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The effects of fatigue on landing in beach volleyball: implications for patellar tendinosis

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THE EFFECTS OF FATIGUE ON LANDING IN BEACH VOLLEYBALL: IMPLICATIONS FOR PATELLAR TENDINOSIS

A thesis submitted in partial fulfilment of the requirements for the award of the degree

MASTERS OF SCIENCE (HONOURS)

from

UNIVERSITY OF WOLLONGONG

by

SUZI EDWARDS

DEPARTMENT OF BIOMEDICAL SCIENCE

2002
Declaration

The work presented in this thesis is the original work of the author except as acknowledged in the text. I hereby declare that I have not submitted any of the material presented in this thesis for a degree at this or any other institution. Copies of original data analysed in the study are held by the Department of Biomedical Science, University of Wollongong.

Suzi Edwards
Dedication

To my brother Luke, my soulmate.

Everyday you give me the faith in my abilities, and drive me to excel for both of our lives together.
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I would like to express my sincere thanks to all the following people without whose assistance this study would have not have been possible.

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Abstract

There is a high incidence of knee injuries in sports that involve excessive and repetitive loading of the body. It is these excessive and repetitive loads that have been identified as a primary risk factor in the development of the commonly occurring knee injury known as patellar tendinosis. It has been suggested that neuromuscular fatigue may interfere with the ability of the patellar tendon to sustain repetitive loads by decreasing the ability of the lower limb to function optimally and to efficiently dissipate the external loads sustained during landing, thereby increasing the risk of developing patellar tendinosis. However, there is a paucity of research examining the effects of fatigue on the landing mechanics in movement tasks known to be associated with patellar tendinosis. Therefore, the purpose of this study was to establish if the landing phase of a spike jump movement (SJM) performed from ground level and a drop jump movement (DJM) performed from a standard bench height differed with respect to landing mechanics, and if fatigue induced by repetitive standing vertical jumps altered the landing mechanics of either experimental task.

Fourteen healthy uninjured subjects (mean age = 26.5 ± 5.6 years) performed two experimental tasks, a SJM and a DJM, in a non-fatigued and a fatigued condition that involved the subject landing on their dominant limb onto a sand surface. Subjects were fatigued by performing a series of weighted standing vertical jump sets, with pre- and post-fatigue blood lactate samples taken. During each trial, the subjects’ sagittal plane motion was recorded using a Northern Digital OptoTrak Position Sensor (500 Hz), and the ground reaction forces generated at landing were recorded (1000 Hz) using a Kistler Multichannel force platform. For each subject’s dominant limb, electromyographic activity was recorded using a Noraxon Telemyo transmitter and receiver (1000 Hz) for vastus lateralis (VL), rectus femoris (RF), vastus medialis (VM), biceps femoris (BF), semitendinosus (ST) and medial gastrocnemius (MG). Time synchronisation of the kinetic, kinematic and electromyographic data was performed using Northern Digital OptoTrak Data Acquisition Unit II.
Analysis of all the variables showed that the two movements, the SJM and the DJM, displayed distinct landing mechanics. That is, during the DJM, subjects generated a significantly higher peak resultant ground reaction force ($F_R$), a shorter time to the peak $F_R$, and a faster rate of loading of the ground reaction forces during landing than when performing a SJM. Furthermore, subjects displayed significantly different segmental motion and alignment during landing between the two experimental tasks. That is, during a DJM the subjects displayed a significantly higher peak vertical jump height, less knee joint flexion, a higher knee joint angular velocity, and a more vertically aligned tibia both at IC and at the time of the peak $F_R$ compared to when performing a SJM. The subjects also displayed less tibial angular displacement during landing, more plantar flexion at the ankle joint at the time of the peak $F_R$, a higher ankle joint angular velocity at the time of the peak $F_R$, a more horizontally aligned foot relative to the ground at IC, and a greater foot angular velocity at the time of the peak $F_R$ during a DJM compared to a SJM. Furthermore, the subjects exhibited significantly longer muscle burst durations for RF, VM, MG and BF; an earlier muscle burst onset time relative to IC for RF and VM; an earlier peak VL activity relative to IC; a later peak muscle activity relative to the time of the peak $F_R$ for RF and VM; and a later muscle burst offset time relative to IC for RF and VM. Despite these between-task differences, the muscle burst intensity displayed during landing did not significantly differ between the two movement tasks.

Based on the between-task comparisons it was concluded that the DJM and the SJM were significantly different from each other with respect to the ground reaction forces generated at landing, lower limb motion and alignment during landing in the sagittal plane, and the synchrony of the lower limb muscle activation patterns. Therefore, removing the take-off component from the SJM to isolate the landing phase for research purposes, as in the DJM, is not valid as the two movement tasks involve distinct landing mechanics.

In terms of fatigue effects, the subjects were truly fatigued following the fatigue protocol, as indicated by a significant decrease in their standing vertical jump height and an increase in their post-fatigue blood lactate concentration. Despite this fatigue there was no significant change in the ground reaction forces generated at landing. However,
fatigue significantly altered the segmental motion and alignment with the subjects displaying a significantly lower knee joint angular velocity at IC and a more vertically aligned tibia both at IC and at the time of the peak FR compared to when non-fatigued. Many of the fatigue effects, however, were task specific. For example, the subjects displayed a higher foot angular velocity at the time of the peak FR during a DJM, a higher ankle joint angular velocity at the time of the peak FR during a DJM, less ankle joint dorsiflexion at the time of the peak FR during a SJM, and a more vertically aligned tibia both at IC and at the time of the peak FR during a SJM in a fatigued compared to a non-fatigued condition. Furthermore, in the SJM when fatigued, the subjects displayed a significant decline in their jumping performance, evident by displaying a lower peak vertical height of the greater trochanter compared to when non-fatigued. However, only minor changes were observed in the synchrony of the muscle activation patterns with a later muscle burst peak activity to IC for RF and VM and a later MG muscle burst peak activity relative to the time of the peak FR.

It was concluded that fatigue induced by a series of standing vertical jumps did not significantly alter the ground reaction forces, or the duration or activation times of lower limb muscles at landing. However, fatigue did alter lower limb motion and alignment, and the synchrony of the lower limb muscle activation patterns displayed by the subjects during landing although most of these were task specific. Of particular importance, was the significant decline in the peak vertical jump height attained by the subjects in the SJM when fatigued. This has direct implications in Beach Volleyball, both during competition and during training, in which players who repetitively perform a SJM may become fatigued, resulting in a decrease in their SJM performance. Furthermore, the decline in the subject's peak vertical jump height may decrease the time available to position their segments approximately to control the deceleration of landing and dissipate the same high impact loads that are experienced when not fatigued. As a result, this may expose the joints of the lower limb, predominantly those superior to the ankle joint such as the knee, to increased loads. How these changes to the lower limb motion and alignment and the synchrony of lower limb muscle activation patterns during landing following fatigue influence patellar tendon loading requires further investigation.
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Chapter 1
The Problem

1.1. INTRODUCTION

Beach Volleyball was first played in the 1920s in Santa Monica, California, United States of America (USA), as a family game in which teams of six played against each other following rules similar to those of Indoor Volleyball. In the 1930s, it gradually developed into the two-person game characteristic of the sport today with the first Beach Volleyball tournament held in 1947 (FIVB, 2002; http://www.fivb.ch/EN/TheGame/). Since its introduction into the Atlanta Olympic Games in 1996, Beach Volleyball has increased in popularity with over 150 National Volleyball Federations currently administering Beach Volleyball activities, over 2,000 professional Beach Volleyball players worldwide and a World Tour offering $US3.66 million in prize money (FIVB, 2002; http://www.fivb.ch/EN/TheGame/). Although proving a highly popular recreational and competitive pursuit, one potential negative consequence of this increased participation in Beach Volleyball may be a concurrent increase in the number of injuries sustained in the sport, particularly overuse injuries such as patellar tendinosis which are also prevalent in Indoor Volleyball (Schafle et al., 1990; see Section 2.5). For example, it has been reported that 30 to 40% of elite Indoor Volleyball players have suffered from patellar tendinosis sometime in their playing careers (Ferretti et al., 1990; Kujala et al., 1989).

Although there is limited research pertaining to the specific factors that cause patellar tendinosis in volleyball, it is acknowledged that there are two general categories of risk factors, intrinsic and extrinsic risk factors. Intrinsic risk factors are considered those factors from within the body (Barker, 1997), or factors related to an individual’s physical characteristics and/or psychological traits (Lysens, 1991). Examples of intrinsic risk factors for patellar tendinosis include patellofemoral malalignment, leg length discrepancy, foot type, various lower limb malalignments, and muscular strength imbalances (see Section 2.5.2.). Extrinsic risk factors are those factors arising from outside the body (Barker, 1997) or factors that relate to the type of sports activity, the manner in which the sport is practiced, environmental conditions or equipment used (Barker, 1997). Examples of extrinsic risk factors for patellar tendinosis include
excessive external loading of the body, the landing surface type and training errors (see Section 2.5.3.). Extrinsic risk factors are the focus of this thesis as they are thought to be easier to modify than intrinsic risk factors and therefore may provide information upon which to devise effective injury prevention strategies for patellar tendinosis.

The main extrinsic risk factor associated with patellar tendinosis is considered to be excessive loading of the body during movements involving repetitive jumping*. The potential for injury during such movements is created by the large forces and moments applied to the lower limbs during the impact of landing (McNitt-Gray, 1991) and has been demonstrated by a relationship between high landing forces and lower extremity injury (Dufek & Bates, 1991; Hewett et al., 1999). These high forces generated during the impact of landing can be absorbed by materials external to the body, such as the landing surface, or internally by deformation of soft tissue structures such as ligaments and tendons and/or by contracting muscles surrounding the joint(s) (Radin, 1986). As the major structure connecting the quadriceps muscles to the leg, the patellar tendon must transmit the often high forces developed by contraction of the quadriceps muscles (see Section 2.4.) across the patellofemoral joint to produce knee joint motion with minimal deformation or energy loss, and to stabilise the patellofemoral joint (Kirkendall & Garrett, 1997; Martin et al., 1998). In landing after a jump, the patellar tendon may sustain forces as high as 8,000 N, compared to relative lower values of approximately 500 N in activities such as walking (Stanish et al., 1986). A consequence of these large loads placed on the knee joint during activities involving repetitive jumping, such as in volleyball, is the development of injuries such as patellar tendinosis (Cook et al., 2000; Harries, 1994).

A second extrinsic risk factor linked to lower extremity injury is neuromuscular fatigue (Coutts, 1982; Nyland et al., 1994; Wojtys et al., 1996). Neuromuscular fatigue can be defined as an acute impairment in performance, accompanied by an increased perceived sense of effort and an inability to produce the desired force (Bigland-Ritchie, 1981; Enoka & Stuart, 1992). There is no single mechanism of fatigue, but rather a series of

*(Colosimo & Bassett, 1990; Cook, Khan, Kiss, & Griffiths, 2000; Eifert-Mangine et al., 1992; Ferretti, 1986; Ferretti et al., 1983; Griffiths & Selesnick, 1998; Lian et al., 1996; Martens et al., 1982; Powell et al., 1998; Roels et al., 1978; Romeo & Larson, 1999; Sanchis-Alfonso et al., 2001).
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acute effects, either central or peripheral in origin within the human body, that impair motor performance (see Section 2.5.4.5.; Avela & Komi, 1998; Bigland-Ritchie, 1981; Bigland-Ritchie et al., 1995; Enoka, 1995; Enoka & Stuart, 1992; Pinniger, 1996). The decrease in muscle performance evident in landing tasks as a consequence of fatigue is thought to be partially a result of less efficient use of elastic energy (Avela & Komi, 1998). Furthermore, fatigued muscle is less able to absorb repetitive shock or stress (Kannus & Natri, 1997). As the patellar tendon already sustains high loads during repetitive jumping activities, a decreased ability of the lower limb muscles to absorb the shock, as a result of fatigue, may contribute to the development of patellar tendinosis by increasing the repetitive shock or stress transferred to patellar tendon during landing.

Fatigue can also alter the lower limb muscle activation patterns displayed at landing by causing a variety of responses including later muscle burst onsets relative to foot-ground contact (Arendt-Nielsen, 1991; Nyland et al., 1994), increased (Hortobagyi, 1991) and/or decreased muscle burst duration (Arendt-Nielsen, 1991), and increased muscle activation levels (Strojnik & Komi, 1998). If these altered lower limb muscle activation patterns reduce the ability of the muscle to efficiently dissipate forces generated at landing, the patellar tendon may again be predisposed to injury when fatigued due to the need for the tendon to withstand the excessive and repetitive forces generated. However, only one study was located which had investigated the effects of fatigue on synchrony of the lower limb muscles during a Beach Volleyball landing task performed on a sand surface (Edwards et al., 2001). Edwards et al. (2001) found that, irrespective of the fatigue condition, players generated the same vertical ground reaction forces at landing when they performed a modified drop jump movement. Despite generating the same impact forces at landing the players displayed different muscle recruitment patterns when fatigued compared to when non-fatigued. For example, the players displayed a delay in peak rectus femoris muscle activity when fatigued. The authors speculated this finding may indicate that players may be less efficient in using their knee extensors to control deceleration of their body mass during landing and, in turn, create a higher patellar tendon force when fatigued, thereby increasing the risk of developing patellar tendinosis (Edwards et al., 2001). Although the results of the study by Edwards et al. (2001) confirmed that fatigue can affect muscle recruitment strategies during a drop jump movement, the task investigated was performed by subjects stepping
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from a constant bench height. That is, the movement did not include a take-off component. Therefore, it is not known if fatigue would directly alter the landing component of a typical volleyball movement, such as a spike jump, where the peak height above the ground is not held constant (that is, the players take-off from ground level). Further investigation is therefore warranted to investigate the effects of fatigue on landing performance during a typical Beach Volleyball movement in which the peak jump height is not held constant so that we can gain a better understanding of the relationship among fatigue, lower limb landing mechanics and possible extrinsic factors that contribute to developing patellar tendinosis.

1.2. STATEMENT OF THE PROBLEM

The purpose of this study was twofold:

(1) to establish if the landing phase of a drop jump movement performed from a bench and a spike jump movement performed from ground level differed with respect to landing mechanics; and

(2) if fatigue induced by repetitive standing vertical jumps altered the landing mechanics of either a drop jump movement or a spike jump movement.

1.3. SIGNIFICANCE OF THE STUDY

There is a high incidence of patellar tendinosis in sports that involve repetitive jumping (see Section 2.5.3.1.). Knowledge of intrinsic and extrinsic risk factors that increase the risk of patellar tendinosis is very limited, particularly with respect to the effects of fatigue. How fatigue affects the coordination of lower limb musculature, peak resultant ground reaction forces and the kinematics displayed during landing in Beach Volleyball is unknown. The findings of this study will therefore assist in expanding our understanding of the etiology of patellar tendinosis and will have immediate application in establishing a basis upon which to develop prevention strategies for this prevalent injury.
1.4. RESEARCH HYPOTHESES

Based on previous literature it was hypothesized that:

1. The two experimental tasks, the drop jump movement and the spike jump movement, would not significantly differ from each other with respect to:
   a. the ground reaction forces generated at landing;
   b. lower limb alignment and motion during landing in the sagittal plane; and
   c. synchrony of the lower limb muscle activation patterns and muscle burst intensity displayed during landing.

2. Fatigue induced by a series of standing vertical jumps would result in the following for both the drop jump movement and the spike jump movement:
   a. no change in the ground reaction forces generated at landing;
   b. changes in the lower limb alignment and motion during landing in the sagittal plane; and
   c. alterations to the lower limb muscle activation patterns, accompanied by increases in muscle burst intensity displayed during landing.

1.5. ASSUMPTIONS

It was assumed that any variation in lower limb mechanics displayed by the subjects at landing was a result of the experimental tasks being performed or fatigue and not to some other extraneous variable.

1.6. LIMITATIONS

It is acknowledged that the following factors may limit the results of this study:

1. The sample was not a truly random sample of the wider population, as the study was restricted to subjects who volunteered to participate.

2. Subjects may have varied their normal landing action as a result of performing the experimental tasks in front of the “special” camera and in a laboratory environment, despite being familiarised with the experimental procedure.

3. Despite currently being active, the present fitness and training status may have varied between the volleyball players who were recruited as subjects.
This was not expected to affect the level of fatigue obtained by each subject, as this was individually standardised.

1.7. DELIMITATIONS

The following factors delimited the study:

(1) The results of this study may not be applicable to other populations (for example, patellar tendinosis patients) or to other sporting activities due to the sample group being restricted by age group, healthy lower limb status and sporting activity.

(2) As this study analysed only the two-dimensional motion of the lower limb, any motion outside of the sagittal plane of progression was not analysed.

The methodology adopted for the study contained other limitations and delimitations that may have also influenced the results of the study. These include errors inherent in the raw kinematic, electromyography and force data and other factors which are described in Chapter 3.
Chapter 2

Literature Review

2.1. INTRODUCTION

In a wide variety of sports, such as Indoor Volleyball, there has been an increase in the number of overuse knee injuries that has been associated with the repetitive and excessive loads sustained by the lower limb, particularly the overuse injury of patellar tendinosis (Aagaard & Jorgensen, 1996). Despite the prevalence of this injury, there is limited literature on risk factors for patellar tendinosis* and even less literature investigating injuries in Beach Volleyball (Aagaard et al., 1997; Briner & Ely, 1999; Frey et al., 1996). Before focusing on the experimental procedures within this thesis, it is necessary to gain a general understanding of the sport of Beach Volleyball, the structure and function of the knee joint and the patellar tendon, injuries associated with these structures, and methods to investigate potential risk factors for developing patellar tendinosis in Beach Volleyball. Therefore, literature pertaining to the following topics was reviewed and presented in this chapter:

(1) Beach Volleyball versus Indoor Volleyball;
(2) Injuries in Beach and Indoor Volleyball;
(3) The Extensor Mechanism; and
(4) Patellar Tendinosis: Risk Factors Associated and the Relevance of Fatigue.

2.2. BEACH VOLLEYBALL VERSUS INDOOR VOLLEYBALL

In 1895 volleyball was invented in the USA by William Morgan as a recreational game for male exercise and sport classes that was less violent and intensive for older individuals compared to sports such as basketball (http://www.fivb.ch). From this origin, Beach Volleyball was developed, initially based on the rules and regulations of Indoor Volleyball. Today, however, the rules and regulations of Beach Volleyball

*(Bergstrom et al., 2001; Blazina et al., 1973; Cook, Khan, Kiss, & Griffiths, 2000a; Cook, Khan, Kiss, Purdam et al., 2000b; Cook, Khan, Maffulli, et al., 2000c; Duri & Aichroth, 1995; Duri et al., 1999; Ferretti, 1986; Ferretti et al., 1983; Ferretti et al., 1984; Johnson, 1996; Lian, Engebretsen et al., 1996a; Martens et al., 1982; Panni et al., 2001; Richards et al., 1996; Roels et al., 1978; Sanchis-Alfonso et al., 2001; Witvrouw et al., 2001).
differ to its indoor counterpart. For example, Beach Volleyball is played on a sand surface, whereas Indoor Volleyball is played on a sprung wooden floor or concrete surface. Furthermore, whereas there are six players per team in the indoor version of the sport, there are only two players per team in Beach Volleyball. As the dimensions of the court for the two sports are identical, the lesser numbers of players per team in Beach Volleyball demand that players require broader all-round skills, greater stamina, strength and concentration than their Indoor Volleyball counterparts (Schutz, 1999).

Despite obvious differences in the demands of the two volleyball codes, previous research pertaining to volleyball has been restricted to examining characteristics only of Indoor Volleyball movement skills. These studies have investigated the spike jump (Adrian & Laughlin, 1983; Cisar & Corbelli, 1989; Coleman et al., 1993; Coutts, 1982; Huang et al., 1998, 1999; Weiki & Dangre, 1985), the block jump (Adrian & Laughlin, 1983; Dufek & Zhang, 1996; Stacoff et al., 1988), the jump set (Tant & Greene, 1993) and the serve (Depra et al., 1998). The spike jump is of particular importance to both volleyball codes as it is a difficult offensive skill that is vital to the success of a team. It involves a highly integrated and coordinated movement pattern consisting of an approach, jump, attack and recovery phase (Cisar & Corbelli, 1989). A more detailed description of the spike jump movement is provided by Cisar & Corbelli (1989) and Coleman et al. (1993). Even though these movements are also performed in Beach Volleyball, the nature of the sand surface and the lower number of team players may change the way the movement skills are executed when compared to Indoor Volleyball. However, only one study was located that investigated the biomechanics of a Beach Volleyball movement skill performed on a sand surface (Edwards et al., 2001; see Section 1.1.). Therefore, further research pertaining to biomechanical characteristics of Beach Volleyball skills is warranted, particularly skills such as the spike jump movement which is of particular importance to the outcome of a game (Cisar & Corbelli, 1989).
2.3. INJURIES IN BEACH AND INDOOR VOLLEYBALL

Similar to research pertaining to volleyball movement skills, injuries occurring in Indoor Volleyball have been extensively investigated. In contrast, only three studies were located which have investigated injuries occurring in Beach Volleyball (Aagaard et al., 1997; Briner & Ely, 1999; Frey et al., 1996). Aagaard et al. (1997) recorded injury profiles of 295 volleyball players during the 1993 Beach Volleyball season and the following 1993-94 Indoor Volleyball season. Equal numbers of men and women completed two identical questionnaires with both elite and recreational players being represented. Twenty-four injuries were reported in Beach Volleyball, with 286 injuries recorded in Indoor Volleyball, representing an incidence of 4.9 and 4.2 injuries per 1000 hours, respectively. The most frequent injuries in volleyball overall were acute injuries involving the ankle and finger, and overuse injuries involving the knee and shoulder (see Figure 2-1).

![Figure 2-1: Number of injuries incurred in Beach Volleyball and Indoor Volleyball by anatomical site and type (adapted from Aagaard et al., 1997, p. 219).](image-url)
Indoor and Beach Volleyball injury patterns differ (see Figure 2-2). That is, in Beach Volleyball most of the injuries occur during field defence and spiking (Coleman et al., 1993; Huang et al., 1998, 1999), with shoulder overuse injuries as the most frequently occurring injury. In Indoor Volleyball, most of the injuries occur during blocking and spiking, resulting most frequently in acute finger and ankle injuries (see Figure 2-3). These differences in the anatomical site of injury between the two volleyball codes probably reflect differences in the types of play characteristic of each sport. For example, a Beach Volleyball player is required to perform more serves and spikes per game than their Indoor Volleyball counterpart. That is, despite the same size court in both codes, a Beach Volleyball player must cover a larger area of the court as there are only two players per team compared to six players per Indoor Volleyball team (see Section 2.2.). This increased repetition of skills requiring upper limb motion in Beach Volleyball may account for the greater incidence of shoulder injuries. Fewer blocks in Beach Volleyball may account for the lower number of finger injuries in the sport relative to Indoor Volleyball whereas the less frequent occurrence of acute ankle injuries in Beach Volleyball may reflect the smaller chance of a collision and/or landing on another player’s foot due to the lower number of players taking the court compared to Indoor Volleyball. The sand surface characteristic of Beach Volleyball also allows a longer reaction time to absorb the impact forces generated during landing than the less compliant Indoor Volleyball surfaces, thus allowing for more correction possibilities of the lower limb (Aagaard et al., 1997). However, the compliant sand surface has also lead to an injury unique to Beach Volleyball called hyper-plantar flexion injuries (sand toe; Frey et al., 1996).
Figure 2-2: Percentage of injuries in Beach Volleyball compared to Indoor Volleyball at four major anatomical sites; *$p = 0.004$; **$p < 0.05$ (adapted from Aagaard et al., 1997, p. 219).

Figure 2-3: Percentage of injuries in Beach Volleyball compared to Indoor Volleyball in the largest three causes of injuries with reference to play event; *$p < 0.001$; **not significant $p = 0.057$ (adapted from Aagaard et al., 1997, p. 219).
Despite these between-sport differences in injury patterns, elite and recreational players in both volleyball codes incur a similar percentage of knee injuries, particularly overuse knee injuries (Aagaard et al., 1997). For example, Schafle et al. (1990) reported that in Indoor Volleyball, two-thirds of all the knee injuries observed were overuse and degenerative problems as opposed to sprains. Overuse injuries are those injuries thought to have been caused by repetitive microtrauma associated with activities such as jumping and running (Cowan, 1996).

