.02)</td>
<td>7.61 (2.63)</td>
<td>0.54</td>
<td>-1.92</td>
<td>3.01</td>
<td>.66</td>
</tr>
<tr>
<td>Peak Velocity (deg.sec⁻¹)</td>
<td>-188.43 (53.86)</td>
<td>-153.87 (63.27)</td>
<td>-34.56</td>
<td>-80.56</td>
<td>11.44</td>
<td>.14</td>
</tr>
<tr>
<td>AMS (°)</td>
<td>-2.31 (1.17)</td>
<td>-2.74 (1.39)</td>
<td>0.43</td>
<td>-0.57</td>
<td>1.43</td>
<td>.39</td>
</tr>
<tr>
<td>VMS (deg.sec⁻¹)</td>
<td>0.97 (58.94)</td>
<td>5.78 (52.44)</td>
<td>-4.81</td>
<td>-53.10</td>
<td>43.48</td>
<td>.84</td>
</tr>
<tr>
<td>Knee Rotation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak Angle (°)</td>
<td>3.90 (1.75)</td>
<td>3.75 (1.22)</td>
<td>0.15</td>
<td>-1.25</td>
<td>1.55</td>
<td>.83</td>
</tr>
<tr>
<td>Excursion (°)</td>
<td>7.83 (4.22)</td>
<td>9.06 (3.02)</td>
<td>-1.24</td>
<td>-4.62</td>
<td>2.15</td>
<td>.47</td>
</tr>
<tr>
<td>Peak Velocity (deg.sec⁻¹)</td>
<td>226.03 (88.65)</td>
<td>260.60 (68.26)</td>
<td>-34.57</td>
<td>-106.14</td>
<td>36.99</td>
<td>.34</td>
</tr>
<tr>
<td>AMS (°)</td>
<td>1.25 (2.25)</td>
<td>0.59 (1.74)</td>
<td>0.65</td>
<td>-1.16</td>
<td>2.47</td>
<td>.47</td>
</tr>
<tr>
<td>VMS (deg.sec⁻¹)</td>
<td>-13.42 (75.74)</td>
<td>-53.75 (76.94)</td>
<td>40.33</td>
<td>-22.82</td>
<td>103.48</td>
<td>.20</td>
</tr>
</tbody>
</table>

### Table G.5. Ankle joint group mean discrete variables, difference, 95% CI for the difference and result from the independent samples t-test comparing the 2002 and 2005 control group.