In volleyball, overuse knee injuries predominately occur during spiking, with the rate of overuse injuries increasing from 16% to 47% over the past 10 years (Aagaard & Jorgensen, 1996). The main overuse knee injury incurred by volleyball players is patellar tendinosis (see Section 2.5.). It has been reported that 27% to 65% of Indoor Volleyball players have suffered from patellar tendinosis (Ferretti et al., 1990; Kujala et al., 1989). In a study of 42 athletes with patellar tendinosis, 50% of the athletes were volleyball players (Panni et al., 2001). Interestingly, the front row players had three times the injury rate compared to the back row players, with the middle-hitter/middle-blocker and the strong-side hitter having the highest rate of injury (Schafle et al., 1990). Briner & Ely (1999) noted that players were more likely to present with patellar tendinosis after jump training with 50% of all injuries occurring during jump training. As plyometric and other forms of jump training are used frequently in both Indoor and Beach Volleyball, overuse knee injuries may be attributed to the high frequency of jumping in the sport during both competition and practice.

As these injuries sustained by volleyball players can be debilitating physically, emotionally and financially, further research is required to examine which specific factors in repetitive jumping contribute to overuse knee injuries in Beach Volleyball. However, before investigating potential factors that may contribute to the development of patellar tendinosis, a review of the structure and function of the anatomical components most involved, that is, components of the extensor mechanism, is necessary.
2.4. KNEE EXTENSOR MECHANISM

The knee extensor mechanism is comprised of the quadriceps muscles, quadriceps tendon, patella, patellar retinaculum, and patellar tendon (Nicholas et al., 1984). The following subsections describe how these components act together to achieve efficient patellofemoral joint function.

2.4.1. Anatomy of the Patella

The patella is a triangular sesamoid bone that develops within the quadriceps muscle-tendon unit (see Figure 2-4; Grelsamer & Klein, 1998; Harries, 1994; Safran et al., 1998). The superior third surface of the patella is rough and receives the insertion of quadriceps tendon, with the quadriceps tendon blending into the patellar tendon. Continuing distally over the anterior patella surface, the patellar tendon forms the deep fascia, covering and attaching to the patella. The distal third is incarcerated within the patellar tendon (Harries, 1994). A further detailed description of the anatomy of the patella is provided by Safran et al. (1998) and Basso et al. (2001).

The patella performs a number of important functions. It protects the femoral condyles (Eifert-Mangine et al., 1992; Hungerford & Barry, 1979; Safran et al., 1998), the trochlea (Eifert-Mangine et al., 1992; Hungerford & Barry, 1979) and the patellar tendon from friction (Nicholas et al., 1984), and the knee joint as a whole from trauma (Renström et al., 1994). It also provides a low-friction articular surface to increase efficiency of the extensor mechanism glide, and to sustain high compressive loads (Hungerford & Barry, 1979; Nicholas et al., 1984).

In addition to its protective role, the primary function of the patella is to act as a lever, enhancing the mechanical efficiency of extensor mechanism* (see Figure 2-5) by increasing the lever arm of the extensor mechanism (Buff et al., 1988; Grelsamer & Klein, 1998; Harries, 1994; Renström et al., 1994; Safran et al., 1998; Yamaguchi & Zajac, 1989). With increasing knee joint flexion, there is also an increase in the area of contact between the patella and the femur in order to more effectively distribute

compressive stresses on the femur (Hungerford & Barry, 1979). Thus, the patella, by functioning as a lever, allows flexion and extension of the knee to occur with a decreased quadriceps force (Grelsamer & Klein, 1998).

(A)

![Anterior surface of patella](image)

(B)

![Posterior surface of patella](image)

**Figure 2-4:** Right patella (A) anterior surface and (B) posterior surface (from Martini & Timmons, 1997, p. 195).

The patella can also redirect the force generated by the quadriceps muscles and, in turn, change the magnitude of the torque (Grelsamer & Klein, 1998). The ratio between the quadriceps tendon force and the patellar tendon force has been found to be a function of knee angle (Buff et al., 1988; Goldstein et al., 1986; Grelsamer & Klein, 1998). This is
due to the change in the length of the moment arms of the active quadriceps force and the resistive patellar tendon force relative to the contact point between the patella and trochlea (Grelsamer & Klein, 1998; Kellis & Baltzopoulos, 1999b; Yamaguchi & Zajac, 1989). This enhances transmission of the quadriceps force to the patellar tendon, an essential element of the knee joint mechanism.

Figure 2-5: The mechanical role of the patella: The patella increases the moment arm of the extensor mechanism, that is, the distance between the vector of applied force and the knee’s instant centre of rotation (from Browner, 1998, p. 2084).

A complex interaction of the quadriceps muscles, the patellofemoral ligaments, the trochlea geometry, and the quadriceps angle (see Section 2.5.2.5.), guide the tracking pattern of the patella (Grelsamer & Weinstein, 2001; Heegaard et al., 1994). At full extension, the patella lies slightly lateral and just proximal to the opening of the trochlea (Grelsamer & Weinstein, 2001; Hungerford & Barry, 1979; Nicholas et al., 1984) and follows a path down the trochlea (Grelsamer & Weinstein, 2001). The patella shifts laterally and rotates medially during knee flexion, with different patella tilt directions (Heegaard et al., 1994; Mizuno et al., 2001). For a more detailed description of patella tracking during knee motion see Grelsamer & Weinstein (2001). The patella forms only part of the extensor mechanism. Therefore, the structure and function of the quadriceps tendon and patellar tendon, components of the extensor mechanism that guide the patella, need to be reviewed to assist in better understanding the function of the extensor mechanism and its role in developing patellar tendinosis.
2.4.2. Structure and Function of the Patellar Tendon and Quadriceps Tendon

2.4.2.1. Material Properties of Tendons

Tendons link muscle(s) to bone, transmitting forces developed by muscle contractions across joint(s) to produce joint motion or to stabilise the joint (Martin et al., 1998). They also act to absorb energy (Martin et al., 1998), to transmit forces with minimal deformation or energy loss (Kirkendall & Garrett, 1997), as well as providing a proprioceptive function through mechanoreceptors contained within them.

Tendons consist of closely packed collagen fibres aligned parallel to each other. They are primarily composed of Type I collagen (approximately of 90%) with a small percentage of Type III collagen (Kirkendall & Garrett, 1997; O'Brien, 1997). As collagen can only elongate approximately 4% before failure, the extensibility of tendons is gained by elastin, which is able to lengthen up to 70% (O'Brien, 1997). The ground substance in the tendons gives the tendon its viscoelastic properties (Kirkendall & Garrett, 1997; O'Brien, 1997) as well as lubrication and spacing between the fibres that are important for efficient gliding and cross-tissue interactions (O'Brien, 1997). As a consequence of its viscoelastic property, tendons exhibit stress relaxation under constant deformation, creep under constant load, and hysteresis with repeated cycles of loading (Kumar, 2001; Lucas et al., 1999; Martin et al., 1998). Tendons also exhibit crimp which allows longitudinal elongation without fibrous damage during initial tension and acts as a shock absorber along the length of the tissue (O'Brien, 1997). Between 90% to 96% per cycle of the elastic strain energy generated when tendons are stretched can be recovered by the tendon (Lucas et al., 1999). During repeated loading and unloading of a tendon, such as during repeated jumping, the strain within a tendon increases for a given constant stress level, the higher the strain rate the stiffer the tendon (Lucas et al., 1999). The stiffness and ultimate stress of a tendon is also affected by skeletal maturity and age (Lucas et al., 1999).

Metabolism of tendons is relatively slow, having a lower oxygen consumption compared to skeletal muscle, but the tendon's metabolism is increased in response to injury and exercise (O’Brien, 1997). In the event of an injury, even though there is an increase in the tendon’s metabolism, the time to repair the damage sustained may take longer relative to other tissues, due to the slower metabolism and lower oxygen
consumption of the tendon. Furthermore, compromised blood supply occurs at sites of friction, torsion and compression (O'Brien, 1997). Evidence has emerged that, due to mechanical irritation, there is hypervascularity secondary to low grade inflammation that may prevent tissue hypoxia in tendons (O'Brien, 1997). Due to the poorer recovery of tendons and the longer time for tendons to repair themselves relative to other tissue, tendons can often become injured particularly with repetitive use as there is insufficient time for repair.

2.4.2.2. The Quadriceps Tendon
The common quadriceps tendon is a blending of the tissue extending from rectus femoris, vastus lateralis, vastus intermedialis and vastus medialis (Nicholas et al., 1984; Norkin & Levangie, 1983), inserting on the superior border of the patella, to provide proximal support to the patella (Safran et al., 1998). The fibres of the quadriceps tendon continue distally from the superior border of the patella tendon over the anterior patella surface, blending into the patellar tendon (Norkin & Levangie, 1983).

2.4.2.3. The Patellar Tendon
The patellar tendon, or patellar ligament* as it sometimes referred to, is a continuation of the quadriceps muscle tendon which extends from the apex of the patella and continues distally over the anterior surface of the tibia to the proximal aspect of the tibial tuberosity (Norkin & Levangie, 1983; Rasch, 1989). Components of the patellar tendon known as the lateral and medial retinaculum act to reinforce the patella (Nicholas et al., 1984). The lateral retinaculum is made of superficial and deep transverse layers, connecting the iliotibial band on the lateral side of the knee (Safran et al., 1998). The medial retinaculum includes the patellofemoral ligament and is composed of tendinous tissue supporting the patella and helping to prevent lateral dislocation (Safran et al., 1998). Blood is supplied to the patellar tendon by the extraosseous arterial ring that branches from the geniculate arteries on the anterior surface and the intraosseous arterial ring from the mid-patella vessels and polar vessels

*Technically, soft tissue structures joining bone to bone are termed "ligaments". However, the structure connecting the patella to the tibia is typically referred to as the patellar tendon. Even though this is not a true tendon but a ligament, it is referred to as a tendon as it is an extension of the quadriceps tendon that connects the quadriceps muscles to the tibial tuberosity via the patella.
from the extraosseous vascular ring (Browner, 1998). Further anatomical details of the patellar tendon are described by Browner (1998), Harries (1994) and Safran et al. (1998).

2.4.3. Muscles Associated with the Extensor Mechanism

The quadriceps muscles provide the main motive force for the extensor mechanism, and include the rectus femoris, vastus lateralis, vastus medialis and vastus intermedialis (see Section 3.3.4.). The rectus femoris, vastus lateralis and vastus medialis are the superficial muscles of the extensor mechanism (Nicholas et al., 1984) that act together to extend the leg at the knee joint (Tortora & Grabowski, 1993). Although all of the quadriceps muscles insert on the patella (see Section 2.4.2.2.), each of the four components arise from different origins resulting in different functions.

Originating on the anterior inferior iliac spine, the rectus femoris crosses both the hip and knee joint, acting as a biarticular muscle with the primary functions of hip joint flexion and knee joint extension (Basmajian & De Luca, 1985; Batman & Van Capelle, 1994). Vastus intermedialis originates from the anterior and lateral surfaces of the body of the femur to cause extension of the knee joint (Tortora & Grabowski, 1993). Vastus lateralis arises from the greater trochanter and linea aspera of the femur causing knee joint extension (Tortora & Grabowski, 1993). Vastus medialis originates from the linea aspera of the femur to cause extension of the knee joint (Tortora & Grabowski, 1993) and to assist in preventing lateral dislocation of the patella near the end of knee joint extension (Basmajian & De Luca, 1985).

Vastus medialis also acts as the main dynamic stabiliser of the extensor mechanism (Eifert-Mangine et al., 1992; Farahmand et al., 1998; Heegaard et al., 1994; Hungerford & Barry, 1979; Kirkendall & Garrett, 1997). The static stabilisers of the extensor mechanism include the contours of the femoral sulcus, geometry of the patella, thickening of the capsule, and surrounding ligamentous structures (Eifert-Mangine et al., 1992; Farahmand et al., 1998; Hungerford & Barry, 1979). In the last 30 degrees of knee extension when weight bearing, only a small amount of vastus medialis and vastus lateralis activity is evident, indicating that there is less need for muscular force to
stabilise the knee joint in this range of joint motion when weight bearing (Reynolds et al., 1983).

Antagonists to the four quadriceps muscles are the hamstring muscles. The hamstring muscles are formed by biceps femoris, semitendinosus and semimembranosus, which act primarily to flex the leg at the knee and extend the thigh at the hip (Batman & Van Capelle, 1994; Tortora & Grabowski, 1993). Originating from the ischial tuberosity and linea aspera of femur, the biceps femoris inserts on the head of the fibula and the lateral tibial condyle (Tortora & Grabowski, 1993). Semitendinosus originates from the ischial tuberosity and inserts on the proximal part of the medial surface of the tibial shaft, whereas semimembranosus also originates from the ischial tuberosity but inserts onto the medial condyle of the tibia (Tortora & Grabowski, 1993). Semitendinosus and semimembranosus also act to medially rotate the leg at the knee joint and thigh at the hip joint (Batman & Van Capelle, 1994), with the biceps femoris acting to laterally rotate these lower limb segments.

The gastrocnemius muscle originates from the lateral and medial condyles of the femur and the posterior capsule of knee and inserts on the calcaneus by way of the calcaneal (Achilles) tendon (Jenkins & Hollinshead, 1998; Norkin & Levangie, 1983; Tortora & Grabowski, 1993). Although not a direct component of the extensor mechanism, gastrocnemius is a biarticular muscle that crosses both the ankle and knee joints (Maton & Le Pellec, 2001) and therefore can influence knee joint motion. It primarily plantar flexes the foot (Jenkins & Hollinshead, 1998), but during non-weight bearing the gastrocnemius can also help to flex the leg at the knee joint (Jenkins & Hollinshead, 1998). Furthermore, when the foot is fixed during weight bearing, the gastrocnemius maintains extension of the leg as flexion at the knee joint cannot occur without dorsiflexion at the ankle joint (Jenkins & Hollinshead, 1998). During weight bearing, gastrocnemius can also cause talocalcaneonavicular inversion (varus of the calcaneus) and elevation of the longitudinal arch of the foot (Norkin & Levangie, 1983).

During landing, the muscles of the extensor mechanism and the gastrocnemius act eccentrically. That is, the muscles lengthen as they contract to actively resist the external forces applied to the muscles which exceed the force produced by these
muscles. This results in braking and storage of elastic recoil energy in the muscles (see Section 2.5.4.1.; Lindstedt et al., 2001). During the eccentric loading the muscles function as shock absorbers by lengthening to dissipate the energy absorbed and subsequently shorten to recover the stored elastic recoil potential energy (Lindstedt et al., 2001). Furthermore, during an eccentric contraction, the primary muscle group involved in the action will be opposite to the observed joint action (Batman & Van Capelle, 1994). However, during landing, rectus femoris contracts eccentrically to actively control flexion at the knee joint to prevent the knee collapsing as the body decelerates. Therefore, the muscles of the extensor mechanism and those associated with it, are crucial in assisting in dissipating the impact forces sustained during landing, such as the forces sustained by the patellar tendon, and therefore have implications in developing overuse injuries such as patellar tendinosis.

2.5. PATELLAR TENDINOSIS

The terms Jumper’s Knee, patellar tendinitis and patellar tendinosis have all been used in the literature to describe a chronic overload lesion to the patellar tendon. Although the traditional label of tendinitis has been used to indicate a pathology of the tendon, advances in our understanding of this tendon pathology have revealed that the correct label is in fact tendinosis (Khan, Cook, Maffulli et al., 2000c; Khan, Cook, Tauton et al., 2000b). This is due to increased evidence that indicates injury resulting from overuse of the patellar tendon is not from an inflammatory process but rather from degeneration of the patellar tendon (Alfredson et al., 2001; Khan, Cook, Maffulli et al., 2000c; Khan, Cook, Tauton et al., 2000b). Therefore, the correct name, patellar tendinosis, will be used to describe this overload lesion of the patellar tendon from here onwards.

Patellar tendinosis is any microtearing along the knee extensor mechanism from the quadriceps tendon insertion on the upper pole of the patella, to the proximal patellar tendon insertion on the inferior pole of the patella and the tibial tuberosity (see Figure 2-6; Brukner & Khan, 1993; Garrick & Webb, 1990). Clinical signs and/or symptoms of patellar tendinosis are shown in Table 2-1 whereas various methods which can be used to assist in diagnosing patellar tendinosis are listed in Table 2-2.
The level of severity of patellar tendinosis can be classified via symptoms using classification systems such as those according to Blazina et al. (1973) or Roels et al. (1978) as shown in Table 2-2. The severity of patellar tendinosis can also be determined by scales such as the Victorian Institute of Sport Assessment (VISA) Score (Visentini et al., 1998), which is a reliable index of severity of patellar tendinosis, reflecting subtle changes in symptoms. However, the VISA score is not a diagnostic test and is irrelevant for patients with other knee conditions. Another method for classifying patellar tendinosis was devised by McLoughlin et al. (1995) who used magnetic resonance imaging (MRI) to objectively grade damage associated with patellar tendinosis based on the enthesial region at MRI.
Table 2-1: Clinical signs and/or symptoms of patellar tendinosis.

<table>
<thead>
<tr>
<th>Clinical Signs and/or Symptoms</th>
<th>References</th>
</tr>
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<tbody>
<tr>
<td>Local tenderness on palpation of the patella</td>
<td>Alfredson et al. (2001), Blazina et al. (1973), Cook, Khan, Maffulli et al. (2000c), Eifert-Mangine et al. (1992), Lian, Engebretsen et al. (1996a), Martens et al. (1982), McLoughlin et al. (1995), Panni et al. (2001), Roels et al. (1978), Terslev et al. (2001), and Witvrouw et al. (2001).</td>
</tr>
<tr>
<td>Minor signs and/or symptoms including quadriceps atrophy</td>
<td>Colosimo &amp; Bassett (1990), Cook, Khan, Maffulli et al. (2000c), Eifert-Mangine et al. (1992), Panni et al. (2001), and Roels et al. (1978).</td>
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Table 2-2: Diagnosis of patellar tendinosis.

<table>
<thead>
<tr>
<th>Method of Diagnosis</th>
<th>Diagnostic Signs</th>
<th>References</th>
</tr>
</thead>
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<tr>
<td>Detailed patient history</td>
<td>Pain during activities</td>
<td>Cook, Khan, Maffulli et al. (2000c), Lian, Engebretsen et al. (1996a), Martens et al. (1982), McLoughlin et al. (1995), and Terslev et al. (2001).</td>
</tr>
<tr>
<td>Physical examination:</td>
<td></td>
<td></td>
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<tr>
<td>• patellar tendon</td>
<td>• tenderness during palpation</td>
<td>Cook, Khan, Maffulli et al. (2000c), Lian, Engebretsen et al., (1996a); Martens et al. (1982), Panni et al. (2001), Terslev et al. (2001), and Watson (1988).</td>
</tr>
<tr>
<td>• quadriceps function</td>
<td>• atrophy</td>
<td>Cook, Khan, Maffulli et al. (2000c), Eifert-Mangine et al. (1992) Martens et al. (1982), Panni et al. (2001), Roels et al. (1978), and Watson (1988).</td>
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<tr>
<td>• knee stability</td>
<td></td>
<td>Sandmeier &amp; Renström (1997), and Thomeé et al. (1995).</td>
</tr>
<tr>
<td>• patellofemoral joint</td>
<td>• hypermobility &amp; poor tracking</td>
<td>Eifert-Mangine et al. (1992), and Sandmeier &amp; Renström (1997).</td>
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<td>• lower extremity alignment</td>
<td>• Q-angle, patellar alta, tibial torsion, excessive pronation</td>
<td>Blazina et al. (1973), Eifert-Mangine et al. (1992), Griffiths &amp; Selesnick (1998), Panni et al. (2001), Thomeé et al. (1995), and Watson (1988).</td>
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<td>• extensibility</td>
<td>• poor dorsiflexion range of motion</td>
<td>Eifert-Mangine et al. (1992) and Sandmeier &amp; Renström (1997).</td>
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<td>• patella</td>
<td>• orientation, glide, tilt, rotation &amp; anterior-posterior position</td>
<td>Blazina et al. (1973), Eifert-Mangine et al. (1992), and Griffiths &amp; Selesnick (1998).</td>
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<td>• patella grinding test</td>
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<td>Thomeé et al. (1995).</td>
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<td>• McConnell test</td>
<td>• to exclude PFPS</td>
<td>Watson (1988).</td>
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<td>Imaging</td>
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<td>Cook, Khan, Kiss, Coleman, &amp; Griffiths (2001), Khan et al. (1999), Lian, Holen et al. (1996a), Panni et al. (2001), Terslev et al. (2001), and Witvrouw et al. (2001).</td>
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<td>• ultrasound</td>
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<tr>
<td>• x-ray</td>
<td>• patellar height, soft tissue calcification, tendon thickness.</td>
<td>Blazina et al. (1973), Kujala et al. (1989), Martens et al. (1982), Panni et al. (2001), and Sandmeier &amp; Renström (1997)</td>
</tr>
<tr>
<td>• computed tomography</td>
<td>• morphological</td>
<td>Davies et al. (1991), Guzzanti et al. (1994), and Mourad et al. (1988).</td>
</tr>
<tr>
<td>• magnetic resonance imaging</td>
<td>• morphological</td>
<td>Cook, Khan, Kiss, Coleman et al. (2001), Khan et al. (1996), Khan et al. (1999), McLoughlin et al. (1995), Popp et al. (1997), and Yu et al. (1995).</td>
</tr>
</tbody>
</table>
Table 2-3: The phases of patellar tendinosis classified by symptoms according to (A) Blazina et al. (1973) and (B) Roels et al. (1978).

<table>
<thead>
<tr>
<th>Phase</th>
<th>A</th>
<th>B</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Pain after activity only. No undue functional impairment.</td>
<td>Pain at the infrapatella or suprapatella region after practice or after an event.</td>
</tr>
<tr>
<td>2</td>
<td>Pain during and after activity. Still able to perform to a satisfactory level.</td>
<td>Pain at the beginning of an activity, disappearing after warm-up and reappearing after completion of activity.</td>
</tr>
<tr>
<td>3</td>
<td>Pain during and after activity and more prolonged. Patient has progressively increasing difficulty in performing at a satisfactory level.</td>
<td>The pain remains during and after the activity and the patient is unable to participate in sports.</td>
</tr>
<tr>
<td>4</td>
<td>------</td>
<td>Represents a complete rupture of the tendon.</td>
</tr>
</tbody>
</table>

2.5.1. Etiology of Patellar Tendinosis

Currently there is limited research on the intrinsic and extrinsic factors that increase the risk of patellar tendinosis* (see Section 1.1.). Intrinsic risk factors are considered those factors from within the body (Barker, 1997) or factors related to an individual’s physical characteristics and/or psychological traits (Lysens, 1991). These factors are not easily modifiable. Therefore, the emphasis in this present thesis is on extrinsic factors, that is, those factors external to the body that impact upon it. However, to gain a comprehensive understanding of patellar tendinosis, literature pertaining to intrinsic risk factors will be briefly reviewed.

*(Bergstrom et al., 2001; Blazina et al., 1973; Cook, Khan, Kiss, & Griffiths, 2000a; Cook, Khan, Kiss, Purdam et al., 2000b; Cook, Khan, Maffulli, et al., 2000c; Duri & Aichroth, 1995; Duri et al., 1999; Ferretti, 1986; Ferretti et al., 1983; Ferretti et al., 1984; Johnson, 1996; Lian, Engebretsen et al., 1996a; Martens et al., 1982; Panni et al., 2001; Richards et al., 1996; Roels et al., 1978; Sanchis-Alfonso et al., 2001; Witvrouw et al., 2001).
2.5.2. Intrinsic Risk Factors

2.5.2.1. Anatomy of the Patella

Differences in the anatomy of the patella are thought to be an intrinsic factor in the risk of developing patellar tendinosis. For example, Shalaby & Almekinders (1999) observed a relationship between the length of the non-articular surface of the patella and the presence of signal changes in MRI in subjects with patellar tendinosis. The authors suggested that a traction osteophyte might represent the long non-articular portion, caused by repeated high tensile forces in this area and/or age-related tendon degeneration.

Patellar alta is defined as a patellar tendon length 20% greater than the height of the patella (Eifert-Mangine et al., 1992). Johnson (1996) observed that 25% of 19 subjects studied with chronic patellar tendinosis had an increased Insall-Salvati patella height index, a method of measuring patella height. Similarly, Kannus & Natri (1997) associated patellar alta with the risk of developing patellar tendinosis.

Grelsamer et al. (1994) classified patients with patellar tendinosis into either Type I, II or III, based on the ratio of patella height to the length of the articular surface of the patella. Type II patellae typically had a smaller articular surface length and a longer inferior pole, whereas Type III patellae patients had a significantly shorter patella height and a longer patellar tendon length. The authors noted that patients with either Type II or Type III patellae were more likely to present with patella abnormalities. Singerman et al. (1994) stated that by increasing patella height, the patellofemoral resultant contact force increases, thus increasing the force that the knee joint must withstand.

A thickening of a central ridge of patellar tendon fibres on the patella has been noted in patellar tendinosis patients during ultrasound (Karlsson et al., 1991 cited in Basso et al., 2001) and MRI (el-Khoury et al., 1992; Johnson, 1996). Basso et al. (2001) stated that this posterior ridge of fibres may play a role in the thickening appearance of the patellar tendon in patellar tendinosis. It may also explain why the posterior fascicles of the patellar tendon are more commonly affected in patients with patellar tendinosis, as they are shorter than those fibres on the anterior side of the patella and therefore sustain a greater strain during knee flexion. By causing a poor patellofemoral movement
mechanism, this may lead to an increased risk of developing patellar tendinosis. This is supported by associations of an increased risk of developing patellar tendinosis with a high riding patella (Kannus & Natri, 1997), excessive lateral displacement of the patella (Kannus & Natri, 1997), patella hypermobility (Blazina et al., 1973; Cook, Khan, Maffulli et al., 2000c), patella hypomobility (Cook, Khan, Maffulli et al., 2000c), and subluxation (Blazina et al., 1973).

2.5.2.2. Patellofemoral Malalignment

Patellofemoral abnormalities caused by patellofemoral malalignment appear to be a dynamic phenomenon (Guzzanti et al., 1994). Guzzanti et al. (1994) found that unrecognised patellofemoral malalignments could be revealed when patients contracted their quadriceps muscles. For example, the authors found that lateralisation and tilting of the patella were more severe when patients contracted their quadriceps muscles. Sanchis-Alfonso et al. (2001, unpublished data) observed that 49% of patients with patellar tendinosis had patellofemoral malalignment. However, in 42 athletes with patellar tendinosis, none of the athletes had any extensor malalignments (Panni et al., 2001). Therefore, the association between patellofemoral malalignments and the risk of developing patellar tendinosis requires further investigation.

2.5.2.3. Leg Length Discrepancy

Leg length discrepancy is thought to contribute to the development of patellar tendinosis by causing compensatory changes that alter lower limb function. Discrepancy in leg length can be caused either structurally by anatomical lengthening/shortening of bones, or functionally by soft tissue shortening/relaxation causing compensatory changes in the lower limb. For example, leg length inequality can occur secondary to a rotated pelvis and/or by environmental factors, such as an uneven surface (Blustein & D'Amico, 1985; McCaw, 1992; Neely, 1998b). Leg length discrepancy can also cause the subtalar joint to maximally pronate on the longer limb (Blustein & D'Amico, 1985; Gross, 1995; Kannus & Natri, 1997) or shorter limb (Kannus & Natri, 1997), and produce contralateral subtalar joint supination, increasing the calcaneal inclination, decreasing the talocalcaneal angle, and decreasing the talar declination to lengthen the shorter limb and rebalance the pelvis (Blustein & D'Amico, 1985; Gross, 1995; Kannus & Natri, 1997).
1985). Excessive pronation has been associated with patellar tendinosis (see Section 2.5.2.4.).

Controversy surrounds the exact value for which a significant leg length discrepancy is considered to exist. Values range from a between-limb length difference of 0.64 cm (Messier & Pittala, 1988) to 1.5 cm (Subotnick, 1976 cited in Neely, 1998a). Subotnick (1976, cited in Neely, 1998a) stated that a minor leg length discrepancy that would cause insignificant problems in a non-athlete, could be magnified in an active athlete causing significant symptoms. However, Witvrouw et al. (2001) found no association between leg length discrepancy and patellar tendinosis. Until the controversy surrounding what constitutes a leg length discrepancy is resolved, this intrinsic risk factor cannot be appropriately assessed to determine if it can lead to patellar tendinosis.

2.5.2.4. Foot Type

Subtalar joint pronation provides a shock absorbing mechanism for the body during landing (Maeda, 1998). That is, the impact of landing is attenuated by the cushioning effect of subtalar joint motion. However, pronation affects internal rotation of the lower extremity and may also cause medial translation of the entire knee in the frontal plane relative to the fixed foot and pelvis (Gross, 1995). Excessive pronation of the foot can therefore disrupt patellofemoral mechanics by causing internal tibial rotation and increasing femoral anteversion (Krivickas, 1997). This can cause abnormal patellofemoral joint forces, torques and stresses (Gross, 1995) and thereby contribute to the development of patellar tendinosis. This notion has been supported by Cook, Khan, Maffulli et al. (2000c), who associated an excessive range of pronation, an excessively fast pronation rate even with a normal range, pes planus, rigid cavus feet, and poor dorsiflexion range of motion due to anterior impingement syndrome with patellar tendinosis.

2.5.2.5. Q-angle

The Q-angle, another possible intrinsic risk factor, is formed between a line connecting the anterior superior iliac spine of the pelvis to the midpoint of the patella and a line connecting the tibial tubercle and the midpoint of the patella (Cowan et al., 1996; Greisamer & Klein, 1998; Hahn & Foldspang, 1997; Kannus & Niittymäki, 1994;
Livingston, 1998; Messier et al., 1991). Different studies have reported different mean values and ranges for Q-angles in male and females. For example, Q-angles have been reported to range from 10° to 20° (Thomeé et al., 1995), 6° to 24° (Caylor et al., 1993) and 6° to 13° (Hahn & Foldspang, 1997) with mean Q-angle values reported of 12° (Livingston, 1999) and 17° (France & Nester, 2001). These discrepancies between studies most likely stem from the Q-angle being highly sensitive to measurement error (France & Nester, 2001). However, irrespective of differences in specific Q-angles, there is consensus that Q-angle is gender dependent with females tending to have larger Q-angles than males due to their wider pelvis (Caylor et al., 1993; Hahn & Foldspang, 1997; Krivickas, 1997). In females and males respectively, Q-angles have been reported of 11° and 10° (Livingston, 1999), 16° and 11° (Horton & Hall, 1989, cited in Caylor et al., 1993), and 17° and 14° (Percy & Strother, 1985, 1989, cited in Caylor et al., 1993). Hahn & Foldspang (1997) stated that high quadriceps muscular strength and tonus tended to straighten the Q-angle. The authors proposed that the smaller Q-angles in males than women were due to men being typically stronger, and thus straightening the Q-angle rather than just differences in bony alignment.

Controversy also surrounds the definition of an abnormal Q-angle value (Hahn & Foldspang, 1997), with values of greater than 17° (Messier et al., 1991; Messier & Pittala, 1988) or 15° in males and 20° in females (Hahn & Foldspang, 1997) being defined as abnormal. A large Q-angle may be caused by several factors including an increased lateral displacement of the tibial tuberosity (Messier et al., 1991), increased femoral anteversion (Caylor et al., 1993; Messier et al., 1991), relative knee valgus (Norkin & Levangie, 1983), external tibial torsion (Caylor et al., 1993; Messier et al., 1991) and genu valgum (Caylor et al., 1993; Greilsamer & Klein, 1998; Messier et al., 1991).

During quadriceps contractions, the patella should move within the femoral trochlear notch (Messier et al., 1991), drawing the anterior superior iliac spine and the tibial tuberosity towards each other, causing a decrease in the Q-angle (Hahn & Foldspang, 1997). As the quadriceps muscles contract, an increased Q-angle predisposes the patella to a lateral deviation, pressing the patella against the underlying lateral femoral condyles, in turn, increasing the lateral patellofemoral contact pressure (Messier et al.,

28
1991; Mizuno et al., 2001) and the risk of patella subluxation or dislocation (Mizuno et al., 2001). Eifert-Mangine et al. (1992) suggested that the greater valgus vector resulting from an increased Q-angle could increase the lateral pull of the patella and may cause microtears in the infrapatellar tendon. As patellar tendinosis is defined as any microtearing along the knee extensor mechanism, this may have implications in the development of patellar tendinosis.

Increased Q-angles, however, have not always been shown to increase the risk of patellar tendinosis. For example, Witvrouw et al. (2001) reported finding no difference in Q-angles between physical education students who developed patellar tendinosis and those students who did not develop the injury over two years. Furthermore, most studies examining the relationship between Q-angles and patellar tendinosis have restricted Q-angle assessment to static measurements of the angle. In a dynamic measurement of Q-angle, the Q-angle changes depending upon the type of landing performed. For example, in female netball players, a significantly larger Q-angle was noted at the touch down phase of landing in a propping task compared to a two-to-one foot landing (Hume et al., 1996). The authors stated that when interpreting lower limb data, Q-angles should be adjusted for initial standing stance, and therefore measuring the change in Q-angle may be more sensitive than the actual Q-angle magnitude. Additionally, there is a large degree of inaccuracy in measuring the Q-angle due to the difficulties arising in locating the centre of the patella and the tibial tuberosity (France & Nester, 2001), such that subtle changes in the angle may be masked by measurement errors. Therefore, further research in both static and dynamic situations is required to assess the relationship between increased Q-angle, and the risk of developing patellar tendinosis.

2.5.2.6. Femoral Internal Rotation

Femoral anteversion is a pathological increase in the angle of femoral torsion that occurs in the transverse plane between the axis of the femoral neck and the axis of the femoral condyles (Norkin & Levangie, 1983). An individual with anteversion will tend to internally rotate their leg during gait to keep the femoral head in the acetabulum (Nordin & Frankel, 1989). Increased femoral internal rotation can cause compensatory changes that lead to external tibial rotation and subtalar pronation, in turn, producing
lateral patellofemoral tracking (Carson et al., 1984, cited in Eifert-Mangine et al., 1992; Tibero, 1987, cited in Eifert-Mangine et al., 1992). Increased femoral anteversion can also cause an abnormal line of muscle pull, which can lead to patellofemoral joint incongruity and malalignment, in turn, unevenly distributing the shearing and compressive forces on the patellofemoral joint, focusing these forces onto a small part of the joint surface (Sikorski et al, 1979 cited in Neely, 1998a).

Femoral anteversion has been associated with patellar tendinosis (Cook, Khan, Maffulli et al., 2000c; Duri & Aichroth, 1995; Duri et al., 1999). For example, Duri & Aichroth (1995) and Duri et al. (1999) noted that in 24% and 23% of patients with patellar tendinosis, there was excessive femoral anteversion. Blazina et al. (1973) also noted external tibial torsion present in patients with patellar tendinosis. It would appear that the compensatory adaptations required in response to excessive internal femoral rotation may contribute to the development of patellar tendinosis by causing abnormal patellofemoral joint forces, torques and stresses, and muscular imbalance.

2.5.2.7. **Genu Varum/Valgum**

Genu valgum (also known as "knock knees") is the condition when the medial tibiofemoral angle is greater than 195° (Norkin & Levangie, 1983). This lower limb alignment increases the compressive forces on the lateral tibial condyle as well as increasing the tensile forces on the medial aspect of the tibia (Norkin & Levangie, 1983). Genu varum ("bow legs") results when the medial tibiofemoral angle is less than 180°. In such cases the compressive forces increase on the medial aspect of the tibia and the tensile forces increase on the lateral aspect of the tibia (Norkin & Levangie, 1983).

Despite associating patellar tendinosis with genu valgum (Blazina et al., 1973; Kannus, 1992) and genu varum (Kannus, 1992), no evidence was located directly linking the risk of genu varum or genu valgum with the development of patellar tendinosis. Similar to femoral internal rotation, it is thought that genu varum/valgum may contribute to the development of patellar tendinosis by causing abnormal patellofemoral joint forces, torques and stresses (Blazina et al., 1973; Kannus, 1992). However, further research is warranted to justify this notion.
2.5.2.8. **Muscular Extensibility**

Poor extensibility of the quadriceps muscles, hamstring muscles, iliotibial band and/or calf muscles may increase loads on the patellofemoral joint and the patellar tendon (Cook, Khan, Maffulli et al., 2000c; Eifert-Mangine et al., 1992), by restricting ankle and knee joint motion (Cook, Khan, Maffulli et al., 2000c). Tight hamstring muscles can also cause abnormal patella tracking (Brukner & Khan, 1993; Eifert-Mangine et al., 1992), iliotibial band tightness can pull the patella laterally during knee flexion (Eifert-Mangine et al., 1992), and gastrocnemius tightness can restrict dorsiflexion at the talocrural joint causing compensatory subtalar joint pronation (Eifert-Mangine et al., 1992). The relationship between muscle tightness and patellar tendinosis was highlighted in a two year prospective study in which decreased quadriceps muscle and hamstring muscle extensibility was found to be a predisposing factor for the development of patellar tendinosis in physical education students (Witvrouw et al., 2001). Blazina et al. (1973) also found tight hamstring muscles and tight Achilles tendons to be associated with patellar tendinosis. Duri et al. (1999) and Duri & Aichroth (1995) found that hamstring tightness was present in 75% and 37% of the knees of patients with patellar tendinosis, respectively. The large discrepancy in the percentages between these two latter studies can be partially attributed to the fact that only 21 patients were recruited by Duri & Aichroth (1995) compared to 40 patients being recruited by Duri et al. (1999).

2.5.2.9. **Muscle Strength Imbalance**

Muscle strength imbalance caused by an asymmetry between an agonist and an antagonist muscle group in one extremity, asymmetry between the extremities, or a differential for a given muscle relative to a defined normal value (Kannus & Natri, 1997), may predispose an athlete to musculotendinous injuries by decreasing the energy-absorption capacity of the muscle-tendon unit (Jozsa & Kannus, 1997, cited in Kannus, 1997). For example, during landing from a jump, eccentric muscle contraction generates high forces in the ankle plantar muscles, knee extensor muscles and hip extensor muscles, to slow the body's downward momentum by elongating the muscles (Watson, 1988). If there is a muscular imbalance from weakness or previous lower limb muscle injury, this may cause tensile overload of the quadriceps muscles leading to patellar tendinosis (Watson, 1988).
Atrophy of the quadriceps muscles has been observed in patients with patellar tendinosis by Duri et al. (1999), Duri & Aichroth (1995) and Martens et al. (1982) who observed quadriceps atrophy in 85%, 67% and 19% of cases with patellar tendinosis, respectively. The vastus medialis obliquus is important in preventing lateral rotation of the tibia, and thus decreasing the Q-angle, and lateral patella traction. A weaker vastus medialis near extension can cause a lateral shift and tilt of the patella (Bull et al., 1998; Sakai et al., 2000). However, Witvrouw et al. (2001) reported finding no association between isokinetic quadriceps or hamstring muscle strength and patellar tendinosis.

The authors cautioned though that measuring muscle strength isokinetically was a poor representation of functional strength during explosive movements. Therefore, the association between muscle strength imbalances and the risk of developing patellar tendinosis requires further investigation, particularly as it relates to strength deficits displayed during dynamic movements such as jumping.

2.5.2.10. Other Intrinsic Risk Factors

Other intrinsic risk factors that have received only minor investigation and/or are controversial with respect to their feasibility as a risk factor to patellar tendinosis are shown in Table 2-4. As these risk factors, like most other intrinsic risk factors, are difficult to modify, attention in this thesis will be directed towards extrinsic risk factors.

Table 2-4: Intrinsic risk factors possibly associated with patellar tendinosis.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Supporting references</th>
<th>Non-supporting references</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint hypermobility</td>
<td>Bergstrom et al. (2001), and Martens et al. (1982).</td>
<td></td>
</tr>
<tr>
<td>Height</td>
<td>Duri &amp; Aichroth (1995), and Duri et al. (1999).</td>
<td>Martens et al. (1982), and Witvrouw et al. (2001).</td>
</tr>
<tr>
<td>Age</td>
<td>Cook, Khan, Kiss, &amp; Griffiths (2000a).</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>Cook, Khan, Kiss, &amp; Griffiths (2000a).</td>
<td>Witvrouw et al. (2001).</td>
</tr>
<tr>
<td>Injury History</td>
<td>Blazina et al. (1973), Duri &amp; Aichroth (1995), and Duri et al. (1999).</td>
<td></td>
</tr>
</tbody>
</table>
2.5.3. **Extrinsic Risk Factors**

Extrinsic risk factors are more readily changed than intrinsic risk factors as extrinsic factors arise from outside the body (see Section 1.1.). Therefore, literature pertaining to extrinsic risk factors for patellar tendinosis will be explored in more depth than intrinsic risk factors in the following sections.

2.5.3.1. **Excessive External Load**

Excessive loads combined with high repetitions of the load on the body is considered to be the main risk factor associated with patellar tendinosis*. Fredberg & Bolvig (1999) proposed that when a tendon becomes overused, there is a mismatch between the stress on a given tissue and the ability of that tissue to withstand the stress (see Figure 2-7). Damage to the tendon is continued by repetitive trauma, that may further weaken the tendon’s collagen cross-linking (Harries, 1994; Järvinen et al., 1997) and the non-collagenous matrix, as well as disturb the micro and macrovasculature of the tendon. This, in turn, can cause insufficient local blood circulation leading to local tissue hypoxia, and impaired nutrition and energy metabolism of the tendon structure (Järvinen et al., 1997).

![Figure 2-7: The pain cycle (adapted from Harries, 1994).](image)

*(Colosimo & Bassett, 1990; Cook, Khan, Kiss et al., 2000a; Eifert-Mangine et al., 1992; Ferretti, 1986; Ferretti et al., 1983; Griffiths & Selesnick, 1998; Lian, Engebretsen et al., 1996a; Martens et al., 1982; Powell et al., 1998; Roels et al., 1978; Romeo & Larson, 1999; Sanchis-Alfonso et al., 2001).*
Activities involving rapid acceleration and deceleration, such as jumping and landing movements, place a large stress on the extensor mechanism (Fredberg & Bolvig, 1999). For example, during an eccentric muscle contraction, such as in landing, the leg extensor muscles decelerate the downward motion of the body (Rodacki & Fowler, 2001). It is these extensor muscles of the lower limbs which act to dissipate the kinetic energy generated during landing (Hoffman et al., 1997). In jumping, the patellar tendon may sustain forces as high as 8,000 N, compared to only 500 N in the less vigorous task of walking (Stanish et al., 1986). It is this large load placed on the extensor mechanism, which is repetitively sustained during activities involving repeated jumping, that is thought to be a major extrinsic risk factor for patellar tendinosis (Cook, Khan, Kiss, & Griffiths, 2000a; Harries, 1994).

Although a relationship between high repetitive loading and tendon injury has been established, the relationship between landing technique, impact loading and patellar tendinosis is less clear. For example, Lian, Engebretsen et al. (1996a) showed that athletes who performed significantly better in a standardised series of vertical jump and power tests of vertical jumping also tended to have a higher incidence of patellar tendinosis. The authors suggested that the risk of patellar tendinosis was related to the load placed on the extensor mechanism during jumping. However, due to the small sample size ($n = 12$), these results need to be interpreted carefully as variations in ground reaction forces may not always reflect variations in joint loading.

The way an individual dissipates the ground reaction forces generated during landing can also vary greatly and can be observed in the vertical ground reaction force time curves. Nigg (1983) classified three different types of landing based on the resulting vertical ground reaction force time curves as follows (see Figure 2-8):

1. A Type 1 landing demonstrated a first dominant impact peak followed by a higher active peak, whereby the first impact peak was thought to be caused by a definite rearfoot strike with the landing surface;

2. A Type 2 landing consisted of two impact peaks followed by an active peak. The second impact peak was thought to be caused by either the impact of the heel landing after the initial forefoot contact of the surface or landing on the lateral to the medial aspect of the foot; and
(3) A Type 3 landing had no impact peak and therefore had only an active peak. This landing was thought to be characteristic of a midfoot landing.

Figure 2-8: Different types of landing patterns displayed during landing (a) Type 1 (b) Type 2 and (c) Type 3 (adapted from Nigg, 1983, p.18).
The forces generated at landing can therefore be differentiated by variations in foot placement strategy during landing (Kovacs et al., 1999; Nigg, 1983). Using a forefoot landing is thought to protect bones and joints superior to the impact site from excessive loading by the ankle plantar flexor muscles functioning to absorb the energy associated with impact attenuation (Kovacs et al., 1999). However, a forefoot landing may predispose the small bones of the foot itself to injury as a consequence of the impact loading they must sustain. In a heel-toe landing, the impact of landing cannot be attenuated using the ankle plantar flexor muscles, as the centre of pressure of the foot at initial contact is located at the calcaneus and nearer to the ankle joint centre (Kovacs et al., 1999). This, in turn, decreases the angular displacement of the ankle joint causing a significantly smaller amount of dorsiflexion and, in turn, greater knee and hip flexion to dissipate the impact force during the heel-toe landing (Kovacs et al., 1999).

In a landing skill specific to volleyball, Richards et al. (1996) found that a typical vertical ground reaction force time curve obtained at landing for a spike jump performed by elite male volleyball players was characterised by an initial impact peak followed by a second larger impact peak (Type 1 landing). The vertical ground reaction force, was on average, 5.6 to 6.0 times the body weight of the subjects, with one player sustaining forces averaging 9.5 times body weight when performing a spike jump landing. These values were approximately double those recorded during the spike jump take-off, the block jump landing or running. Adrian & Laughlin (1983) reported similarly high ground reaction forces at landing in Indoor Volleyball, where players performing the spike jump sustained the highest peak vertical forces, whereas those performing the stationary block jump had the least. The larger ground reaction forces generated during the spike jump landing in volleyball, relative to other skills characteristic of the game, would be assumed to increase the patellofemoral joint load and may, in turn, create excessive load on the patellar tendon. This notion is supported by Richards et al. (1996) who found that significant predictors of patellar tendinosis in elite volleyball players (n = 10) were a larger VGRF, together with a high knee extensor moment rate and greater knee flexion at landing.

Studies examining the landing phase of movement skills other than those typical of volleyball have reported times to the peak vertical ground reaction force during a
forefoot landing of 68 ms in a drop jump landing (Kovacs et al., 1999) and 47 ms in a single-limb netball landing (Steele & Milburn, 1989). During a heel-toe landing these values were 89 ms during a drop jump landing (Kovacs et al., 1999) and 21 ms in a netball landing (Steele & Milburn, 1989). On different landing surfaces, McNitt-Gray et al. (1993) reported times to the peak vertical ground reaction force during landing of 57 ms and 51 ms from a low and high vertical jump height upon a stiff matt, and 64 ms and 55 ms from a low and high vertical jump height onto a soft matt.

In an effort to reduce the vertical forces generated during landing, Dufek & Bates (1990) suggested that players should land with adequate knee flexion and a toe-heel landing. However, Hoffman et al. (1997) claimed that by bending their knees more, novice jumpers may cause the landing forces to be dissipated primarily on one part of the body, such as the patellar tendon. In contrast, experienced jumpers have been shown to generate greater extensor moments during landing, with a stiffer landing pattern which was reflected by a smaller amount of knee flexion, allowing for a more rapid landing sequence to dissipate the impact forces fast and efficiently (Hoffman et al., 1997). This notion was supported by Viitasalo et al. (1998) who observed that skilled triple jumpers adopted different neuromuscular strategies during drop jump landings so they were more efficient to resist the high stretching speeds and resultant ground reaction forces, than the control subjects, thereby flexing their knees less at landing.

Stacoff et al. (1988) found that the impact forces during a block jump landing performed by skilled volleyball players (n = 12) increased the less the subjects flexed their knees. The authors stated that the lowering of the thigh and the upper body from the peak vertical height of the block jump and the impact of landing caused the knee joint to flex at a faster angular velocity due to the contribution of the increased impact. During knee flexion, the patellar tendon and the quadriceps muscles are under tension (Stacoff et al., 1988). The greater the knee angular displacement, the more efficient the patellar tendon is able to dissipate the impact force as it has a longer duration over which to dissipate the impact forces sustained during landing (Lafortune, 1985). This notion was supported by McNitt-Gray et al. (1993), who found that landing with less knee joint flexion enabled more range of motion over which to accommodate
unexpected events, allowing the athlete to distribute the load between the tibiofemoral and patellofemoral joints more efficiently. However, in previous work McNitt-Gray (1991) observed that during drop jump landings performed by both male gymnasts and recreational athletes, the peak impact forces significantly increased with increases in impact velocity and lead to increased knee and hip joint flexion. The author suggested that the major adjustments during landing were made by the knee and hip joints rather than the ankle joint due to their larger range of motion.