<table>
<thead>
<tr>
<th></th>
<th>Cohort mean (sd)</th>
<th>2002</th>
<th>2005</th>
<th>mean diff</th>
<th>95% CI diff</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td></td>
<td></td>
<td></td>
<td>lower</td>
<td>Upper</td>
</tr>
<tr>
<td>Ankle Dorsi flexion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak Angle (°)</td>
<td>25.47 (3.52)</td>
<td>25.25 (4.40)</td>
<td>0.22</td>
<td>-2.82</td>
<td>3.26</td>
<td>.88</td>
</tr>
<tr>
<td>Excursion (°)</td>
<td>19.61 (4.98)</td>
<td>18.07 (2.91)</td>
<td>1.54</td>
<td>-2.40</td>
<td>5.48</td>
<td>.43</td>
</tr>
<tr>
<td>*Peak Velocity (deg.sec⁻¹)</td>
<td>329.42 (107.16)</td>
<td>273.80 (33.93)</td>
<td>55.61</td>
<td>11.19</td>
<td>100.03</td>
<td>.19</td>
</tr>
<tr>
<td>Ankle Eversion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak Angle (°)</td>
<td>-3.15 (1.21)</td>
<td>-2.88 (0.98)</td>
<td>-0.27</td>
<td>-1.26</td>
<td>0.71</td>
<td>.58</td>
</tr>
<tr>
<td>Excursion (°)</td>
<td>6.18 (2.41)</td>
<td>5.80 (1.86)</td>
<td>0.38</td>
<td>-1.57</td>
<td>2.32</td>
<td>.70</td>
</tr>
<tr>
<td>Peak Velocity (deg.sec⁻¹)</td>
<td>-154.36 (57.54)</td>
<td>-169.80 (35.03)</td>
<td>15.44</td>
<td>-30.21</td>
<td>61.10</td>
<td>.50</td>
</tr>
<tr>
<td>AMS (°)</td>
<td>-1.43 (1.18)</td>
<td>-1.56 (0.83)</td>
<td>0.12</td>
<td>-0.82</td>
<td>1.07</td>
<td>.79</td>
</tr>
<tr>
<td>VMS (deg.sec⁻¹)</td>
<td>-3.04 (48.78)</td>
<td>9.04 (40.70)</td>
<td>-12.09</td>
<td>-51.77</td>
<td>27.60</td>
<td>.54</td>
</tr>
<tr>
<td>Tibial Rotation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak Angle (°)</td>
<td>-5.96 (2.57)</td>
<td>-5.14 (1.96)</td>
<td>-0.82</td>
<td>-2.90</td>
<td>1.25</td>
<td>.43</td>
</tr>
<tr>
<td>Excursion (°)</td>
<td>10.57 (5.23)</td>
<td>9.35 (4.31)</td>
<td>1.22</td>
<td>-3.03</td>
<td>5.47</td>
<td>.57</td>
</tr>
<tr>
<td>Peak Velocity (deg.sec⁻¹)</td>
<td>-246.53 (136.35)</td>
<td>-243.87 (123.84)</td>
<td>-2.65</td>
<td>-114.64</td>
<td>109.33</td>
<td>.96</td>
</tr>
<tr>
<td>AMS (°)</td>
<td>-8.36 (2.23)</td>
<td>-2.16 (1.17)</td>
<td>-1.70</td>
<td>-3.55</td>
<td>0.15</td>
<td>.07</td>
</tr>
<tr>
<td>VMS (deg.sec⁻¹)</td>
<td>53.63 (107.33)</td>
<td>85.51 (84.99)</td>
<td>-31.88</td>
<td>-116.74</td>
<td>54.99</td>
<td>.46</td>
</tr>
</tbody>
</table>

*Unequal variances (Levene's test), variance separated and degrees of freedom adjusted (from 42 to 32.4)

(SPSS Applications guide pp109, 1999)

### G.3 Check for reliability at the 3-year data collection point: Reliability study #2

#### G.3.1 Method

Five subjects from the 2005 control cohort also underwent a repeat gait assessment (treadmill running) 48 hours after the first measurement. These data were used to calculate the between-day reliability at the 3-year data point. Coefficients of multiple correlation (CMC) were calculated to assess the reliability of the joint angle waveforms, and Bland and Altman's (1986) limits of
agreement (LOA) were calculated for the peak of each joint angle to provide a measure of absolute agreement between data over the two test days.

### G.3.2 Results

The CMCs were all ≥ .89 (Table G.6), this indicates good reliability according to recommendations by Schache et al. (2002). The LOAs were also comparable to the previous reliability study (Table G.7). Based on these results, the between-day reliability was deemed excellent.

**Table G.6.** Between-day mean adjusted CMCs and sd for treadmill running. The data had the daily mean subtraction (DMS) adjustment (see section 5.6.2.1).

<table>
<thead>
<tr>
<th>Joint angle</th>
<th>CMC Run</th>
<th>mean</th>
<th>Sd</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip</td>
<td>Add. – Abd.</td>
<td>.968</td>
<td>0.009</td>
</tr>
<tr>
<td></td>
<td>Int. – Ext. rot.</td>
<td>.914</td>
<td>0.089</td>
</tr>
<tr>
<td>Knee</td>
<td>Flex. – Ext.</td>
<td>.998</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>Add. – Abd.</td>
<td>.978</td>
<td>0.006</td>
</tr>
<tr>
<td></td>
<td>Int. – Ext. rot.</td>
<td>.920</td>
<td>0.037</td>
</tr>
<tr>
<td>Ankle</td>
<td>Dors. – Plant. Flex.</td>
<td>.996</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>Inv. – Eve.</td>
<td>.890</td>
<td>0.076</td>
</tr>
<tr>
<td>Tibia</td>
<td>Int. – Ext. rot.</td>
<td>.923</td>
<td>0.041</td>
</tr>
</tbody>
</table>