Plyometric training exercises, whereby players perform repetitive jumping movements, are frequently performed by both Indoor and Beach Volleyball players to develop their ability to jump high (McGown et al., 1990; Newton et al., 1999). By increasing the number of jumps performed, it would be assumed that the load sustained by an individual is increased and, in turn, the risk of developing patellar tendinosis would be increased. However, it has been shown by Hewett et al. (1996) that, following a six week plyometric training program, male and female high school volleyball players were able to decrease the forces generated at landing despite increasing their vertical jump height. Therefore, learning correct landing technique can lessen the loads generated at landing (Hewett et al., 1996). However, no study was located to identify what constitutes good landing technique in Beach Volleyball when players are required to land on a sand surface.

2.5.3.2. Surface Type

The type of landing surface is also considered a primary extrinsic risk factor in the development of patellar tendinosis. However, the relative risk of different types of surfaces in the development of patellar tendinosis has only received limited investigation (Duri & Aichroth, 1995; Duri et al., 1999; Ferretti et al., 1984).

An injury analysis of the elite USA volleyball players during the 1996 USA Olympic Festival revealed that volleyball injuries were less likely to occur on sand than on hard surfaces (see Figure 2-9; Briner & Ely, 1999). However, it must be noted that the mechanical properties of sand surfaces between beaches can vary greatly. For example, Barrett et al. (1997) observed that the surface stiffness of a compacted sand surface was six times higher compared to a dry non-compacted sand surface, with the wet
compacted sand surface being of similar stiffness to synthetic surfaces tested by Nigg (1985, cited in Barrett et al., 1997). The wet compact sand surface also had an increased impact force, a decreased contact time and a decreased surface penetration compared to a non-compacted sand surface. Furthermore, due to the nature of a sand surface, the poor stability of the surface may in fact increase the risk of injury due to increased rear foot motion (Barrett et al., 1997). Barrett et al. (1997) stated that the differences in sand surface characteristics might have implications for injury prevention and performance enhancement in sand runners. These variations in sand characteristics may also have implications for injury prevention in Beach Volleyball players.

Ferretti et al. (1984) reported that the incidence of patellar tendinosis in Indoor Volleyball players varied depending on the type of playing surface. That is, on a cement surface, 37.5% of the players were affected by patellar tendinosis, whereas on a wooden surface, only 4.7% of the players were affected by patellar tendinosis. Fredberg & Bolvig (1999) postulated that harder surfaces increased the stress on the bone-tendon unit, whereas surfaces that had a moderate degree of elasticity partially mitigated the stress. The role of different surfaces in jumping activities may also have implications in the risk of developing patellar tendinosis, with harder surfaces such as concrete possibly

\[ \text{Figure 2-9: Surface on which injury was reported to have occurred during the 1996 USA Olympic Volleyball Festival (adapted from Briner & Ely, 1999, p. 10).} \]
increasing the risk of patellar tendinosis when compared to softer surfaces such as wooden floors or soft sand. Duri et al. (1999) and Duri & Aichroth (1995) supported this notion by showing that training on hard surfaces had an influence on the development of patellar tendinosis in 55% and 63%, respectively, of patients with patellar tendinosis. Nigg et al. (1988) assumed that a decrease in overload injuries might occur by reducing landing loads by increasing the ability of the surfaces to deform.

The use of different surface types has also been shown to influence landing mechanics. For example, greater knee flexion during landing from a drop jump bench is observed to occur on a stiff mat compared to a soft mat (McNitt-Gray et al., 1993). Apart from surface effects on knee flexion, Streepey et al. (2000) observed that the type of floor influenced the muscle activation patterns and muscle loading of basketball players performing a similar basketball task. The authors observed that a composite floor might protect the ankle plantar flexor muscles from fatigue during landing from this simulated basketball task, as there was a smaller decrease in maximum muscle force on the composite floor compared to the wood floor. However, it is not known how landing on different volleyball surfaces, such as a sand surface, affects the mechanics of landing or the risk of developing patellar tendinosis.

2.5.3.3. Training
Another factor identified as an extrinsic risk factor for patellar tendinosis is the manner in which a player trains. For example, the incidence of patellar tendinosis was shown to increase from 3.2% to 41.8% in volleyball players training twice per week compared to greater than four training sessions per week (Ferretti et al., 1984). This finding was supported by Duri & Aichroth (1995) who observed that an increased frequency of training was involved in 44% of patients diagnosed with patellar tendinosis. However, Witvrouw et al. (2001) observed no difference in the type and amount of external sports activities between groups of physical education students who developed patellar tendinosis and those students who did not develop the injury. The authors concluded that patellar tendinosis was primarily caused by an intrinsic risk factor, and not the external overload that is commonly accepted to be the main cause of patellar tendinosis. Similarly, Ferretti et al. (1984) observed there was no difference in the incidence of
patellar tendinosis in volleyball players who practiced weight lifting or plyometric training. However, in a survey of high school ski students, an increased level of activity was related to patellar tendinosis (Bergstrom et al., 2001). No research was located examining the effects of training in Beach Volleyball on knee injury potential or how fatigue from excess training may contribute to the development of lower limb injuries.

2.5.4. Neuromuscular Fatigue

Another factor linked to lower extremity injury is fatigue (Coutts, 1982; Nyland et al., 1994; Wojtys et al., 1996). For example, Gabbett (2001) observed an increase in the incidence of injuries, as the number of matches progressed, from 99.2 per 1000 playing hours in the first match to 694.4 per 1000 hours in the fourth match of a Rugby League Sevens tournament. However, no studies were located that have investigated the effect of neuromuscular fatigue in the risk of developing patellar tendinosis, although impaired motor performance caused by neuromuscular fatigue may predispose athletes to developing this injury. As the relationship between fatigue, landing technique and lower limb injury is not well defined, literature associated with these topics will be further examined in this chapter.

Neuromuscular fatigue is an acute impairment of performance, with an increased perceived sense of effort and an inability to produce the desired force (Bigland-Ritchie, 1981; Enoka & Stuart, 1992). Currently there are numerous studies pertaining to neuromuscular fatigue and its effect on performance*. Evidence from these studies tends to show there is no single mechanism of fatigue, but rather a series of acute effects that impair motor performance (Avela & Komi, 1998; Bigland-Ritchie, 1981; Bigland-Ritchie et al., 1995; Enoka, 1995; Enoka & Stuart, 1992; Pinniger, 1996).

Fatigue can be either peripheral or central in origin (see Figure 2-10 and Figure 2-11). Peripheral fatigue is the result of impaired force generation at the muscle, whereas central fatigue is caused by a failure in neural drive (Macera, 1989). Peripheral fatigue can occur at the neuromuscular junction and the muscle cell membrane, the calcium ion (Ca\(^{2+}\)) release mechanism, or at the level of the sliding filaments (Macera, 1989).

**Figure 2-10:** Peripheral fatigue sites. Pre-synaptic failure (1), inability to develop an action potential at the motor end plate (2), failure of sarcolemma to sustain an action potential (3), loss of coupling of excitation between the T-tubule and sarcoplasmic reticulum (4), depressed Ca\(^{2+}\) release from the sarcoplasmic reticulum (5), reduced binding affinity of troponin for Ca\(^{2+}\) (6), a failure of the cross bridge cycle (7), delayed cross bridge dissociation (8), and depressed Ca\(^{2+}\) re-accumulation by the sarcoplasmic reticulum (9; adapted from Green, 1987, p. 8S).

One of the primary causes of fatigue in tasks requiring explosive strength is thought to be changes in central drive rather than peripheral fatigue. Changes in central drive have resulted in a decrease in drop jump performance when compared to fatigue in maximal strength tasks that primarily involve peripheral fatigue (Linnamo et al., 1998). Interestingly, the amount of time to recovery from fatigue may be shorter if the fatigue is mainly a result of central fatigue rather than peripheral fatigue (Linnamo et al., 1998). That is, to counteract a decrease in force due to peripheral fatigue mechanisms during
submaximal contractions, subjects can increase their motor commands by increasing their motor unit recruitment and firing frequency (Enoka & Stuart, 1992).

**Figure 2-11:** Central fatigue sites. Supraspinal failure (1), segmental afferent inhibition (2), depression of motorneuron excitability (3), loss of excitation at branch points (4) and presynatic failure (5) (adapted from Green, 1987, p.12S).

When the movement to be performed is changed or the task progresses, the mechanisms of fatigue can also vary (Bigland-Ritchie, 1981; Bigland-Ritchie et al., 1995; Botterman, 1995; Enoka, 1995; Enoka & Stuart, 1992). Variables able to be manipulated that may alter the mechanism(s) of fatigue include the motivation level of the subject, the intensity and duration of the task, the speed of the muscular contraction required to perform the task, and the degree to which the task is continuously sustained (Bigland-Ritchie, 1981; Bigland-Ritchie et al., 1995; Enoka & Stuart, 1992). These factors can influence the central nervous system drive to motor neurons, the muscle or the motor units activated (neural strategy), neuromuscular propagation, excitation-contraction coupling, the availability of metabolic substrates, the intracellular milieu, the contractile apparatus, and/or muscle blood flow (Bigland-Ritchie, 1981; Bigland-Ritchie et al., 1995; Enoka & Stuart, 1992).

### 2.5.4.1. Stretch-Shortening Cycle in Fatigue

Most studies examining fatigue have focused on activities involving concentric muscle contractions. However, increasing research has investigated the effects of fatigue
during movements involving a combination of eccentric and concentric muscle contractions, known as the stretch-shortening cycle (SSC; Norman & Komi, 1979). During explosive movements, such as performing a landing immediately followed by a rapid vertical jump, neuromuscular function is characterised by the SSC (Viitasalo et al., 1993; see Figure 2-12). That is, during the eccentric landing phase, the active muscles are stretched, or passively stretched muscles are suddenly activated, such that tension in these muscles increases, resulting in the storage of potential elastic strain energy in the series elastic component of the muscles and muscle-tendon unit (Komi, 1984). In the following concentric phase, when the active muscles are concentrically contracted to propel the body vertically upward, a portion of this stored energy is recovered and used to potentiate performance (Avela et al., 1996; Komi, 2000), if the concentric phase occurs immediately after the eccentric phase (Cavagna et al., 1968). If this muscle stretch is maintained for too long, the stored energy can be wasted as heat (Komi, 1984). As a result of SSC exercise, performance during the concentric phase of the movement is enhanced as is evident in an increased force at a given velocity compared to a pure concentric action (Komi, 1983, 2000). This enhanced performance is mainly attributed the stored elastic energy (Cavagna et al., 1965) as well as to the contribution of the stretch reflex in the potentiation of force (Komi, 2000).

![Figure 2-12: Stretch shortening cycle. (A) To resist the impact of landing, the lower limb extensor muscles are preactivated before landing, (B) the active braking phase in which the muscles are stretched (eccentric contraction), and (C) the stretch phase is followed by a shortening (concentric) action (adapted from Komi, 2000, p. 1198).](image)
The SSC is repetitively performed in many sporting activities, particularly those which involve repetitive vertical jumping movements such as volleyball. Therefore, an increasing number of studies have investigated how fatigue affects the SSC in terms of factors such as generating external loads, changes in metabolic substrates, muscle damage, and motor performance (Avela & Komi, 1998; Avela et al., 1996; Horita et al., 1996; Hortobagyi, 1991; Jones & Watt, 1971; Komi, 2000; MacLaren, 1982; Nicol et al., 1996). Factors affected by fatigue during landing and jumping movements relevant to this thesis are summarised in following sections.

2.5.4.2. External Loading and Fatigue

In a review of the literature, Enoka & Stuart (1992) concluded that the larger the force exerted, the greater the work done, or the faster the work performed during a task, the larger the muscular fatigue experienced. However, this rate-force relationship can be altered by factors such as the contractile properties of muscles, by muscle temperature and by muscle length (Gandevia, 1992). For example, muscular strength is a more important factor in fatigability than muscle fibre type characteristics (Kroll et al., 1980). Kroll et al. (1980) suggested that muscle mass was important in terms of both the maximum isometric strength achieved and fatigability. That is, an individual who is weaker will have to perform more effort to achieve the same amount of work than an individual who is stronger. This would cause the weaker individual to fatigue more rapidly than the stronger individual under a constant load. Blazina et al. (1973), Duri & Aichroth (1995), Duri et al. (1999) and Martens et al. (1982) observed quadriceps atrophy in individuals with patellar tendinosis. Thus, it could be assumed that an individual who is weaker may fatigue faster and may, in turn, be at a greater risk of developing patellar tendinosis in activities involving repetitive contractions of the extensor muscles of the leg than a stronger individual. In a study of isolated rabbit muscles, fatigued muscles were less able to absorb energy compared to non-fatigued muscles (Mair et al., 1996). As energy absorption is critical during the eccentric phase of a SSC, such as during landing, the decrease in energy absorption capacity of muscles caused by fatigue may increase the risk of injury due to the inability of the muscles to cope with the load during landing. Furthermore, as excessive and repetitive loading of the body is considered the main risk factor associated with patellar tendinosis (see
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Section 2.5.3.1.), fatigue may alter the forces characteristic of landing, potentially increasing the risk of patellar tendinosis.

As fatigue progresses, changes in ground reaction forces during SSC exercises have been observed and have been attributed to subjects having difficulty in maintaining constant angular displacement during contact with the ground (Komi, 2000). For example, Komi (2000) related the reduction in impact force in submaximal and maximal SSC exercises noted by Nicol et al. (1991) and in marathon running noted by Horita et al. (1996), to the faster and longer flexion moments displayed by the subjects. The author also stated that the reduction in impact forces indicated changes in mechanical performances that signified a loss of tolerance to imposed stretch loads (Komi, 2000). Fatigue from submaximal SSC exercises has also been shown to result in a decreased movement efficiency (Gollhofer, 1987), and a decline in maximal isometric force (Hortobagyi, 1991). Similarly, after subjects performed 50 half squats at 15%, 22% and 30% of their one repetition maximum, Hattin et al. (1989) noted that the articular force components of the knee joint were significantly altered, with a 51% increase in the anterio-posterior forces, 33% increase in the compressive forces, and 10% increase in the medial lateral shear forces.

Only one study was located examining the effects of fatigue on the external loads generated during Beach Volleyball (Edwards et al., 2001). In this study Beach Volleyball players (n = 12) were fatigued by performing a series of standing vertical jumps while wearing a weighted belt (approximately 10% of body weight). Irrespective of the fatigue condition, the Beach Volleyball players generated the same vertical ground reaction forces when landing on a sand surface after performing a spike jump movement from a 50 cm high bench (Edwards et al., 2001). However, as the subjects landed from a drop jump bench with its height held constant throughout all trials, the authors recommended further investigation of the effects of fatigue on the forces generated at landing in Beach Volleyball when the jump height is not held constant (see Section 1.2.).
2.5.4.3. *Metabolic Substrate Changes with Fatigue*

Energy in cells is released when adenine triphosphate (ATP) is hydrolysed by ATPase to release free energy to do work in the form of adenosine diphosphaste (ADP) and creatine phosphate (CP; McArdle et al., 1991; Vollestad, 1995). The energy that is released is transferred directly to other energy requiring molecules (McArdle et al., 1991). The ATP is resynthesised anaerobically through CP which forms the ATP-ADP cycle in which ATP is converted to ADP and CP, and ADP is rephosphorylised back to ATP ( Sahlin, 1992).

As ATP is essential to perform work, the metabolic substrates involved in the production of energy have been implicated as a mechanism of fatigue (Enoka & Stuart, 1992). During muscle fatigue there is a build-up of metabolites, as a result of the breakdown of ATP, such as hydrogen ions (H⁺), ammonia, and inorganic phosphates (Moritani & Yoshitake, 1998; Strojnik & Komi, 1998) as well as sodium and potassium ion shifts (Moritani & Yoshitake, 1998). These metabolic changes affect the muscle excitation-contraction coupling, including properties of the muscle membrane and propagation of the muscle action potential (Bigland-Ritchie, 1981; Matsumoto et al., 1991; Moritani, 1982; Moritani et al., 1990), and are thought to be responsible for decreased force generation by the muscle (MacLaren, 1989).

During short duration, high intensity exercise, such as during a series of SCC exercises, the accumulation of H⁺ may explain much of muscle fatigue (MacLaren, 1989), through its effect on glycolysis, the contractile process and specific important equilibrium reactions. During vertical jumps Type IIa muscle fibres are primarily recruited, which use anaerobic glycolysis as the main energy source (MacLaren, 1982). In this energy cycle, H⁺ combines with pyruvic acid to form lactic acid. If the rate of H⁺ production is greater than the rate of removal, intracellular acidosis develops (Allen et al., 1992). The build-up of lactic acid in the muscle impairs regeneration of ATP (McArdle et al., 1991) through increasing H⁺ concentration. This, in turns, inhibits lactate dehydrogenase (MacLaren, 1989) and results in a decrease in muscle pH (Allen et al., 1995; Allen et al., 1992; MacLaren, 1989) and a simultaneous decline in performance (Allen et al., 1992). The accumulation of H⁺ inhibits key enzymes in glycolysis (phosphofructokinase and phosphorylase), reducing the rate at which ATP is synthesized from anaerobic glycolysis (Hargreaves et al., 1998; MacLaren, 1989) and alters the equilibrium reaction.
of creatine kinase, resulting in faster depletion of CP. This was observed by Linnamo et al. (1998) who attributed an impaired force development to be most likely caused by a substantial increase in blood lactate concentration noted during maximum strength loading exercises performed by healthy male \((n = 8)\) and female \((n = 8)\) individuals. The impaired contraction process may be caused from the increased \(H^+\) concentration indirectly interfering with the energy cycle (Sahlin, 1995) or directly affecting the contractile apparatus or excitation-contraction coupling (Sahlin, 1995; Tesch et al., 1983). Due to the shorter recovery of force compared to the recovery of lactate and muscle \(pH\), the entire loss in force may have indirectly influenced ADP rephosphorylation (Sahlin, 1995).

Calcium is released from the sacroplasmic reticulum into the myoplasm when an action potential triggers the T-tubules (Allen et al., 1992). The troponin binding sites are filled with \(Ca^{2+}\) allowing maximal interaction between actin and myosin to occur, leading to the cross-bridge cycle and thus a muscle contraction (Allen et al., 1992). Possible mechanisms leading to a decline of force during fatigue may therefore also be due to (i) a decrease in \(Ca^{2+}\) release; (ii) a decrease in the sensitivity of the contractile proteins to \(Ca^{2+}\) due to competitive binding on the troponin binding site; and (iii) sensitivity of \(Ca^{2+}\) not being reduced but the tension achieved at saturation being reduced instead (Allen et al., 1992).

During high intensity anaerobic exercise, such as continuous vertical jumping, CP and ATP decrease significantly (Hirvonen et al., 1992), with the CP decrease playing a vital role in muscle fatigue. Kushmerick (1993, cited in Pinniger, 1996) correlated a decrease in CP concentration with a loss of force associated with muscle fatigue. The decrease in muscle force as a result of fatigue from consecutive leg extensions was not affected by glycogen depletion as only a small decrease was observed (Tesch et al., 1983).

Ninety percent of energy production during an Indoor Volleyball match is from phosphagens, ATP and CP, and anaerobic glycolysis (Tant & Greene, 1993). If a player repeatedly performs several spikes and/or blocks with only short rest intervals, the player may deplete their ATP, CP and oxygen stores, causing an increased anaerobic
glycolysis and an increased production of lactic acid (Viitasalo et al., 1987). In national and international level Indoor Volleyball, replenishment of oxygen stores and resynthesis of phosphogens occurs aerobically during the long time intervals that players experience between points (Viitasalo et al., 1987). In prolonged aerobic exercise, muscle glycogen has a more significant role in muscle fatigue than in anaerobic exercise (MacLaren, 1989). However, in Beach Volleyball there maybe a greater reliance on anaerobic metabolism, particularly the phosphogen energy system, due to the shorter rest periods for the players and the fewer number of players on each team, despite the same area of court to cover (see Section 2.2.). As a Beach Volleyball match progresses, changes in the metabolic substrates that occur as a result of fatigue may potentially increase the risk of developing patellar tendinosis by impairing the ability of the lower limb muscles of a player to effectively dissipate and/or control the impact forces sustained during repetitive landings.

2.5.4.4. **Eccentric Exercise and Muscle Damage**

Eccentric exercises can cause skeletal muscle injury as a result of the high mechanical forces that are generated by active muscles being lengthened (Clarkson & Newham, 1995; Ebbeling & Clarkson, 1989). These high forces applied to the lengthening muscle disrupt the normal myofilament structures in some sarcomeres and cause a loss of intramuscular proteins into the plasma, indicating damage to the sarcolemma (Armstrong, 1990). This muscle damage is due to relatively few motor units producing the high mechanical force in an eccentric contraction, which causes structural protein damage in the muscle fibres (Moritani & Yoshitake, 1998), and lower oxygen consumption by each active muscle fibre (Clarkson & Newham, 1995). As a result of the eccentric-based muscle damage, the muscle experiences delayed onset of muscle soreness (DOMS), which is the sensation of discomfort and pain in skeletal muscle from unaccustomed muscular exertion (Appell et al., 1992). This DOMS is usually associated with a decreased force producing capability of the muscle and a release of muscle proteins into circulation, such as creatine kinase (Ebbeling & Clarkson, 1989). Creatine kinase is a muscle protein that is used as a marker of muscle damage, as it is found almost only in muscle tissue (Ebbeling & Clarkson, 1989; Kyrolainen et al., 1998). Eccentric muscle damage from repeated eccentric exercises may therefore be an important mechanism of neuromuscular fatigue by DOMS inhibiting motor
performance of players, resulting in altered muscle mechanics, joint and muscle stiffness and reflex intervention (Komi, 2000).

As volleyball players perform numerous vertical jumps and other movements that involve a large eccentric component, such as the spike jump (Avela & Komi, 1998; Avela et al., 1996; Grabiner & Owings, 1999; Hattin et al., 1989; Horita et al., 1996; Hortobagyi, 1991; Jones & Watt, 1971; MacLaren, 1982; Nicol et al., 1996), muscle damage can occur during play. A reduction in force and DOMS may result from these pathological changes (Armstrong, 1990). For example, Horita et al. (1999) reported that, after exhaustive SSC exercise, changes in drop jump performances of male subjects occurred with a coinciding increasing in creatine kinase concentrations and a decrease in stiffness regulation and motor control (see Section 2.5.4.6.). The authors suggested that, as a result of muscle damage, the mechanical behaviour of the drop jump could be influenced by modified prelanding motor control (see Section 2.5.4.7.). Severe muscle soreness is suggested to also affect the stretch reflex (Horita et al., 1996, 1999; Nicol et al., 1996; Skurvydas et al., 2000) and stiffness regulation of the whole muscle-tendon complex (Horita et al., 1996, 1999). For example, muscle soreness sustained by the quadriceps muscles may affect the quadriceps muscles and the patellar tendon complex by decreasing the stiffness regulation and motor control of this muscle-tendon complex. This is of particular importance during the impact of landing in which the quadriceps muscles act eccentrically to control deceleration of body mass during landing. As a result of muscle soreness, the quadriceps-patellar tendon complex may be less effective in dissipating the loads sustained during landing, possibly increasing the risk of developing of patellar tendinosis.

2.5.4.5. **Motor Performance and Fatigue**

A decrease in motor performance has been associated with fatigue (Horita et al., 1996, 1999; MacLaren, 1982; Nicol et al., 1996). For example, MacLaren (1982) noted that fatigue induced by three sets of 30 consecutive vertical jumps with a 2 second delay between jumps and a 1 minute rest between sets, caused volleyball players ($n = 10$) to jump lower than when pre-fatigued. Similarly, Horita et al. (1996) and Horita et al. (1999) found that, after healthy male volunteers performed submaximal SSC exercises on a custom-designed sledge apparatus, their drop jump performance was decreased
until the second day post-exercise. Their performance tended to recover 2 hours post-exercise compared to immediately after being fatigued, and this was coupled with a secondary decline in performance two days after the exercise.

Viitasalo et al. (1993) fatigued eight skilled male volleyball players by having the players perform 60 spike jumps, block jumps and various jumping drills with a 30 second rest interval between each separate activity and continuous hurdle jumps for 45 seconds with bilateral foot contacts. Although changes as a result of fatigue were observed in the kinetics and kinematics in the concentric phase of the hurdle jump, there were no significant differences observed in the eccentric phase of the hurdle jump following fatigue. The changes in performance following fatigue were suggested to be a result of changes in the transfer of mechanical energy from the eccentric phase to the concentric phase of contact in a hurdle jump and/or motor unit recruitment and in their firing characteristics.

Komi (2000) proposed an interaction between SSC exercise induced muscle damage and performance reduction (see Figure 2-13). That is, it was thought that stiffness

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**Figure 2-13:** The proposed interaction between stretch-shortening contraction exercise induced muscle damage and performance reduction (from Komi, 2000, p.1204).
regulation changed through muscle damage altering the afferent inputs from the muscle spindles, Golgi tendon organs and Group III and IV afferent nerve endings. As a result of the muscle damage there was a reduction in the stretch reflex sensitivity, disturbing the muscle (and joint) stiffness regulation and decreasing the efficiency of the SSC performance.

2.5.4.6. **Muscle Stiffness, Co-activation, Balance and Fatigue**

Muscle stiffness is the resistance to external loads by a muscle or muscle group as it deforms (Nyland et al., 1994). Muscle stiffness during the eccentric part of the SSC is developed by the net contribution of the stretch reflexes (Komi et al., 1992) and is regulated by activation of the lower limb muscles (Viitasalo et al., 1998). Vertical jumps are dependent upon the spring-like characteristics provided by a relatively stiff muscle, aiding absorption, storage, and release of elastic energy if there is a requirement to rebound upwards after landing (Kubo et al., 1999; Nyland et al., 1994). Muscle stiffness characteristics change as fatigue progresses when the musculotendinosus system becomes less able to absorb force, resulting in less storage of elastic energy in the eccentric phase of a SSC (Komi et al., 1992). After repetitive SSC exercises there is typically a decrease in knee joint stiffness during a drop jump which has been correlated with a decrease in drop jump performance (Horita et al., 1996, 1999). Horita et al. (1999) suggested that myofibrillar disruption and/or connective tissue injury after SSC exercise could affect the stiffness regulation of the whole tendon-muscle complex (see Figure 2-14). Similarly, fatigue from marathon running has been shown to reduce the damping in a drop jump, altering the muscle stiffness characteristics and the amount of elastic energy storage in the braking phase of a drop jump (Nicol et al., 1991). Adjustments to muscle stiffness through pre-activation of the gastrocnemius muscle become more difficult to withstand as fatigue progresses and deterioration of the impact damping occurs, leading to a decrease in marathon performance (Nicol et al., 1991).

In combination with the lower limb muscles regulating muscle stiffness to resist external loads, muscles can be activated in synchrony to stabilise a joint when an external force is applied. This simultaneous activation of agonist and antagonist muscle groups, known as muscle co-activation, can stabilise a joint via the antagonist activation reducing the motive effect of the agonist muscle (Weir et al., 1998). For example, Weir
et al. (1998) revealed that at higher velocities of maximal isokinetic knee extension, co-activation of the quadriceps and hamstring muscles was greater than at lower velocities when there was less need to stabilise the knee joint. However, when fatigued, induced by 50 maximal isokinetic knee extension contractions in 10 healthy male and female subjects, co-activation levels increased but the rate of increase in co-activation was independent of contraction velocity (Weir et al., 1998). Weir et al. (1998) suggested that hamstring co-activation during isokinetic knee extension exercises may add to fatigue and detract from the quadriceps muscle torque generated during maximal knee extensions.

![Figure 2-14:](image)

**Figure 2-14:** The proposed interaction between prelanding motor command and stiffness regulation at touch down in the drop jump after the occurrence of muscle damage (adapted from Horita et al., 1999, p.166).

Fatigue has also been shown to alter balance. For example, Johnston et al. (1998) observed a significant decrease in the ability of healthy subjects ($n=20$) to balance on a Kinaesthetic Awareness Trainer balance device after being fatigued. The authors postulated that fatiguing the quadriceps muscles may inhibit the neuromuscular feedback system of the knee joint, and increase the risk of injury due to the loss of balance in fatigued individuals. However, how fatigue affects balance during landing in
volleyball players remains unknown and further investigation is warranted to examine this relationship.

2.5.4.7. **Neuromuscular Control During Landing and Fatigue**

Before contacting the ground during landing, the lower limb muscles are pre-activated in anticipation of the impeding impact (Avela et al., 1996; Jones & Watt, 1971). Pre-activation of the lower limb muscles enhances the eccentric phase of landing by optimising the timing of the muscles with respect to ground contact (Avela et al., 1996; Viitasalo et al., 1998).

An initial investigation by Jones & Watt (1971) found that a functional stretch reflex did not influence the control of landing from a single step, concluding the movement was programmed and dispatched from higher centres as a single entity, before landing; the correct timing and sequence of muscle contraction having been learned through previous experiences. This notion has been supported by several other researchers in skills such as the drop jump (Avela et al., 1996; Duncan & McDonagh, 2000; Horita et al., 1999; Viitasalo et al., 1998) and rebound jumping movements (Eloranta, 1997). Avela et al. (1996) stated that, although pre-activation was preprogrammed, it could be modified by the vestibular apparatus, proprioceptive and visual inputs if given sufficient time. The authors also noted that the earlier presentation of information of the expectant muscle load along with the lack of experience of the subjects, lead to the subjects modifying the central program to increase the stretch load through higher pre-activation levels of the lower limb muscles.

It has been suggested that pre-activation of the vastus lateralis and biceps femoris muscles can be trained to enable individuals to turn on these muscles earlier before ground contact (Viitasalo et al., 1998). This suggestion was formulated on the basis of research that demonstrated that the pre-activation of vastus lateralis (Viitasalo et al., 1998; Vittasalo & Lahtinen, 1990) and biceps femoris occurred earlier in triple jumpers compared to the control subjects (Viitasalo et al., 1998). However, no studies were located to support the notion that healthy players can be trained to alter the timing of muscle activation patterns during dynamic landing tasks.
It has been shown that fatigue results in changes to the pre-activation patterns of lower limb muscles (Arendt-Nielsen, 1991; Edwards et al., 2001; Hortobagyi, 1991; Nyland et al., 1994). For example, Arendt-Nielsen (1991) observed that the onset time for biceps femoris, semitendinosus and gastrocnemius during walking when fatigued was significantly closer to heel contact compared to a non-fatigued condition. Muscle burst duration was also significantly reduced for vastus lateralis and semitendinosus in fatigued subjects. Similarly, during a fatigued run and rapid stop movement, subjects have displayed a delayed onset of rectus femoris, vastus lateralis, biceps femoris and medial hamstrings (Nyland et al., 1994). After fatiguing 10 subjects (6 male, 4 female) using 50 maximal knee extension contractions, Wojtys et al. (1996) observed that subjects displayed significantly slower muscle responses when fatigued such that it appeared that muscle fatigue affected the dynamic stability of the knee by altering the neuromuscular response to anterior tibial translation. Based on these results, Wojtys et al. (1996) concluded that fatigue was an important part in the pathomechanics of knee injuries by decreasing the potential of the dynamic knee defense mechanisms. Hortobagyi (1991) postulated that the longer duration of muscle activity during a SSC after 50 drop jumps was related to the greater amplitude and/or slower rate of muscle stretch during the eccentric phase that enabled subjects to generate sufficient propulsive force and larger re-use of stored elastic energy.

Strojnik & Komi (1998) investigated the effects of fatigue induced by consecutive sledge jumps at 60% of maximum jump height until exhaustion in healthy male subjects (n = 12). When fatigued, the subjects showed a significant increase in the of the vastus lateralis electromyographic (EMG) activity during a drop jump, indicating an increased motor unit recruitment and synchronisation, a higher firing frequency, and/or a slower conduction velocity than when not fatigued. Similarly, the significant increase in average EMG for the rectus femoris (Nummela et al., 1994) and vastus lateralis (Viitasalo et al., 1993) during the eccentric phase of landing in fatigue was caused by changes in firing characteristics and/or the recruitment of the motor units actively firing. Even minor changes in tasks can significantly alter motor commands (Rube & Secher, 1990, cited in Enoka & Stuart, 1992). As a result of fatigue from uphill walking in soccer players (n = 10), muscle co-ordination and individual muscle activity patterns changed, with no change in the overall movement pattern of the lower limb (Arendt-
Nielsen, 1991). It was also suggested that the pattern generators that control rhythm during locomotion, obtain their input to allow the kinematics of the legs to be sustained despite fatigue (Arendt-Nielsen, 1991). However, after fatiguing 10 subjects (6 men, 4 female) using 50 maximal knee extension contractions, Wojtys et al. (1996) observed that the order of muscle recruitment (quadriceps, hamstrings, gastrocnemius) was not significantly altered by muscle fatigue. It remains unknown if neuromuscular control of landing of a typical volleyball movement changes as a result of fatigue.

Only one study was located that investigated the effects of fatigue on the technique of volleyball players landing on a sand surface. Edwards et al. (2001) examined the effects of fatigue induced by a series of weighted standing vertical jumps displayed by Beach Volleyball players who performed a drop jump landing onto a dry sand surface. The players performed the drop jumps off a 50 cm high bench after spiking a volleyball. Results revealed that the players exhibited a delay in peak rectus femoris muscle activity upon landing when fatigued (see Section 1.1.). The players also exhibited a delayed medial gastrocnemius muscle onset when fatigued. The authors speculated that this delay in medial gastrocnemius onset would reduce the time available for players to plantar flex their feet in preparation for a forefoot landing before impacting the sand, possibly reducing the ankle joint motion available to dissipate the high impact forces. The authors also suggested that this may lead to players developing higher patellar tendon forces when fatigued, thereby increasing the risk of developing patellar tendinous, although further research was recommended to investigate the association between fatigue and patellar tendon forces. A major limitation of this study, however, was the fact that the spike jump movement was performed off a standard height bench in an attempt to isolate the effects of fatigue on the landing phase of the skill. It is possible that fatigue may in fact affect jump height during the take-off phase in the spike jump movement, in turn, altering the landing phase. It is therefore unknown how fatigue may influence the muscle activation patterns during landing after performing a complete spike jump movement and, if any changes in landing influence the risk of developing patellar tendinosis.
2.6. SUMMARY

Large stresses are placed on the extensor mechanism during activities involving rapid acceleration and deceleration such as jumping and landing. In an effort to control and dissipate the impact forces of landing, the lower limb muscles must act together to decrease the load sustained by the extensor mechanism. It is this high and repetitive loading in sports that involve repetitive jumping that has lead to an increased incidence of patellar tendinosis in sports such as Beach Volleyball. Fatigue is known to affect the lower limb muscle recruitment strategies used during landings. However, despite the prevalence of patellar tendinosis in volleyball, there has been limited research into how fatigue may affect the ability of the lower limb muscles to dissipate the loads sustained during landing of a typical Beach Volleyball movement, and whether this may increase the risk of developing patellar tendinosis by increasing the repetitive shock or stress transferred to the patellar tendon. Only one study was located which has investigated the effect of fatigue on the synchrony of lower limb muscle activation patterns during landing of a modified spike jump movement (Edwards et al., 2001). However, in this study, the peak jump height attained was held constant in the modified spike jump movement. It is therefore not known whether fatigue would cause similar effects if the peak jump height was not held constant. Therefore, the primary purpose of this study was to examine the effects of fatigue on landing performance during a typical volleyball movement in which the peak jump height was not held constant, and to gain further understanding of the relationship among fatigue, lower limb landing mechanics and possible extrinsic factors that contribute to the risk of developing patellar tendinosis.
3.1. SUBJECTS

Fourteen experienced male Beach Volleyball and/or indoor volleyball players (mean age = 26.5 ± 5.6 years; height = 181.2 ± 5.0 cm; mass = 79.4 ± 13.7 kg) were selected to participate as experimental subjects in the study. As subjects acted as their own controls, no control subjects were required. Subjects were recruited through the South East Volleyball Association within the Illawarra, New South Wales, and from athletes within the University of Wollongong student population. Ethical clearance for the study was obtained from the University of Wollongong Human Research Ethics Committee (see Appendix 3.1) and all testing was conducted according to the National Statement on Ethical Conduct in Research Involving Humans (2000). Each subject completed a Subject Information Package (see Appendix 3.2) that outlined the testing protocol, a Consent Form (see Appendix 3.3), and a Subject Selection/Screening Questionnaire (see Appendix 3.4) before data collection commenced. The latter questionnaire provided information pertaining to the current activity levels and previous injuries sustained by each subject. It was also used to assist screening potential subjects based on the following subject selection criteria:

1. currently playing Beach and/or Indoor Volleyball at local level or above;
2. aged between 20 and 40 years; and
3. no history of major lower limb injury or disease.

Subjects were selected from Beach and Indoor Volleyball as these sports require players to perform numerous vertical jumps throughout a game. It is this intense and repetitive jumping that has been associated with the high incidence of patellar tendinosis observed in these sports (see Section 2.8.5.1.). All subjects were required to be currently competing in Beach and/or Indoor Volleyball at local level or above so they were proficient in performing the experimental movement tasks. Only male subjects were recruited as neuromuscular control during landing is gender dependent (Cowling & Steele, 1999). Players aged between 20 and 40 years old were selected as above this age range tendons degenerate with increasing age (Kirkendall & Garrett, 1997), and below this age range players may not have reached physical maturity. To minimise
factors that may have confounded results in this study, players with major lower limb injury(s) or disease(s) were excluded from participating.

Fourteen subjects were recruited for the study in order to gain a statistical power of 80% and to restrict Type II errors (Bach & Sharpe, 1989). To estimate statistical power, mean and standard deviation data were calculated for between condition differences in muscle activity, kinematic and kinetic variables based on data obtained from Edwards et al. (2001).

3.2. EXPERIMENTAL PROTOCOL
The experimental protocol used in the present study is outlined in Figure 3-1. Before testing, each subject’s dominant landing limb was defined by observing the limb that contacted the ground first during landing after the subject had executed a preliminary volleyball spike jump movement. Typically, this was the limb on the opposite side of the subject's body to the hand that contacted the ball. Height and body mass were then collected (see Section 3.3.1.) after which subjects were prepared for kinematic data collection (see Section 3.3.3.) and EMG data collection (see Section 3.3.4.). A resting blood lactate sample was then obtained and the value recorded (see Section 3.3.6.) before subjects were familiarised with the movement tasks. The total experimental protocol took approximately 2.5 hours per subject. All testing was conducted in the Biomechanics Research Laboratory, Department of Biomedical Science, University of Wollongong.

3.2.1. Movement Tasks
The subjects completed two main experimental tasks:

(1) a drop jump movement (DJM); and

(2) a spike jump movement (SJM).
Methods

Informed consent

Determination of the dominant landing limb

Height & body mass assessment

Subject preparation:
- EMG
- OptoTrak
- Blood lactate

Subject familiarisation of movement tasks

3 x body weight trials

Maximum SVJ* & SVJW**

5 x DJM†

5 x SJM‡

Fatigue Protocol 1
= 4* x 30 SVJW
+ 1 x 30 SVJ

5 x DJM

5 x SJM

Fatigue Protocol 2
= 2-3* x 30 SVJW
+ 1 x 30 SVJ

5 x DJM

5 x SJM

Blood lactate sample

Figure 3-1: Flow chart of the experimental protocol.

*SVJ = Standing vertical jump.
**SVJW = Standing vertical jump with weight belt.
†DJM = Drop jump movement.
‡SJM = Spike jump movement.

*A SJM was chosen as the main experimental task as 25% of Beach Volleyball injuries occur during spiking (Aagaard et al., 1997). Although more injuries occur during field defence (33%; Aagaard et al., 1997), no one typical movement can be defined to
represent field defence (see Section 2.2.). Furthermore, Edwards et al. (2001) has previously found that fatigue from a series of weighted standing vertical jump sets altered the landing action of Beach Volleyball players, when landing onto sand surface (see Section 2.5.4.7.). However, in this previous study the bench height was held constant and it is not known if fatigue would directly alter the landing component of a complete SJM (see Section 1.1).

3.2.2. Experimental Setup

In both the SJM and the DJM, a Beach Volleyball net was set at regulation height, 2.43 m above a sand surface. A regulation beach volleyball, secured to the ceiling within a plastic bag, was positioned just above the volleyball net (see Figure 3-2). The exact position of the beach volleyball was modified according to each subject’s height, personal striking preference and lower limb dominance. To replicate a suitable Beach Volleyball surface, a bottomless wooden frame (1010 mm x 1010 mm x 190 mm) was then placed on top of a Recopol surface, which directly covered a force platform. A “sand box” was formed by filling the bottomless wooden frame with wet sand (Kurnell sand, Sydney), ensuring an approximate sand depth of 190 mm (see Figure 3-3).

3.2.3. Experimental Procedure

The SJM was performed by each subject jumping initially upwards from the sand surface, hitting the Beach Volleyball across the net and then landing on their dominant landing limb back onto the sand surface (see Figure 3-2). For the DJM, a drop jump bench approximately 0.52 m above the floor surface (0.33 m above the sand surface) was placed next to the sandbox (see Figure 3-3). The subject stood on top of the drop jump bench and stepped off the box to hit the Beach Volleyball positioned above the net on the downward flight from the drop jump bench and then land on the sand surface, dominant limb first. This DJM simulated the landing component of the SJM without the take-off phase, thereby enabling the height above the sand to be standardised.
Methods

During each DJM and SJM trial, an assistant stood on the opposite side of the Beach Volleyball net and displayed a different number of fingers to the subject. While landing, the subject had to call out the number of fingers being displayed by the assistant as a strategy to prevent the subject targeting the force plate.

Before data collection, each subject was familiarised with the experimental protocol and the movement tasks to ensure the movement recorded during testing was characteristic of how the subject would typically perform a SJM or a DJM. Following familiarisation the maximum standing vertical jump height was then determined (see Section 3.3.3.). This was repeated with the subject wearing a weight belt which had a mass equivalent to approximately 10% of the subject’s body mass. This mass was chosen based on extensive pilot testing undertaken by Edwards et al. (2001).

To obtain data which was representative of the two experimental tasks, five successful trials of both the DJM and the SJM were then completed. The order of the movement
trials was counterbalanced to avoid any order effects. Following these trials the subjects were fatigued.

Figure 3-3: Drop jump bench set up for a subject who landed initially on their left lower limb.

3.2.4. Fatigue Protocol
The fatigue protocol consisted of two phases. The first phase involved the subjects completing approximately four sets of 30 standing vertical jumps (SVJ; 30 second rest interval between sets) with their hands placed on their hips and while wearing a weight belt (~10% of body mass; see Figure 3-4). The SVJ movement was standardised as variations in jumping strategy can influence muscle function (see Section 2.5.3.1.). The weight belt was included in the fatigue protocol, that is a standing vertical jump weighted (SVJW), as it was identified, via extensive pilot testing, as an efficient way to sufficiently fatigue the subjects. The SVJW sets continued until each subject reached a 25-30% decrement in their maximal standing vertical jump height after which a final set
of 30 SVJ was performed without the weight belt. Immediately following the fatigue tasks the subject performed either five DJM or five SJM in rapid succession.

The second phase of the fatigue protocol involved the subject repeating the first phase of the fatigue protocol. However, the subjects performed a reduced number of weight belt sets (~2-3 sets) until they achieved the same 25-30% decrement in maximal standing vertical jump height. Again, a final set of SVJ without the weight belt was then performed. Immediately after the second phase of the fatigue protocol, each subject performed either five DJM or five SJM (that is, the movement task that was not performed following the first fatigue protocol) in rapid succession. The order of the DJM and SJM performed after each fatigue protocol was counterbalanced to avoid any order effects. Lastly, a post-fatigue blood lactate sample was taken and the value recorded (see Section 3.3.6.).
3.3. DATA COLLECTION

3.3.1. Height and Body Mass Assessment

The height of each subject was measured to the nearest millimeter (mm) using a Seco stadiometer (Model 220, Germany) with a spirit level attached to the stadiometer arm to ensure it was horizontal. Body mass was measured to the nearest 0.1 kg using Colonial scales (UWE, BW150, Australia). Both instruments were calibrated before testing. Three height and three mass measurements were recorded and the averages calculated as descriptive measures of body stature for each subject. To ensure that the measurements recorded were accurate and reliable, the same experienced researcher assessed all physical measurements. Furthermore, reliability of the anthropometric data was established prior to testing by measuring the height and mass of six subjects over three consecutive days, as per the testing protocol. These data were then analysed using a one way repeated measures analysis of variance (ANOVA) to calculate intraclass correlation coefficients (ICC, Vincent, 1995). ICC values of $R_1 = 0.954$ and $R_1 = 0.993$ were established for body mass and height, respectively, confirming the results were reliable.

3.3.2. Ground Reaction Force Data Collection

During each landing, the ground reaction force data were collected using a 600 mm x 400 x 61 mm Kistler multichannel force platform (Type 9281B) in conjunction with a multichannel charge amplifier (Type 9865A, Kistler, Switzerland). The amplifier gains were set at 50,000 picacoulombs (pC) for both the vertical and mediolateral/anteroposterior force channels. The force platform was placed on four steel mountings embedded in a concrete pedestal below ground level. The platform was covered with a 10 mm Recopol surface to be level with the surrounding floor. A sand box was then placed over the force platform to replicate a suitable Beach Volleyball surface (see Figure 3-2). Before the first set of movement trials, three body weight readings were recorded for each subject. This entailed each subject stepping onto the sand in an area marked directly above the force platform, standing in a relaxed manner for 7 s to establish baseline body weight and electromyographic data (1000 Hz). The baseline body weight data were used later to normalise the force data relative to each subject's body weight. The force platform was calibrated before testing commenced,
using a torque wrench and spirit levels, and the amplifiers were zeroed to account for the sand mass on top of the platform.

During each trial the three orthogonal components and the point of application of the ground reaction forces were sampled in Newtons (N) over 5 s at 1000 Hz. The location of the imprint of the dominant landing foot in the sand was observed to ensure that the subject landed over the force platform situated underneath the sand box*. Trials in which the subject landed either on the edge or outside of the force platform area were classified as unsuccessful and the trial repeated. Data recorded from the four vertical force channels, two mediolateral force channels and two anteroposterior force channels were then saved to later generate force time curves in order to determine the magnitude and timing of the peak ground reaction forces (F_R) generated during each landing.

3.3.3. Kinematic Data Collection

Kinematics of the two main experimental tasks, the DJM and the SJM, and the fatigue protocol movements, SVJ and SVJW, of each subject were recorded from a lateral aspect using an OptoTrak 3020 motion analysis system (Northern Digital, Canada). This precalibrated optoelectric measurement system consists of three one-dimensional charged couple device (CCD) cameras mounted in a Position Sensor that detect and track the position in space of infrared light emitting diode (IRED) active markers (OptoTrak, 1995). The purpose of collecting kinematic data during the two main experimental tasks was to determine whether there were any differences in jump height or body segment alignment displayed by the subjects as a consequence of fatigue. During the SVJ and SVJW task, kinematic data were collected to assess the level of subject fatigue.

*It is acknowledged that not all the force generated during landing was recorded by the force platform as the sand box was not entirely isolated over the force platform. However, this limitation was deemed necessary to minimise the risk of injury to each subject whilst performing the SJM and DJM on the sand box. Extensive pilot testing results indicated the setup was valid in terms of enabling comparisons of forces between conditions, given the within subject design of the study, if each subject’s foot position was closely monitored to ensure subjects landed directly above the force platform.
Methods

The active IRED markers were attached to the skin of each subject’s dominant landing limb over the following locations:

(1) the head of the 5th metatarsal;
(2) the lateral malleolus of the fibula;
(3) the lateral condyle of the femur; and
(4) the superior portion of the greater trochanter of the femur.

These marker locations were selected to enable later computation of foot, leg and thigh segmental alignment and lower limb joint angles. To avoid losing view of the IREDs by the Position Sensor, the subjects wore minimal clothing (a t-shirt and brief shorts). Socks and sports shoes were worn during the fatigue protocol to minimise potential injury but they were removed during the SJM and DJM trials.

The IREDs were attached to each subject’s dominant landing limb with double-sided tape. The IREDs were then connected via wires to a strober unit. The strober unit (57 mm x 77 mm x 24 mm, 94 g) activated the IREDs at 7 volts at a 50% dynamic duty cycle. Before recording the movement trials, the markers were observed in Real Time Display to ensure that all markers functioned properly and were in the Position Sensor’s field of view.