**Table G.7.** Mean peak values (sd) and between day 95% limits of agreement.

<table>
<thead>
<tr>
<th>Peak angle(°)</th>
<th>μ day 1</th>
<th>sd</th>
<th>μ day 2</th>
<th>sd</th>
<th>Mean bias (CI)</th>
<th>95% LOA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip Add.</td>
<td>4.2</td>
<td>1.3</td>
<td>4.0</td>
<td>0.8</td>
<td>0.1 (-0.6 – 0.9)</td>
<td>-1.1 – 1.3</td>
</tr>
<tr>
<td></td>
<td>2.4</td>
<td>3.3</td>
<td>2.4</td>
<td>2.2</td>
<td>0.0 (-1.2 – 1.3)</td>
<td>-2.0 – 2.1</td>
</tr>
<tr>
<td>Knee Flex.</td>
<td>41.3</td>
<td>3.4</td>
<td>41.4</td>
<td>2.8</td>
<td>-0.2 (-2.6 – 2.3)</td>
<td>-4.0 – 3.7</td>
</tr>
<tr>
<td></td>
<td>-4.0</td>
<td>1.6</td>
<td>-4.0</td>
<td>1.3</td>
<td>-0.0 (-0.7 – 0.7)</td>
<td>-1.2 – 1.1</td>
</tr>
<tr>
<td></td>
<td>3.9</td>
<td>1.2</td>
<td>3.8</td>
<td>1.2</td>
<td>0.1 (-0.5 – 0.7)</td>
<td>-0.9 – 1.1</td>
</tr>
<tr>
<td>Ankle Dors.</td>
<td>26.1</td>
<td>4.9</td>
<td>26.5</td>
<td>6.1</td>
<td>-0.4 (-2.3 – 1.5)</td>
<td>-3.4 – 2.6</td>
</tr>
<tr>
<td></td>
<td>-2.9</td>
<td>1.1</td>
<td>-2.8</td>
<td>1.2</td>
<td>-0.0 (-0.5 – 0.4)</td>
<td>-0.7 – 0.6</td>
</tr>
<tr>
<td></td>
<td>-4.8</td>
<td>1.9</td>
<td>-4.6</td>
<td>0.7</td>
<td>-0.3 (-1.8 – 1.3)</td>
<td>-2.7 – 2.2</td>
</tr>
</tbody>
</table>

Annex G. Systematic bias and reliability
ANNEX H. Publications, communications and grants from the thesis

H.1 Papers


II. Wills AK, Ramasamy A, Ewins DJ, Etherington J. Risk factors for anterior knee pain: a prospective study of physical, social and training characteristics in male Army recruits. (In preparation)


IV. Wills AK; Ewins DJ; Etherington J. Risk factors for overuse anterior knee pain: A review. (In preparation).

H.2 Abstracts/ Slide presentation


II. Wills, AK; Ramasamy, A; Ewins, DJ. (2003). The incidence and epidemiology of overuse anterior knee pain in military recruits. Rheumatology. 42(S1): BSR 20th Meeting


H.3 Presentations/ Talks


H.4 Research grants originating from the research program

I. ATRA. The development and investigation of biomechanical indices to predict anterior knee pain in military recruits. To University of Surrey from November 2005 for 30 months £5,804.

II. ATRA. A randomised controlled trial into the effect of an exercise intervention to reduce the incidence of anterior knee pain in military recruits. To DMRC Headley Court from June 2005 for 24 months £8,000.
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