The Position Sensor was positioned approximately 2.2 m and 3.25 m away from the sand box and force platform, respectively, perpendicular to the volleyball net and the plane of progression of the tasks. The Position Sensor sequentially (multiplexed) sampled the location of each IRED at 500 Hz. Both the strober unit and the Position Sensor were connected to the System Control Unit (SU-05427) via separate cables. The System Control Unit acted as the OptoTrak’s central controlling, interfacing and processing unit, linking the host computer with the Position Sensor and external data sources through the OptoTrak Data Acquisition Unit II (ODAU II; OptoTrak, 1992). Kinematic data were then recorded for 5 s for each SJM and DJM trial, and for 20 s during each SVJ and SVJW set, on an MCTPRO3 personal computer using Collect (Version 1.0.2.) software. To ensure that optimal kinematic data were recorded, the position and settings of the OptoTrak system were extensively pilot tested.
During the fatigue protocol, the greater trochanter IRED marker of each subject was tracked using the OptoTrak system while the subjects performed four maximal SVJ with and without the weight belt (~10% of their body weight). The purpose of tracking the greater trochanter IRED marker was to approximate the height the subject jumped when performing a SVJ. Traditional methods of assessing vertical jump height using a force platform during the SVJ and SVJW tasks, were not feasible due to the inability to readily move the sand box covering the force platform. The greater trochanter marker was also tracked during the fatigue protocol to assess whether a subject had reached a 25-30% decrease in maximal SVJ height (see Section 3.4.1.).

3.3.4. Electromyographic Data Collection

Electromyographic (EMG) data were collected during each SJM and DJM for the muscles rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), biceps femoris (BF), semitendinosus (ST) and the medial head of gastrocnemius (MG; see Figure 3-5). These muscles were chosen based on their the functions during a dynamic landing (see Section 2.4.3.). Additionally, these six muscles were also analysed by Edwards et al. (2001) in a study examining muscle activation patterns displayed by Beach Volleyball players when performing a DJM in both a non-fatigued and fatigued condition, on soft non-compacted and wet compacted sand surfaces.

Figure 3-5: Lower limb muscles monitored during each trial. For all six muscles, the electrodes were aligned parallel to the underlying muscle fibre direction.
The activity of each muscle was detected using surface electrodes as they are the most appropriate method to monitor electrical activity of superficial musculature during dynamic tasks (Winter et al., 1994). To standardise electrode placement across all subjects, the following procedures were used:

1. **RF**: a line representing the line of action of RF was drawn from the groin line to the superior border of the patella on the anterior aspect of the thigh. The muscle belly was then palpated as the subject extended the leg at the knee against resistance.

2. **VL**: a mark was made 50% of the distance from the RF electrode site to the superior border of the patella and then 50% of the distance from this mark on the anterior aspect of the thigh to the most lateral aspect of the thigh. The muscle belly was then palpated in this site as the subject extended the leg at the knee against resistance.

3. **VM**: a mark was made 50% of the distance from the VL site to the superior border of the patella and 50% of the distance from the middle of the anterior aspect of the thigh to the most medial aspect of the thigh. The muscle belly was then palpated as the subject extended the leg at the knee against resistance.

4. **BF**: a line was drawn from the ischial tuberosity to the lateral epicondyle of the tibia. The muscle belly was then palpated as the subject flexed the leg at the knee against resistance.

5. **ST**: a line representing the line of action of ST was drawn from the ischial tuberosity to the medial epicondyle of the tibia. The muscle belly was then palpated as the subject flexed the leg at the knee against resistance.

6. **MG**: the muscle was palpated at the point of greatest girth while the subject plantar flexed the foot.

To reduce impedance between the skin and electrodes (Basmajian et al., 1985), hair clippers (Breville, Australia) followed by a disposable razor were used to remove hair from each electrode site. The skin was then abraded with One-Step Prep Tape (3M, Germany) to remove the dead skin, and then cleansed with a 50:50 water:alcohol solution to remove oil from the skin. The site was then dried using an electric hair dryer (Kambrook Creations 1200, Australia) before applying the electrodes. Bipolar
silver:silver-chloride surface electrodes (3M Red Dot infant disposable, diameter = 32 mm, detection area = 15 mm) were placed longitudinally on each muscle belly with an inter-electrode distance of 20 mm. A common reference electrode was located on the medial tibial condyle of the dominant lower limb. Together, the purpose of the skin preparation procedure and the surface electrode placement was to maximise the fidelity of the desired EMG signal while minimising cross-talk between EMG channels.

At each electrode site, the electrical impedance levels of the skin were recorded using a CardioMetrics Artifact Eliminator® (Model CE01, Australia). Impedance levels greater than 6 kΩ were classified as unsatisfactory and the electrode site was re-prepared following the procedure previously described (see Figure 3-6). The fidelity of the EMG signals from all six muscles was confirmed prior to testing.

Figure 3-6: Re-preparing the VL electrode site after recording a noisy baseline value.

The EMG signals were relayed from the bipolar electrodes via 1.23 m leads to the Noraxon Telemyo 8/16 Transmitter which was powered by a 9 V battery. The amplifier card, housed within the transmitter, was set for an input level of ± 6.8 mV for each channel. The leads were taped to the subject’s skin to minimise movement artifact during the landing tasks, and the two unused leads were short-circuited to prevent cross-
Methods

talk. Using an adjustable belt, the transmitter (Noraxon, USA; 0.96 g) was firmly fixed around the subject’s waist throughout the SJM and DJM. However, during the fatigue protocol, although the leads remained attached to the electrodes, the transmitter was removed from the subject’s waist and an assistant, standing next to the subject, held the transmitter whilst the subject performed the series of SVJ with the weight belt fixed about their waist. At the end of the last weight belt series, the weight belt was removed and the transmitter was firmly fixed again to the subject’s waist. This was performed to avoid any damage to the transmitter during the fatigue protocol by the weight belt.

Signals sampled (1000 Hz) at a frequency bandwidth of 0 to 340 Hz (Noraxon, 1992), were sent from the transmitter via an aerial to the Telemyo receiver and stored on a MCTPRO3 personal computer using Collect (Version 1.0.2) software (see Section 3.3.5). Before testing, data from each channel was visually inspected to ensure minimum movement artifact had occurred, a clear meaningful signal was being received and the electrode placement site was correct. The purpose of recording the EMG data was to determine whether there were any differences in muscle activation patterns and muscle intensity displayed by the subjects during the DJM and SJM, in the non-fatigued and fatigued conditions.

3.3.5. Data Synchronisation

Both the ground reaction force and EMG data were collected via an OptoTrak ODAU II unit (DAU-02051) and relayed through to the System Control Unit (SU-05427) to be recorded on an MCTPRO3 personal computer using Collect (Version 1.0.2) software. This allowed the kinematic data to be simultaneously triggered and synchronised with the force and EMG data (OptoTrak, 1999). The raw data were then automatically stored under a designated file extension after completing each trial. The EMG data were converted from an integer form into volts, using the Collect (Version 1.0.2.) software. The file type of the kinematic, force and EMG data was then converted from a floating point format to an ASCII format using the FL-ASCII program in the Data Analysis Package (DAP, OptoTrak, 1992) software and then saved as a comma delimited file (CSV) in Excel for further processing (see Section 3.4.).
3.3.6. Blood Lactate Data Collection

To confirm the level of player fatigue, blood samples were taken from each subject to test for blood lactate levels at rest and post the second fatigue protocol. Following Department of Biomedical Science Guidelines, blood was obtained by cleansing the tip of the subject's index finger or the ear lobe with an alcohol swab. A reagent strip (Lactate Test Strips, Boehringer Mannheim) was then inserted into an Accusport blood lactate analyser. The surface of their fingertip was then pricked using a disposable auto-lancet (see Figure 3-7). Using a capillary tube, a droplet of blood was collected. The Accusport analyser was then opened and the droplet of blood was immediately expressed onto the reagent strip. The Accusport was closed and after 60 s the blood lactate level was recorded. This method of blood collection has less than a 5% technical error of measurement with blood lactate levels ranging from 2 nmol/L to 17 mmol/L (Gulbin, 2001).

Figure 3-7: Collecting a blood lactate sample from the ear lobe of a subject.

3.4. DATA ANALYSIS

3.4.1. Fatigue Analysis

Fatigue was defined in the present study as an impairment of performance which was accompanied by an increased perceived sense of effort indicated by an inability of the subjects to attain 70-75% of their maximum vertical jump height. Vertical jump height was represented by vertical displacement of the greater trochanter IRED.
measurement was considered to approximate changes in the vertical displacement of the total body centre of gravity of the subject.

Decrement in vertical jump performance were monitored both in the SVJW sets in which the subjects wore the weight belt and in the SVJ sets without the weight belt. During the SVJW sets of the fatigue protocol, greater trochanter motion was continually assessed after each SVJW set and continued until a 25-30% decrement in standing vertical jump height was achieved by the subject on approximately the last five jumps in the set with the weight belt, while the greater trochanter motion was again monitored. A final set was then performed without the weight belt. The lower limb fatigue with weight belt was determined using the following equation:

$$\text{Lower Limb Fatigue With Weight Belt} = \frac{\text{average SVJW (fatigue set)}}{\text{maximum SVJ (pre-fatigue tasks)}} \times 100$$

Equation 3-1

Lower limb fatigue without weight belt was defined as the percentage decrease in maximal SVJ height, performed without the weight belt, and was calculated to represent the overall lower limb muscle fatigue. The percentage was determined by the following equation:

$$\text{Lower Limb Fatigue Without Weight Belt} = \frac{\text{average SVJ (fatigue set)}}{\text{maximum SVJ (pre-fatigue tasks)}} \times 100$$

Equation 3-2

To confirm the level of fatigue, blood lactate samples were also taken pre- and post-fatigue (see Section 3.3.6.).

3.4.2. Ground Reaction Force Data Analysis

Prior to data analysis, each trial was visually inspected with any trial found to be contaminated with noise being disregarded. The data files were then further processed through PROG software (Andrews, 1996; see Appendix 3.9) to sum the four vertical, two mediolateral and two anteroposterior channels to obtain force time curves in the
three orthogonal directions. From these curves, the following variables were calculated for the five trials for each SJM and DJM (see Figure 3-8)*:

1. the peak resultant ground reaction force (FR; BW);
2. the initial peak FR (BW);
3. time of the peak FR (ms);
4. time from IC to the peak FR (ms); and
5. the rate of loading of the FR (BW.ms⁻¹).

The rate of loading was calculated by dividing the FR at the initial impact peak, or the peak FR if there was no impact peak, by the time interval between IC and the initial peak FR.

The times of IC and the peak FR were used against which to compare the relative timing of the muscle activation patterns and kinematic variables, as these times were considered as the most crucial times in the landing task. That is, loading of the dominant limb begins when the subject first establishes contact with the ground during landing and this was represented by IC. The maximum force through the subject’s body was represented by the peak FR when the resultant of the ground reaction forces generated during landing reached its maximum value. These phases of the experimental tasks are also important as they reflect the temporal characteristics of the extensor mechanism muscles acting to create normal patellofemoral function during landing (see Section 2.5.3.1.).

*Although all three orthogonal components of the ground reaction force data were initially collected and analysed, the anteroposterior force (Fₐp) and mediolateral force (Fₘₙ₁) data collected during landing are not reported in this thesis for either the DJM or the SJM. This is because the peak Fₐp and peak Fₘₙ₁ occurred either just prior to, but most frequently after, the peak Fᵥ. It was therefore not known whether these values were influenced by the second foot contacting the sand surface during the landing action. Thus, the peak values and the time that these anteroposterior and mediolateral values occurred were not reported, as they were not thought to truly represent the peak Fₐp and peak Fₘₙ₁ for the initial landing limb. Furthermore, as the experimental tasks were predominately performed in a vertical plane of motion the contribution of the Fₐp and Fₘₙ₁ were inconsequential to the FR, (approximately 6% each). Therefore, as the resultant forces reflected the characteristics of the vertical forces at landing, and the main loading of the body, only data for the resultant forces were presented in this thesis.
Methods

Figure 3-8: Typical force time curve generated at landing during a DJM.

3.4.3. Kinematic Data Analysis
In order to eliminate any gross errors in the kinematic data generated via electronic noise from the OptoTrak system (Winter, 1990), each trial was initially visually inspected. To further reduce any random errors in each trial, the data were then processed using a fourth-order zero-phase-shift Butterworth digital low pass filter (cutoff frequency for all markers $f_c = 15$ Hz) which was applied using DAP (Version 2.2) software. The cutoff frequency was calculated based on a residual analysis of the movement frequency of each of the IREDs (Winter, 1990), and using PROG software (see Appendix 3.6).

For each SJM and DJM, in both the non-fatigued and fatigued condition, the following variables were then determined using Joint Angle software (Megill, 2001; see Figure 3-9):

1. ankle joint angle (°) at IC and at the peak $F_R$;
2. knee joint angle (°) at IC and at the peak $F_R$;
3. foot angle (°) relative to the right-hand horizontal (RHZ) at IC and at peak $F_R$;
4. tibial angle (°) relative to the RHZ at IC and at the peak $F_R$;
5. ankle joint angular displacement (°) from IC to the peak $F_R$;
Methods

(6) knee joint angular displacement (°) from IC to the peak FR;
(7) foot segment angular displacement (°) from IC to the peak FR;
(8) tibial segment angular displacement (°) from IC to the peak FR;
(9) ankle joint angular velocity (°) at IC and the peak FR;
(10) knee joint angular velocity (°) at IC and the peak FR;
(11) foot segment angular velocity (°) at IC and the peak FR; and
(12) tibial segment angular velocity (°) at IC and the peak FR.

Figure 3-9: Convention for calculating segmental and joint angles.

These kinematic variables were selected for analysis to represent the alignment and motion of the lower limb at the time of foot-ground contact and at peak loading at the ground. A non-invasive method of data collection was used to quantify internal movements of the bony segments by placing external markers on the skin of each subject. It is acknowledged that this method is limited by skin motion associated with segment movement, as well by the need to approximate the joint centres during the...
initial application of the external markers (Andriacchi, 1990). Despite other methods being proposed to minimise errors due to skin marker movement, most of these methods present their own unethical and impractical reasons (Steele, 1997) and were therefore not considered suitable in the present thesis.

3.4.4. Electromyographic Data Analysis

In order to eliminate any gross errors in the EMG data generated by cross-talk or other such noise, each trial was initially visually inspected. To eliminate any movement artifact in the EMG signals, the raw signals were initially filtered using a fourth order zero-phase shift high pass filter (Winter, 1990) with a cutoff frequency of 15 Hz. The processed raw signal was then full-wave rectified and low pass filtered (20 Hz) to create a series of linear envelopes which represented each muscle burst of interest. The envelopes were again rectified to eliminate any negative values that had arisen from the filtering process. An example of a processed raw filtered MG trace and its linear envelope is shown in Figure 3-10. The low pass and high pass cutoff frequencies were determined after trialling a series of values whereby the cut-off frequencies that enabled the raw burst to retain the temporal characteristics of the raw muscle bursts, while being smooth enough for threshold analysis, were selected.

Using Digital Signal Processing (DSP) software (Andrews, 1996), the processed raw EMG signal, its linear envelope, and the associated ground reaction force time curve were visually inspected to ensure that the correct muscle bursts were being analysed and that the signal offset had been removed. To determine the onset and offset of each muscle burst, a threshold detector set at 8% of the maximum threshold of the burst of interest was applied to the linear envelope. A variety of thresholds ranging from 6 to 10% were trialled before a threshold of 8% was selected as best representing the muscle burst onsets and offsets based on visual inspection of the data. This output was then inspected along side the processed raw EMG signal and the linear envelope to ensure accuracy of the threshold assessment of the onset and offset times. The combined threshold analysis and visual inspection was performed to reduce the chance of performing a Type I error and is considered to be more reliable than just determining the muscle burst onset or offset manually (Di Fabio, 1987).
For both experimental tasks (SJM and DJM), in the non-fatigued and fatigued conditions, the following temporal variables were analysed for the six muscles (RF, VL, VM, ST, BF, and MG):

(1) muscle burst duration (ms);
(2) time from onset of muscle activity relative to IC (ms);
(3) time from offset of muscle activity relative to IC (ms);
(4) time from offset of muscle activity relative to IC (ms); and
(5) time of peak muscle activity relative to the peak $F_R$ (ms).

The average of five trials per condition for each variable was then calculated as the representative value for a subject. After the temporal variables were determined, the intensity of muscle activity was calculated by integrating the muscle burst of interest 50 ms either side of the peak muscle activity using PROG software (Andrews, 1996) and
then normalising the muscle burst intensity as a percentage of the integrated muscle burst intensity in a non-fatigued SJM for all the six lower limb muscles.

The purpose of the EMG analysis was to determine whether the muscle activation patterns or the muscle intensity displayed during the two experimental tasks were affected by fatigue. The times of IC and the peak FR were assessed because these phases of the experimental tasks reflect important temporal characteristics of the extensor mechanism muscles acting to create normal patellofemoral function during landing (see Section 3.4.2.).

3.5. STATISTICAL ANALYSIS
Mean and standard deviations were calculated for each ground reaction force, kinematic and EMG variable for the two main experimental tasks (DJM and SJM) during each fatigue condition (non-fatigued and fatigued). The normality of the estimated underlying population and the distribution of all the variables were assessed before analysis using a Kolmogorov-Smirnov test and a Levene Median test, respectively.

The ground reaction force, kinematic and EMG variables were then analysed using a repeated measures two-way ANOVA design with two within factors (movement task and fatigue condition). The purpose of this design was to determine whether there was any significant \( p \leq 0.05 \) differences in the kinematic, ground reaction force and muscle activity data between the two movement tasks, with and without fatigue. Where a main effect in movement task or fatigue condition was found, \textit{post hoc} analysis of the data was conducted using a Tukey \textit{post hoc} test. Blood lactates were analysed using a paired \( t \)-test to determine if there was any significant \( p \leq 0.05 \) difference between pre- and post-fatigue blood lactate concentrations. All statistical procedures were conducted using Sigmastat Statistical Software (Version 2.03).
Chapter 4
Results & Discussion

4.1. FATIGUE ANALYSIS
To ensure subjects in the present study were truly fatigued, the percentage reduction in each subject’s standing vertical jump height was monitored, both during the sets in which they were wearing the weight belt (SVJW) and when the subjects were not wearing the weight belt (SVJ), the results of which are presented in Table 4-1. It is evident from these data that the subjects did achieve the desired level of fatigue immediately prior to performing both experimental tasks. Independent paired t-tests also revealed that there was no significant difference in the level of fatigue that the subjects incurred immediately before the SJM and the DJM, either when wearing the weight belt or without the weight belt. This confirmed that the fatigue protocol was successful in ensuring that the subjects were fatigued to the same extent when performing the two experimental movements.

Table 4-1: Means and standard deviations for the percentage reduction in the vertical displacement of the greater trochanter leg marker displayed by the subjects (n = 14) in the spike jump movement (SJM) and the drop jump movement (DJM) after the fatigue sets.

<table>
<thead>
<tr>
<th>Type of Fatigue</th>
<th>SJM</th>
<th>DJM</th>
<th>t</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower Limb Fatigue with Weight Belt (%)</td>
<td>28 ± 11</td>
<td>28 ± 8</td>
<td>-0.295</td>
<td>0.773</td>
</tr>
<tr>
<td>Lower Limb Fatigue without Weight Belt (%)</td>
<td>23 ± 12</td>
<td>22 ± 11</td>
<td>0.657</td>
<td>0.523</td>
</tr>
</tbody>
</table>

The post-fatigue blood lactate levels were also significantly higher than the pre-fatigue blood lactate levels (see Figure 4-1; \( t = -6.04; p <0.001 \)). These findings are consistent with the results of Skurvydas et al. (2000) who observed a significant increase in blood lactate concentrations after subjects performed 100 continuous maximal jumps. Furthermore, Chamari (2001) observed a significant increase in the blood lactate...
Results & Discussion

Figure 4-1: Blood lactate levels pre- and post-fatigue (*indicates a significant difference between the two conditions).

concentration collected for players after they performed only six consecutive maximal jumps. The increase in blood lactate concentration displayed by subjects in the present study when fatigued was thought to be a result of an increase in H⁺ concentration which, in turn, caused a decrease in muscle pH. An accumulation of H⁺ can impair muscle performance through its effect on glycolysis, the contractile process and specific important equilibrium reactions (see Section 2.5.4.3). Therefore, it was assumed that the decline in standing vertical jump performance displayed by the subjects in the present study, following the fatigue protocol, was influenced by metabolic factors. This notion is supported by Chamari (2001), who stated that, during brief maximal effort jumps, the accumulation of blood lactate was a result of anaerobic metabolism which was activated to produce and/or resynthesise CP. The increase in the subjects’ post-fatigue blood lactate concentrations in the present study indicated that the subjects also used anaerobic metabolism during the fatigue protocol. However, as the current subjects performed repeated sets of jumps for longer than the subjects observed by Chamari (2001), aerobic metabolism would have also contributed to the energy
produced during the present fatigue protocol as, after 2 minutes of maximal exercise, 50% of total energy is obtained from aerobic metabolism (McArdle et al., 1991). Other non-metabolic factors may have also contributed to fatigue such as subject motivation, impaired regulation of muscle stiffness and other factors discussed in Section 2.5.4. Irrespective of the mechanism of fatigue, the ability of the subjects to jump vertically in the present study was significantly impaired post-fatigue, which was the primary objective of the fatigue protocol.

4.2. GROUND REACTION FORCE DATA

The means and standard deviations of the resultant ground reaction forces (FR) generated at landing by the subjects in each experiment condition are included in Table 4-2.

Table 4-2: Means and standard deviations for the peak resultant ground reaction forces (FR) generated by the subjects (n = 14) at landing in a spike jump movement (SJM) and a drop jump movement (DJM) when non-fatigued (NF) and fatigued (F).

<table>
<thead>
<tr>
<th>Variable</th>
<th>SJM NF</th>
<th>DJM NF</th>
<th>SJM F</th>
<th>DJM F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak FR (BW)</td>
<td>5.17 ± 1.40</td>
<td>6.33 ± 3.43</td>
<td>4.76 ± 1.22</td>
<td>5.40 ± 1.41</td>
</tr>
<tr>
<td>Time to Peak (ms)</td>
<td>54 ± 15</td>
<td>42 ± 9</td>
<td>58 ± 12</td>
<td>41 ± 8</td>
</tr>
<tr>
<td>Rate of loading (BW.s⁻¹)</td>
<td>9.46 ± 4.31</td>
<td>10.97 ± 4.80</td>
<td>7.62 ± 2.53</td>
<td>10.76 ± 3.08</td>
</tr>
</tbody>
</table>

Interestingly, there was no one typical type of landing pattern displayed by the subjects for either the SJM or the DJM according to the landing types classified by Nigg (1983; see Figure 4-2). That is, the type of landings displayed by the subjects changed within a particular movement (SJM or DJM), within a condition (NF or F) and/or between subjects. This finding was in contrast to Richards (1996), who stated that a SJM landing performed by skilled male Indoor Volleyball players onto a rigid surface was characterised by an initial peak followed by a second larger peak, typical of a Type 1 landing. The different types of ground reaction force time curves displayed by the volleyball players in the present study may reflect the unstable nature of the sand surface, which may have caused the different landing types. That is, even though the sand box was raked before each trial to ensure a level landing surface, the compliance of the sand surface allowed each subject’s foot to penetrate the sand anywhere between 1
cm to 10 cm during landing. Variations in the depth that a subject's foot impacted into the sand may, in turn, have affected their foot motion during the landing action for a given movement trial, thereby influencing the ground reaction force time curves.

**Figure 4-2:** The three predominant types of ground reaction force patterns displayed by the subjects ($n = 14$) during landing.

Irrespective of variations in the patterns of the ground reaction force time curves displayed at landing, the mean peak $F_R$ generated by the subjects during the SJM were similar to values reported in previous landing studies. For example, Adrian & Laughlin (1983) reported a similar peak $F_R$ of 4.8 BW generated by female intercollegiate-level Indoor Volleyball players performing a SJM landing onto a rigid surface. Furthermore,
Richards et al. (1996) reported that elite male Indoor Volleyball players displayed peak vertical ground reaction forces ($F_v$) during a SJM landing on a rigid surface of $5.55 \pm 0.55$ BW and $6.00 \pm 0.50$ BW for the subjects' right and the left landing limbs, respectively. These slightly higher peak $F_v$ in the study of Richards et al. (1996), relative to the present mean $F_R$ data, may be explained by the different landing surfaces and/or variations in the skill level of the subjects between the studies. That is, Richards et al. (1996) observed elite Indoor Volleyball players, who typically landed from a vertical height zenith above the ground of 1.0 m during the SJM. In contrast, the skilled, although not elite, players in the present study typically jumped from a lower peak height (see Section 4.3). Due to the impulse-momentum relationship, a player who jumps higher in the take-off phase of a SJM will be required to generate a greater vertical ground reaction force at landing to change their momentum than a less skilled player whose peak vertical jump height is lower, assuming that the time over which this force is applied is constant. Therefore, more skilled players who attain a higher peak vertical jump height will generate higher vertical forces at landing. This notion was supported by Adrian & Laughlin (1983) who illustrated that when players performed volleyball movements in which they achieved a lower jump height compared to a SJM, lower peak $F_v$ were generated. Attaining lower jump heights in this present study may have been caused by the subjects finding it more difficult to propel themselves from the non-rigid sand surface at take-off. That is, the subjects had to exert more force against the sand surface to jump the same vertical height compared to jumping from a more rigid wooden or concrete surface. Bishop (2001) also found that the vertical jump height of Beach Volleyball players was lower when the players took off from sand surfaces compared to wooden surfaces. The author attributed this decrease in vertical jump height to the sand absorbing almost 100% of the energy generated by the players at take-off (see Section 2.5.3.1.).

None of the previous published studies investigating volleyball landings have reported the time to the peak $F_R$ or the rate of loading displayed by the subjects during landing against which the present data could be compared. However, studies examining the landing phase of non-volleyball movement skills have reported values which are similar to the present data in terms of the times to the peak $F_v$ during a drop jump landing.
(Kovacs et al., 1999; McNitt-Gray et al., 1993; see Section 2.5.3.1.), and a similar rate of loading in a single-limb netball landing (Steele, 1997).

4.2.1. Effect of Movement Task on the Ground Reaction Forces

F-ratios and p-values derived for each source of variance for the ground reaction force variables are presented in Table 4-3. A significant main effect of movement task was noted on all three ground reaction force variables when the data were pooled across the fatigue conditions (see Table 4-3). Post hoc analysis indicated that during a DJM, subjects generated a higher mean peak FR (5.86 ± 0.79 BW SEM; $q = 3.455; p < 0.030$), took less time to reach the peak FR (42 ± 2 ms SEM; $q = 8.275; p < 0.001$) and had a higher mean rate of loading (10.86 ± 0.70 BW.s$^{-1}$ SEM; $q = 3.344; p < 0.034$) than during a SJM (peak FR = 4.97 BW SEM; time to the peak FR = 56 ± 2 ms SEM; rate of loading = 9.19 ± 0.70 BW.s$^{-1}$ SEM). That is, during a DJM landing, the subjects experienced a higher peak FR over a shorter time interval and with a higher rate of loading. Excessive loading combined with high repetitions of these loads on the body is considered to be the main extrinsic risk factor associated with patellar tendinosis (see Section 2.5.3.1.). Therefore, it is postulated that subjects may potentially increase the risk of developing patellar tendinosis when performing a DJM compared to the SJM, due to the inability to efficiently dissipate the high, rapidly applied loads to the lower limb. This notion of higher ground reaction forces increasing the risk of developing patellar tendinosis is supported by Richards et al. (1996), who found that a significant predictor of patellar tendinosis in elite male volleyball players ($n = 10$) was a larger $F_v$ at landing.

Table 4-3: F-ratios and p-values derived for each source of variance for the peak resultant ground reaction forces ($F_R$) generated during landing.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Movement</th>
<th>Condition</th>
<th>Movement x Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$F_{(1,13)}$</td>
<td>p-value</td>
<td>$F_{(1,13)}$</td>
</tr>
<tr>
<td>Peak $F_R$ (BW)</td>
<td>5.970</td>
<td>0.030*</td>
<td>3.082</td>
</tr>
<tr>
<td>Time to Peak (ms)</td>
<td>34.239</td>
<td>&lt;0.001*</td>
<td>1.020</td>
</tr>
<tr>
<td>Rate of loading (BW.ms$^{-1}$)</td>
<td>5.591</td>
<td>0.034*</td>
<td>1.627</td>
</tr>
</tbody>
</table>

* indicates a significant main effect or movement x condition interaction.
4.2.2. Effects of Fatigue on the Ground Reaction Forces

Interestingly, there was no significant main effect of fatigue on any of the three ground reaction force variables when the data were pooled across the movement conditions (see Table 4-3). That is, the subjects generated similar peak FR, at a similar rate whether fatigued or not. However, despite a lack of any significant main effect of fatigue condition on the time to the peak FR, observation of the raw data indicated that there was a trend for a longer time to the peak FR during the SJM when fatigued (54 ± 1 ms SEM) compared to when non-fatigued (58 ± 1 ms SEM). The statistical power of the ANOVA test for this comparison was very low (5.0% at \( p = 0.05 \)) and therefore these data need to be interpreted cautiously. For this reason, further research is recommended into the effects of fatigue on the rate of loading of the ground reaction forces generated during these volleyball skills.

Although there was also a trend for the subjects to display lower peak FR when fatigued, the lack of a significant main effect of fatigue on the peak FR or the time to the peak FR was in contrast to Nicol et al. (1991), who observed a decrease in the peak Fv generated by experienced endurance runners during landing in a sprint and in a “5-jump performance” as a result of fatigue from marathon running (see Section 2.5.4.2.). Nicol et al. (1991) suggested that this decrease in peak Fv as a result of fatigue may have been caused by changes in the subjects’ muscle stiffness characteristics. However, Grant et al. (1996) noted that when basketball players were fatigued after participating in a 40 minute basketball training session, players generated significantly higher Fv at landing in two different rebounding tasks. The difference in the fatigue effects observed between these studies may be related to variations in the vertical jump height attained by the subjects during the experimental task performed in each of these studies. That is, the trend for a decrease in peak FR during landing following fatigue in the SJM in the present study, can be explained by the subjects achieving a lower vertical jump height (see Section 4.3.). Even though a trend towards a decrease in the peak FR was also observed in the DJM when fatigued compared to non-fatigued, the subjects displayed a wide variety of strategies to land when non-fatigued which is reflected in the high standard deviations (see Table 4-2). Irrespective of the fatigue effects, the ground reaction force variables differed significantly between the movement types, indicating
that the DJM and SJM should be considered two unique movement tasks with distinct landing patterns.

4.3. KINEMATIC DATA

The means and standard deviations calculated for the kinematic data displayed during landing for the two experimental movements are presented in Table 4-4 and Table 4-5. The values representing the peak vertical jump height attained by the present subjects during a SJM* in Table 4-4 are relatively similar to the value of 380 mm reported by Adrian & Laughlin (1983) in terms of the height that subjects raised their centre of gravity above their standing height. However, the present data suggest the subjects jumped substantially lower during the SJM than the 1,000 mm reported for elite male volleyball players by Richards et al. (1996; see Section 4.2.). However, neither Adrian & Laughlin (1983) or Richards et al. (1996) reported how the peak vertical jump height attained by the subjects during a SJM was calculated, and therefore direct between-study comparisons should be interpreted with caution. Furthermore, no volleyball studies were located in the literature that reported the time or the horizontal displacement from the peak vertical jump height attained by the subjects to IC.

Table 4-4: Means and standard deviations for the changes in the position of the greater trochanter marker from the peak vertical displacement (YMAX) and its relative horizontal position (XMAX) to the marker’s vertical (YIC) and its relative horizontal position (XIC) at initial contact (IC) displayed by the subjects (n = 14) during a spike jump movement (SJM) and a drop jump movement (DJM) when non-fatigued (NF) and fatigued (F).

<table>
<thead>
<tr>
<th>Variable</th>
<th>SJM NF</th>
<th>DJM NF</th>
<th>SJM F</th>
<th>DJM F</th>
</tr>
</thead>
<tbody>
<tr>
<td>YMAX to YIC (mm)</td>
<td>329 ± 73</td>
<td>407 ± 45</td>
<td>307 ± 76</td>
<td>434 ± 35</td>
</tr>
<tr>
<td>XMAX to XIC (mm)</td>
<td>60 ± 29</td>
<td>219 ± 47</td>
<td>62 ± 29</td>
<td>203 ± 68</td>
</tr>
<tr>
<td>Time from YMAX to IC (ms)</td>
<td>259 ± 44</td>
<td>312 ± 98</td>
<td>243 ± 37</td>
<td>313 ± 43</td>
</tr>
</tbody>
</table>

*The peak vertical jump height attained by the subjects in the present study was calculated based on the change in the vertical position of the greater trochanter marker. That is, as there was no take-off component involved in the DJM, the displacement of greater trochanter from the peak vertical height to IC was considered to be representative of the peak vertical jump height during both movement tasks.
Table 4-5: Means and standard deviations for the angular displacement and angular velocity at initial contact (IC) and at the time of peak resultant ground reaction force (peak FR) displayed by the subjects ($n = 14$) during landing in a spike jump movement (SJM) and a drop jump movement (DJM) when non-fatigued (NF) and fatigued (F).

<table>
<thead>
<tr>
<th>Variable</th>
<th>SJM NF</th>
<th>DJM NF</th>
<th>SJM F</th>
<th>DJM F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Angle at IC (°)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee</td>
<td>166 ± 6</td>
<td>171 ± 4</td>
<td>168 ± 6</td>
<td>170 ± 4</td>
</tr>
<tr>
<td>Tibial</td>
<td>83 ± 4</td>
<td>91 ± 3</td>
<td>86 ± 4</td>
<td>91 ± 3</td>
</tr>
<tr>
<td>Ankle</td>
<td>126 ± 12</td>
<td>129 ± 11</td>
<td>131 ± 11</td>
<td>129 ± 11</td>
</tr>
<tr>
<td>Foot</td>
<td>136 ± 12</td>
<td>143 ± 10</td>
<td>136 ± 11</td>
<td>141 ± 10</td>
</tr>
<tr>
<td><strong>Angle at peak FR (°)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee</td>
<td>155 ± 7</td>
<td>160 ± 5</td>
<td>155 ± 7</td>
<td>160 ± 5</td>
</tr>
<tr>
<td>Tibial</td>
<td>76 ± 5</td>
<td>84 ± 2</td>
<td>78 ± 5</td>
<td>84 ± 3</td>
</tr>
<tr>
<td>Ankle</td>
<td>90 ± 8</td>
<td>102 ± 10</td>
<td>96 ± 11</td>
<td>102 ± 5</td>
</tr>
<tr>
<td>Foot</td>
<td>162 ± 11</td>
<td>163 ± 7</td>
<td>162 ± 11</td>
<td>162 ± 12</td>
</tr>
<tr>
<td><strong>Angular displacement (°) from IC to peak FR</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee</td>
<td>13 ± 12</td>
<td>13 ± 10</td>
<td>12 ± 4</td>
<td>9 ± 6</td>
</tr>
<tr>
<td>Tibial</td>
<td>9 ± 3</td>
<td>7 ± 2</td>
<td>8 ± 3</td>
<td>5 ± 5</td>
</tr>
<tr>
<td>Ankle</td>
<td>32 ± 12</td>
<td>26 ± 10</td>
<td>33 ± 17</td>
<td>36 ± 18</td>
</tr>
<tr>
<td>Foot</td>
<td>24 ± 9</td>
<td>20 ± 8</td>
<td>26 ± 10</td>
<td>22 ± 15</td>
</tr>
<tr>
<td><strong>Angular velocity at IC (°.s⁻¹)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee</td>
<td>66 ± 65</td>
<td>113 ± 36</td>
<td>48 ± 42</td>
<td>70 ± 44</td>
</tr>
<tr>
<td>Tibial</td>
<td>48 ± 50</td>
<td>71 ± 33</td>
<td>39 ± 39</td>
<td>51 ± 40</td>
</tr>
<tr>
<td>Ankle</td>
<td>319 ± 99</td>
<td>419 ± 148</td>
<td>395 ± 152</td>
<td>425 ± 140</td>
</tr>
<tr>
<td>Foot</td>
<td>270 ± 108</td>
<td>346 ± 130</td>
<td>362 ± 141</td>
<td>375 ± 116</td>
</tr>
<tr>
<td><strong>Angular velocity at peak FR (°.s⁻¹)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee</td>
<td>361 ± 82</td>
<td>474 ± 89</td>
<td>386 ± 51</td>
<td>465 ± 100</td>
</tr>
<tr>
<td>Tibial</td>
<td>209 ± 44</td>
<td>273 ± 49</td>
<td>205 ± 50</td>
<td>271 ± 49</td>
</tr>
<tr>
<td>Ankle</td>
<td>572 ± 154</td>
<td>669 ± 176</td>
<td>598 ± 148</td>
<td>819 ± 189</td>
</tr>
<tr>
<td>Foot</td>
<td>366 ± 142</td>
<td>407 ± 196</td>
<td>373 ± 119</td>
<td>562 ± 181</td>
</tr>
</tbody>
</table>

Considering the lower limb kinematics, the knee joint angles generated by the subjects during both experimental tasks in the present study are similar to values reported in previous landing studies. For example, McNitt-Gray (1991) observed knee joint angles of 156° and 155° in recreational athletes performing drop landings from medium and high bench heights. Similarly, Kovacs et al. (1999) observed knee joint angles at IC of 147° in subjects landing with a heel-toe footfall pattern and 149° when landing with a forefoot landing. Compared to the present results, higher peak knee joint angular
velocity data have been recorded by McNitt-Gray (1991), who reported knee joint angular velocity at IC during drop jump landings from a low, medium and high bench heights in recreational athletes of 525 ± 59°.s\(^{-1}\), 718 ± 78°.s\(^{-1}\) and 912 ± 107°.s\(^{-1}\), respectively.

No previous volleyball studies, which reported tibial segment angles or angular velocities at IC and/or the peak \(F_R\), were located in the literature. However, other landing studies have reported these kinematic variables. For example, mean tibial segment angle values higher than those in the present study have been reported by Steele (1997) who observed angles of 108 ± 10° at IC and 114 ± 5° at the peak \(F_R\) in a single-limb landing task after a leap to catch a ball. Pinniger (1996) reported a mean tibial segment angle of 97 ± 2.5°.s\(^{-1}\) for rugby league players immediately prior to IC in the stride cycle of sprinting. These studies also reported mean tibial segment angular velocities of 164 ± 57°.s\(^{-1}\) at IC and 459 ± 49° at the time of the peak \(F_R\) (Steele, 1997), and 409 ± 85°.s\(^{-1}\) immediately prior to IC of the stride cycle in sprinting (Pinniger, 1996). The smaller tibial segment angles and the lower angular velocities reported in the current study, compared to those reported in these previous studies, are most likely due to differences in the experimental tasks. That is, the subjects studied by Steele (1997) performed a three step approach to leap horizontally forward and land, and the subjects studied by Pinniger (1996) performed sprint running. Both of these tasks have much greater horizontal momentum compared to the more vertical momentum typical of the SJM and DJM performed in the present study. As a result, there was a more vertical alignment of the tibia at IC and less rapid tibial angular motion in the present study compared to the tibial motion displayed by subjects in the studies of Steele (1997) and Pinniger (1996).

Ankle joint angles similar to the present values have also been previously reported. For example, McNitt-Gray (1991) observed ankle joint angles at IC of 130° and 129° in recreational athletes landing from a medium and high bench height, respectively. However, Kovacs et al. (1999) observed that foot placement strategy modified the ankle joint angle at IC with ankle joint angle varying from 60° to 101° in a heel-toe landing and a forefoot landing, respectively. Peak ankle joint angular velocity at IC during drop
jumps from low, medium and high bench heights have been reported for recreational athletes of $959 \pm 167^\circ \cdot s^{-1}$, $1,160 \pm 126^\circ \cdot s^{-1}$ and $1,351 \pm 151^\circ \cdot s^{-1}$, respectively (McNitt-Gray, 1991). These values are substantially higher than those reported in the present study. This between-study difference in peak ankle joint angular velocity may be due to the lower peak vertical jump heights obtained in both experimental tasks in the present study combined with the more compliant sand landing surface. That is, subjects observed by McNitt-Gray (1991) performed drop jumps from a peak vertical height of 0.32 m, 0.72 m and 1.28 m, and that with increasing vertical jump height, the peak ankle joint angular velocities increased. In the present study, the subjects jumped from a lower peak vertical height of 0.41 m in the DJM compared to the two highest take-off bench heights of McNitt-Gray (1991). However, this does not explain the between-study differences for the lowest peak vertical jump height of 0.32 m whereby a higher ankle joint angular velocity was still noted relative to the data recorded in the present study. This may be due to the compliance of the sand surface, as the subjects’ feet often penetrated deep into the sand, and this between-study surface difference may have influenced the ankle joint angular velocity during landing. No data were located in the literature pertaining to foot segment angles or angular velocities at IC and/or peak FR during a comparable landing task, against which the present results could be compared.

4.3.1. Effect of Movement Task on the Kinematics

4.3.1.1. Vertical Jump Height

The F-ratios and p-values for each source of variance for the variables characterising vertical jump height are presented in Table 4-6. There was a significant main effect of movement task on all the variables characterising greater trochanter motion during landing when the data were pooled across the fatigued conditions (see Table 4-6). That is, post hoc analysis indicated that in the DJM, there was a longer time from the peak vertical jump height to IC with the sand ($315 \pm 16$ ms SEM; $q = 4.699; p < 0.008$) compared to during to during a SJM ($Y_{MAX}$ to IC = 239 ± 16 ms). Furthermore, in the DJM, there was a larger vertical displacement of the greater trochanter marker from its peak vertical height to IC ($417 \pm 1.8$ mm SEM; $q = 4.776; p < 0.006$) and a larger relative horizontal marker displacement between these positions ($214 \pm 15$ mm SEM; $q = 10.466; p < 0.001$) compared to a SJM ($Y_{MAX}$ to YIC = 327 ± 20 mm SEM; $X_{MAX}$ to XIC = 51 ±16
Table 4-6: F-ratios and p-values derived for each source of variance for the changes in the position of the greater trochanter marker from the peak vertical displacement (YMAX) and its relative horizontal position (XMAX) to the marker’s vertical position (YIC) and its relative horizontal position (XIC) at initial contact (IC) displayed by the subjects (n = 14).

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Movement</th>
<th>Condition</th>
<th>Movement x Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F(1,6)</td>
<td>p-value</td>
<td>F(1,6)</td>
</tr>
<tr>
<td>Time from YMAX to IC (ms)</td>
<td>11.841</td>
<td>0.005*</td>
<td>1.154</td>
</tr>
<tr>
<td>YMAX to YIC (mm)</td>
<td>13.102</td>
<td>0.003*</td>
<td>2.744</td>
</tr>
<tr>
<td>XMAX to XIC (mm)</td>
<td>55.542</td>
<td>&lt;0.001*</td>
<td>0.861</td>
</tr>
</tbody>
</table>

* indicates a significant main effect or movement x condition interaction.

Although there was a significant main effect of movement task on the time from the peak vertical jump height to IC and the vertical displacement of the greater trochanter marker from its peak vertical height to IC, these effects were dependent upon the fatigue condition as shown by Figure 4-3 and Figure 4-4. That is, although there was a significant difference in the time from the peak vertical jump height to IC between the two movement tasks when fatigued, this difference was not significant when non-fatigued. Furthermore, although there was a significant difference in the vertical displacement of the greater trochanter marker from its peak vertical height to IC between the two movement tasks, this difference was larger in a fatigued compared to a non-fatigued condition. This significant difference in the time from the peak vertical jump height to IC between the two movements tasks when fatigued merely reflects the lower vertical jump height obtained by the subjects in the SJM relative to the set bench height in the DJM. As the subjects became fatigued this differences was more noticeable (see Section 4.3.2.1.). Furthermore, in the SJM, the subjects’ motion was predominately in the vertical plane whereby the subjects demonstrated minimal horizontal motion of their greater trochanter marker from the peak of the jump until IC. In contrast, in a DJM, the subjects landed significantly further forward when stepping off the bench to contact the sand, displaying more horizontal motion relative to a SJM. Therefore, it is clearly evident that the two landing phases of the experimental tasks are
very different with respect to vertical and horizontal displacement of the body, and that these differences are amplified via fatigue.

![Graph showing time from peak vertical jump to IC for non-fatigued and fatigued conditions for DJM and SJM.

Figure 4-3: Significant movement type x fatigue condition interaction for the time from the peak vertical jump to IC.

4.3.1.2. Knee Joint and Tibial Motion

The F-ratios and p-values for each source of variance for the lower limb kinematic variables are presented in Table 4-7. Although there was no significant main effect of movement task on knee joint angular displacement, there was a significant main effect of movement task on the knee joint angle both at IC and at the time of the peak FR when the data were pooled across the fatigue conditions (see Table 4-7). That is, the knee joint angles generated by the subjects during a DJM were larger at IC (171 ± 1° SEM; q = 4.538; p < 0.008) and at the peak FR (160 ± 1° SEM; q = 4.562; p < 0.009) than when performing a SJM (IC = 167 ± 1° SEM; peak FR = 156 ± 1° SEM; see Table 4-7). Therefore, although the subjects displayed the same total range of motion at the knee during both tasks, they performed this motion from a position of greater knee extension during the DJM.
A significant main effect of movement task was also noted on the knee joint angular velocity at both IC and at the time of the peak $F_R$ when the data were pooled across the fatigue conditions (see Table 4-7). Post hoc analysis indicated that the knee joint angular velocity generated by subjects during a DJM were higher at IC ($97 \pm 12^\circ.s^{-1}$ SEM; $q = 3.786; p < 0.023$) and at the time of the peak $F_R$ ($466 \pm 18^\circ.s^{-1}$ SEM; $q = 5.468; p < 0.003$) than when performing a SJM ($IC = 52 \pm 12^\circ.s^{-1}$ SEM; peak $F_R = 364 \pm 20^\circ.s^{-1}$ SEM). Therefore, the more extended knee posture displayed by the subjects during the DJM was accompanied by great angular velocity at the joint during landing.
Table 4-7: F-ratios and p-values derived for each source of variance for the angular displacement and angular velocity at initial contact (IC) and at the time of peak resultant ground reaction force (peak FR) during landing displayed by the subjects (n = 14).

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Movement</th>
<th>Condition</th>
<th>Movement x Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angle at IC (°)</td>
<td>F(1,12)</td>
<td>p-value</td>
<td>F(1,12)</td>
</tr>
<tr>
<td>Knee</td>
<td>10.613</td>
<td>0.006*</td>
<td>1.236</td>
</tr>
<tr>
<td>Tibial</td>
<td>41.649</td>
<td>&lt;0.001*</td>
<td>6.187</td>
</tr>
<tr>
<td>Ankle</td>
<td>0.001</td>
<td>0.971</td>
<td>1.999</td>
</tr>
<tr>
<td>Foot</td>
<td>8.008</td>
<td>0.015*</td>
<td>0.507</td>
</tr>
<tr>
<td>Angle at peak FR (°)</td>
<td>F(1,12)</td>
<td>p-value</td>
<td>F(1,12)</td>
</tr>
<tr>
<td>Knee</td>
<td>10.388</td>
<td>0.007*</td>
<td>0.194</td>
</tr>
<tr>
<td>Tibial</td>
<td>79.430</td>
<td>&lt;0.001*</td>
<td>7.571</td>
</tr>
<tr>
<td>Ankle</td>
<td>7.082</td>
<td>0.027*</td>
<td>0.030</td>
</tr>
<tr>
<td>Foot</td>
<td>0.140</td>
<td>0.716</td>
<td>0.311</td>
</tr>
<tr>
<td>Angular displacement (°) from IC to peak FR</td>
<td>F(1,12)</td>
<td>p-value</td>
<td>F(1,12)</td>
</tr>
<tr>
<td>Knee</td>
<td>0.706</td>
<td>0.419</td>
<td>1.092</td>
</tr>
<tr>
<td>Tibial</td>
<td>6.176</td>
<td>0.027*</td>
<td>1.577</td>
</tr>
<tr>
<td>Ankle</td>
<td>0.021</td>
<td>0.888</td>
<td>0.996</td>
</tr>
<tr>
<td>Foot</td>
<td>1.988</td>
<td>0.179</td>
<td>0.042</td>
</tr>
<tr>
<td>Angular velocity at IC (°.s⁻¹)</td>
<td>F(1,12)</td>
<td>p-value</td>
<td>F(1,12)</td>
</tr>
<tr>
<td>Knee</td>
<td>7.474</td>
<td>0.020*</td>
<td>7.116</td>
</tr>
<tr>
<td>Tibial</td>
<td>1.712</td>
<td>0.215</td>
<td>4.362</td>
</tr>
<tr>
<td>Ankle</td>
<td>2.876</td>
<td>0.112</td>
<td>0.356</td>
</tr>
<tr>
<td>Foot</td>
<td>1.966</td>
<td>0.182</td>
<td>1.670</td>
</tr>
<tr>
<td>Angular velocity at peak FR (°.s⁻¹)</td>
<td>F(1,12)</td>
<td>p-value</td>
<td>F(1,12)</td>
</tr>
<tr>
<td>Knee</td>
<td>16.448</td>
<td>0.002*</td>
<td>0.024</td>
</tr>
<tr>
<td>Tibial</td>
<td>25.999</td>
<td>&lt;0.001*</td>
<td>0.002</td>
</tr>
<tr>
<td>Ankle</td>
<td>9.913</td>
<td>0.012*</td>
<td>3.128</td>
</tr>
<tr>
<td>Foot</td>
<td>5.507</td>
<td>0.042*</td>
<td>3.019</td>
</tr>
</tbody>
</table>

* indicates a significant main effect or movement x condition interaction.

Changes in the knee joint angle during landing are dependent upon the relative position of the tibial segment during landing. It was therefore not surprising to note that, similar to the knee joint results, there was a significant main effect of movement task on the...
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tibial segment angles at IC and at the time of the peak $F_R$ when the data were pooled across the fatigue conditions (see Table 4-7). Post hoc analysis indicated that the tibial segment angles generated by the subjects during a DJM were more vertical both at IC ($91 \pm 1^{\circ} \text{SEM}; q = 9.127; p < 0.001$) and at the time of the peak $F_R$ ($84 \pm 1^{\circ} \text{SEM}; q = 12.539; p < 0.001$) than when performing a SJM ($IC = 85 \pm 1^{\circ} \text{SEM}; peak F_R = 76 \pm 1^{\circ} \text{SEM}$). This more vertical alignment of the tibial reflects the more extended knee posture displayed by the subjects during the DJM relative to the SJM. Interestingly, unlike the knee motion data, the main effect of movement task on the tibial segment angle at both IC, was dependent upon the fatigue condition (see Figure 4-5). That is, although there was a significant difference in the tibial segment angle at IC between the two movement tasks, this significant difference was greater in the non-fatigued condition (SJM NF = $83 \pm 1^{\circ} \text{SEM}; DJM NF = 91 \pm 1^{\circ} \text{SEM}; q = 9.510; p < 0.001$) than in the fatigued condition (SJM = $86 \pm 1^{\circ} \text{SEM}; DJM = 91 \pm 1^{\circ} \text{SEM}; q = 5.764; p < 0.001$). Irrespective of the fatigue effects on tibial alignment, subjects landed with a more vertical tibial segment during the DJM compared to the SJM.

![Figure 4-5](image.png)

**Figure 4-5:** Significant movement type x fatigue condition interaction for the tibial segment angle at IC.
A significant main effect of movement task was also noted for the tibial segment angular displacement from IC to the time of the peak FR when the data were pooled across the fatigue conditions (see Table 4-7). That is, the mean tibial segment angular displacement displayed by the subjects during a DJM from IC to the time of the peak FR was less ($6 \pm 1^\circ$ SEM; $q = 3.513; p < 0.029$) than when performing a SJM ($8 \pm 1^\circ$ SEM). Although statistically significant, the difference in these means was only $2^\circ$. Considering the errors inherent in determining segmental alignment via data provided using external skin mounted markers, this difference may not be considered clinically relevant and warrants further investigation.

No significant main effect of movement task was noted on the tibial angular velocity at peak IC when the data were pooled across the fatigue conditions (see Table 4-7). However, a significant main effect of movement task was noted on the tibial angular velocity at the time of the peak FR (see Table 4-7). That is, post hoc analysis indicated that the mean tibial angular velocity generated by the subjects during a DJM were greater at the time of the peak FR ($275 \pm 9^\circ.s^{-1}$ SEM; $q = 7.211; p < 0.001$) than when performing a SJM ($207 \pm 9^\circ.s^{-1}$ SEM). These results again reflect the more rapid motion of the tibial segment about the knee during the DJM relative to the SJM.

Landing technique can influence the impact loads generated at foot-ground contact and the tension on the muscles and tendons (Stacoff et al., 1988). During the DJM, subjects landed with a more vertical tibia and less knee joint flexion, combined with a higher angular velocity of the joint, a higher rate of loading of the ground reaction forces and a higher peak FR compared to SJM. Previous research has shown that peak FR values are influenced by knee joint angle during landing (Dufek & Bates, 1990; Grant et al., 1996; Hewett et al., 1996; Lafortune, 1985; Stacoff et al., 1988). The findings of the present study are in agreement with Stacoff et al. (1988) who found that the less a subject flexed their knees at landing the greater the initial impact force. The authors also observed that, when the knee was less flexed at landing, the knee joint was forced into greater flexion. However, this notion was not observed in the present study, as there was no significant difference between the total knee joint angular displacement between the two
experimental tasks, despite differences in knee joint angle at both IC and at the time of the peak $F_R$.

Knee joint angle during landing has been associated with patellar tendinosis (Lafortune, 1985). During knee flexion, the patellofemoral joint reaction force increases (McGinty et al., 2000) and, in turn, the patella tendon must withstand higher tension (Lafortune, 1985). Lafortune (1985) noted that basketball players with patellar tendinosis landed with less knee joint flexion and with a straighter leg compared to healthy athletes who had more hip and knee joint excursion during landing. However, it is possible that these athletes with patellar tendinosis were displaying a compensatory adaptation to their injury rather than a factor that may have predisposed them to the injury initially. That is, the athletes may have landed with less knee joint flexion to reduce loading on the patellar tendon. Richards et al. (1996) identified that a significant predictor of patellar tendinosis and an increased risk of a player having patellar tendinosis was associated with a larger knee joint angular displacement. Therefore, a player landing with less knee joint flexion but who is forced into a greater range of knee joint flexion may possibly increase the risk of developing patella tendinosis (Richards et al., 1996; Stacoff et al., 1988), by increasing the patellofemoral joint reaction force, and in turn, increasing the load on the patellar tendon. In the present study, although the subjects landed with less knee joint flexion during the DJM, they did not display a greater angular displacement at the knee joint from IC to the time of the peak $F_R$ compared to the SJM. Therefore, it is difficult to speculate if the subjects may be predisposed to greater patellofemoral joint reaction forces or higher patellar tendon loading during a DJM compared to a SJM. As the patellar tendon sustains higher forces with greater knee joint flexion, a player performing a SJM may be at a greater risk of developing patellar tendinosis due to more flexed knee joint posture sustained during landing. However, in the present study, knee joint flexion angles were only analysed between IC and the time of the peak $F_R$, and not throughout the entire landing action. These data may therefore not be a true reflection of the maximum knee joint flexion angle obtained during landing and, as such, should be interpreted with caution. Further research is therefore warranted, to predict the forces that the patellar tendon withstands during the landing phase of both a SJM and a DJM to better understand the relationship between knee joint flexion and patellar tendon loading during landing in these volleyball skills.
There was no significant main effect of movement task on the ankle joint angle at IC or the ankle joint angular displacement from IC to the time of the peak FR (see Table 4-7). Therefore, despite sustaining a higher peak FR in the DJM, subjects displayed similar ankle joint angular displacement in both experimental tasks. This result is in contrast to the finding reported by Grant et al. (1996), who observed that the peak Fv generated at landing was influenced by the ankle joint angular position. However, due to small number of subjects (n = 7), the results of Grant et al. (1996) need to be interpreted carefully.

A significant main effect of movement task on the ankle joint angle at the time of the peak FR was noted when the data were pooled across the fatigue conditions (see Table 4-7). Post hoc analysis indicated that the ankle joint angles displayed by the subjects during a DJM were larger at the time of the peak FR (101 ± 2° SEM; q = 3.628; p < 0.034) than when performing a SJM (91 ± 2° SEM). That is, the subjects displayed a more plantar flexed position at the ankle during the DJM compared to the SJM. Although there was a main effect of movement task on the ankle joint angle at the time of the peak FR, this effect was dependent upon the fatigue condition (see Figure 4-6). That is, although there was a significant difference in the ankle joint angles at the time of the peak FR between the two movement tasks when non-fatigued, this difference was not significant after being fatigued.

Although there was no significant main effect of movement task on the ankle joint angular velocity at IC when the data were pooled across the fatigue conditions (see Table 4-7), a significant main effect of movement task on the ankle joint angular velocity at the time of the peak FR was evident (see Table 4-7). Post hoc analysis indicated that the ankle joint angular velocity at peak FR displayed by the subjects during the DJM were higher (727 ± 30°.s⁻¹ SEM; q = 4.473; p < 0.014) than when performing the SJM (585 ± 33°.s⁻¹ SEM).
Kovacs et al. (1999) stated that decreased ankle joint angular displacement limited the range of motion over which the ankle joint dorsiflexor muscles could act to dissipate the forces during impact, causing the knee and hip joints to have to contribute more to this force dissipation. However, in the present study, no significant main effect of movement task was observed for either the ankle joint or the foot segment angular displacement displayed by the subjects from IC to the time of the peak $F_R$ when the data were pooled across fatigue conditions. It should be noted, however, that there were difficulties quantifying foot and ankle motion in the present study. That is, as the subjects’ feet often penetrated deep into the sand, the marker placed on the 5th metatarsal head was often not in the field of view of the Position Sensor, such that this data could not be recorded. This loss of the 5th metatarsal head marker accounts for missing data and, in turn, the low degrees of freedom reported in Table 4-7. Large standard deviations in the ankle joint and foot segment angles at IC and at the time of the peak $F_R$, are also evident in Table 4-5 indicating that the subjects used a variety of
individual foot and ankle strategies to dissipate the landing forces. Further research is therefore recommended to identify which of these foot and ankle strategies could be used to minimise loading of the patellar tendon during landing and, in turn, reducing the risk of developing patellar tendinosis.

Although no significant main effect of movement task was noted on the foot segment angular displacement, a significant main effect of movement task was noted on the foot segment angle at IC when the data were pooled across the fatigue conditions (see Table 4-7). That is, post hoc analysis indicated the subjects aligned their feet more horizontally relative to the ground at IC during a DJM (142 ± 2° SEM; q = 3.979; p < 0.016) than when performing a SJM (136 ± 2° SEM). The greater ankle joint angle noted during the DJM was therefore caused by the more vertical tibial alignment rather than a more plantar flexed foot at IC. However, there was no significant main effect of movement task on the foot segment angle at the time of the peak FR when the data were pooled across the fatigue conditions (see Table 4-7). This lack of a main effect of movement task on the foot segment angle at the time of the peak FR reflects the fact that the foot was in full contact with the sand surface in both experimental tasks by the time the peak FR were generated.

There was no significant main effect of movement task on the foot segment angular velocity at IC when the data were pooled across the fatigue conditions (see Table 4-7). However, there was a significant main effect of movement task noted on the foot segment angular velocity at the time of the peak FR (see Table 4-7). That is, post hoc analysis indicated that the mean foot segment angular velocity at the time of the peak FR displayed by the subjects during a DJM was higher (463 ± 23°.s⁻¹ SEM; q = 3.385; p < 0.044) than when performing a SJM (381 ± 26°.s⁻¹ SEM). Although there was a significant main effect of movement task on the foot segment angular velocity at the time of the peak FR, this effect was dependent upon the fatigue condition (see Figure 4-7). That is, although there was a significant difference in the foot segment angular velocity at the time of the peak FR between the two movement tasks when fatigued (DJM F = 562 ± 43°.s⁻¹; SJM F = 357 ± 53°.s⁻¹), this difference was not significant prior to the fatigue protocol (DJM NF = 364 ± 43°.s⁻¹; SJM NF = 405 ± 43°.s⁻¹).
In summary, the data presented in this section indicates that the subjects displayed, in general, different segmental motion and alignment during landing between the two movement conditions. That is, during the DJM the subjects displayed significantly less knee flexion, a higher knee joint angular velocity, and a more vertical tibial alignment all at IC and at the time of the peak $F_R$ relative to the SJM. They also displayed a smaller tibial segment angular displacement, less dorsiflexion at the ankle joint at the time of the peak $F_R$, a higher ankle joint angular velocity, a more vertically aligned the foot segment at IC and a higher foot segment angular velocity at the time of the peak $F_R$ relative to the SJM. These significant differences clearly indicate that the landing phase of the two experimental tasks involve different segmental kinematics. Therefore, using a DJM to replicate the landing phase of a SJM is not valid. Furthermore, there were large between-subject variations in the strategies adopted at landing, reflected by the large standard deviations recorded in the segmental motion and alignment between the two experimental task variables. These findings are in agreement with McNitt-Gray et
al. (1993) who noted that, when landing from different heights, female gymnasts self-selected their landing strategy by altering the multijoint coordination plan they adopted via neuromuscular control. Which particular landing strategy would minimise the risk of developing injuries, such as patellar tendinosis, warrants further investigation.

4.3.2. Effect of Fatigue on the Kinematics

4.3.2.1. Vertical Jump Height

Although there was no significant main effect of movement task on any of the greater trochanter displacement variables, there was a significant movement x condition interaction for the time from Y_{MAX} to IC (see Figure 4-3) and the change in the position of the marker from Y_{MAX} to Y_{IC} (see Figure 4-4). However, post hoc analysis did not support a significant effect of fatigue on the time from Y_{MAX} to IC. The power of this statistical test was very low (6.2% at \( p = 0.05 \)) and therefore these data need to be interpreted cautiously. In contrast, post hoc analysis indicated that in a SJM subjects displayed a lower vertical peak height of the greater trochanter marker relative to IC when fatigued (30.5 ± 14 cm SEM; \( q = 4.924; p < 0.004 \)) than when non-fatigued (Y_{MAX} to Y_{IC} = 35.0 ± 1.0 cm SEM). That is, when performing a SJM, the subjects were unable to jump as vertically high when fatigued than when non-fatigued. This finding of the present study is in agreement with MacLaren (1982) who found that, after male volleyball players were fatigued from a series of consecutive vertical jumps, the players attained a lower jump height compared to when non-fatigued (see Section 2.5.4.5.). In the DJM, the effect of fatigue on the greater trochanter marker displacement was not evident, as the subjects performed the movement from a standard height bench.

4.3.2.2. Knee Joint and Tibial Motion

There was no significant main effect of fatigue on the knee joint angles at either IC or at the time of the peak \( F_R \) or the knee joint or tibial segment angular displacement when the data were pooled across movement tasks (see Table 4-7). That is, fatigue did not significantly alter the amount of knee joint flexion in either experimental movement. The lack of a significant main effect of fatigue needs to be interpreted somewhat cautiously, due to the low statistical power. Acknowledging this caution, the results of the present study do not support the results of Grant et al. (1996), who observed that the increase in peak \( F_R \) during a basketball landing in a fatigued condition, caused the
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subjects to increase their knee joint angle at IC and their maximum knee joint angular displacement. However, as a result of fatigue, none of the knee joint alignment variables or the peak $F_R$ during landing were altered in either movement task in the present study.

In contrast, a significant main effect of fatigue was noted on the knee joint angular velocity at IC (see Table 4-7). That is, post hoc analysis indicated the mean knee joint angular velocity displayed by subjects when fatigued was lower ($60 \pm 8^\circ.s^{-1} \text{SEM}; q = 3.757; p < 0.024$) than when performing the movements non-fatigued ($90 \pm 7^\circ.s^{-1} \text{SEM}$). This finding of a reduced knee joint angular velocity was inconsistent with the results of Nicholas et al. (1984), who postulated that the decrease in force after impact in landing in a fatigued state was related to the faster and longer flexion moment. However, in the present study, there was no significant main effect of fatigue condition on the knee joint angular velocity at the time of the peak $F_R$. Therefore, although subjects flexed their knee joint at a slower velocity at the time of IC when fatigued, this velocity difference between the two conditions was not evident by the time the peak $F_R$ forces were generated. It remains unclear how this change on the knee joint angular velocity at IC as a result of fatigue would impact on patellar tendon loading, particularly considering the lack of difference at the time of the peak $F_R$. Further research is therefore recommended to predict the forces that the patellar tendon sustains during landing to more clearly determine how the kinematic variables influence these patellar tendon forces, in both a non-fatigued and fatigued state.

Although fatigue did not significantly influence knee joint alignment, a significant main effect of fatigue was noted on the tibial segment angles at IC and at the time of the peak $F_R$ when the data were pooled across movement conditions (see Table 4-7). That is, post hoc analysis indicated the subjects displayed a significantly more vertical tibial alignment when fatigued at IC ($88 \pm 0.4^\circ \text{SEM}; q = 3.518; p < 0.029$) and at the time of the peak $F_R$ ($F = 81 \pm 0.4^\circ \text{SEM} \; q = 3.917; \; p < 0.0179$) than when performing the movements in a non-fatigued condition ($IC = 87 \pm 0.4^\circ \text{SEM}; \; at \; the \; time \; of \; peak \; F_R = 79 \pm 0.4^\circ \text{SEM}$). However, although there was a significant main effect of fatigue on the tibial segment angles at IC, this effect was dependent upon the movement condition.
shown in Figure 4-5. That is, although there was a significant difference in the tibial segment angle at IC between the two fatigue conditions in a SJM (NF = 83 ± 1°; F = 86 ± 1°), this difference was not statistically significant in a DJM (NF = 91 ± 1°; F = 91 ± 1°). In the DJM, the lack of change in the tibial segment alignment displayed by the subjects between fatigue conditions may be attributed to the subjects stepping off the standard height bench onto the sand surface, thereby permitting a more constrained tibial segment. Furthermore, during a DJM, there was more time from the peak vertical jump height to IC with the sand compared to a SJM (see Section 4.3.1.1.). Therefore, in the DJM, the longer and consistent time interval may have allowed the subjects to better prepare themselves for landing relative to the SJM, enabling the subjects to achieve a constant tibial segment alignment at IC in a fatigued condition and in a non-fatigued condition.

4.3.2.3. Ankle Joint and Foot Motion

There was no significant main effect of fatigue on any of the ankle joint variables at either IC or at the time of the peak FR when the data were pooled across movement conditions (see Table 4-7). However, despite a lack of any significant main effect of fatigue, a significant movement x condition interaction was noted on the ankle joint angle at the time of the peak FR (see Table 4-7). Post hoc analysis revealed a significant difference in the ankle joint angle at the time of the peak FR in a SJM, whereby the subjects displayed less ankle joint dorsiflexion in a fatigued condition (96 ± 2° SEM; q = 8.711; p < 0.036) compared to a non-fatigued condition (87 ± 3° SEM). The ankle joint plantar flexor muscles, such as the MG, act eccentrically to control ankle joint dorsiflexion during landing and to absorb the energy associated with impact attenuation (Kovacs et al., 1999; see Section 2.5.3.1.). Less ankle joint dorsiflexion during landing in a fatigued condition compared to a non-fatigued condition indicates that the ankle joint plantar flexor muscles may have a reduced ability to act to dissipate the impact force of landing and, in turn, these forces may be dissipated proximal to the ankle joint. How this change to the ankle joint angle at IC as a result of fatigue would impact on the patellar tendon loading remains unclear and warrants further investigation.
Despite a lack of a significant main effect fatigue on any of the foot segment variables at either IC or at the time of the peak $F_R$ (see Table 4-7), there was a significant movement $x$ condition interaction on the foot angular velocity at the time of the peak $F_R$ (see Table 4-7). That is, the subjects displayed a significantly greater mean foot segment angular velocity at the time of the peak $F_R$ in a fatigued DJM (-562 ± 43°.s$^{-1}$ SEM; $q = 4.730; p < 0.005$) than when performing a non-fatigued DJM (-365 ± 43°.s$^{-1}$ SEM). This indicates that when the subjects were fatigued in a DJM, they were less able to control the rate of ankle joint dorsiflexion during landing. This provides further indication that the ankle joint plantar flexor muscles were less able to act to dissipate the impact of landing compared to when non-fatigued.

Overall, fatigue had only minor effects on the segmental motion and alignment during landing in both experimental tasks. That is, fatigue lead to a lower knee joint angular velocity at IC in both experiment tasks, a more perpendicular tibial segment angle at IC and at the time of the peak $F_R$ in the SJM, a larger ankle joint angle at the time of the peak $F_R$ in the SJM and a faster angular velocity at peak $F_R$ in the DJM compared to a non-fatigued condition. Furthermore, despite fatigue not significantly altering any of the ground reaction force variables, the lower limb motion and alignment displayed by the subjects during landing in both experimental tasks were significantly altered. This suggests that when fatigued, the subjects were less efficient in dissipating the similar ground reaction forces generated at landing by subjects in a non-fatigued condition. How fatigue caused these minor lower limb motion and alignment changes during landing, may be explained by how fatigue influenced the muscle activation patterns displayed by subjects during landing.

### 4.4. MUSCLE ACTIVATION DATA

The means and standard deviations for the muscle activation variables generated during landing for the two experimental movements are presented in Table 4-8, Table 4-9 and Table 4-10. Similar muscle burst duration data have previously been reported in the literature. For example, Steele (1997) reported muscle burst durations ranging from to 262 ± 73 ms for gastrocnemius to 375 ± 95 ms for VL for subjects at landing after performing a deceleration task whereby the subjects leapt forward to catch a ball and land abruptly. In a similar single-limb deceleration landing task involving catching,
Cowling (1998) reported muscle burst durations ranging from $301 \pm 129$ ms for SM, to $328 \pm 125$ ms for gastrocnemius, to $375 \pm 102$ ms for VL. Therefore, irrespective of whether a landing task involves abrupt deceleration following a horizontal leap to catch a pass or a vertical jump to spike a ball, the duration of muscle activity in a rapid landing appears relatively constant.

Table 4-8: Means and standard deviations for the muscle burst duration and onset/offset times variables displayed by the subjects ($n = 14$) during landing in a spike jump movement (SJM) and a drop jump movement (DJM) when non-fatigued (NF) and fatigued (F).

<table>
<thead>
<tr>
<th>Muscle</th>
<th>SJM NF</th>
<th>DJM NF</th>
<th>SJM F</th>
<th>DJM F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle burst duration (ms)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VL</td>
<td>$310 \pm 71$</td>
<td>$326 \pm 91$</td>
<td>$304 \pm 61$</td>
<td>$361 \pm 95$</td>
</tr>
<tr>
<td>RF</td>
<td>$288 \pm 68$</td>
<td>$341 \pm 82$</td>
<td>$267 \pm 58$</td>
<td>$334 \pm 97$</td>
</tr>
<tr>
<td>VM</td>
<td>$298 \pm 41$</td>
<td>$377 \pm 100$</td>
<td>$295 \pm 73$</td>
<td>$389 \pm 97$</td>
</tr>
<tr>
<td>MG</td>
<td>$223 \pm 75$</td>
<td>$316 \pm 124$</td>
<td>$236 \pm 45$</td>
<td>$273 \pm 60$</td>
</tr>
<tr>
<td>BF</td>
<td>$289 \pm 47$</td>
<td>$350 \pm 95$</td>
<td>$329 \pm 95$</td>
<td>$376 \pm 186$</td>
</tr>
<tr>
<td>ST</td>
<td>$324 \pm 78$</td>
<td>$303 \pm 92$</td>
<td>$320 \pm 62$</td>
<td>$313 \pm 118$</td>
</tr>
<tr>
<td>Muscle burst onset time to IC (ms)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VL</td>
<td>$-73 \pm 34$</td>
<td>$-107 \pm 81$</td>
<td>$-74 \pm 29$</td>
<td>$-83 \pm 36$</td>
</tr>
<tr>
<td>RF</td>
<td>$-75 \pm 51$</td>
<td>$-104 \pm 69$</td>
<td>$-63 \pm 43$</td>
<td>$-82 \pm 54$</td>
</tr>
<tr>
<td>VM</td>
<td>$-77 \pm 23$</td>
<td>$-109 \pm 56$</td>
<td>$-71 \pm 25$</td>
<td>$-89 \pm 36$</td>
</tr>
<tr>
<td>MG</td>
<td>$-115 \pm 40$</td>
<td>$-170 \pm 72$</td>
<td>$-111 \pm 23$</td>
<td>$-144 \pm 59$</td>
</tr>
<tr>
<td>BF</td>
<td>$-68 \pm 57$</td>
<td>$-98 \pm 59$</td>
<td>$-67 \pm 35$</td>
<td>$-92 \pm 52$</td>
</tr>
<tr>
<td>ST</td>
<td>$-87 \pm 29$</td>
<td>$-92 \pm 38$</td>
<td>$-84 \pm 27$</td>
<td>$-89 \pm 33$</td>
</tr>
<tr>
<td>Muscle burst offset time to IC (ms)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VL</td>
<td>$233 \pm 57$</td>
<td>$225 \pm 43$</td>
<td>$230 \pm 54$</td>
<td>$230 \pm 171$</td>
</tr>
<tr>
<td>RF</td>
<td>$216 \pm 54$</td>
<td>$235 \pm 46$</td>
<td>$210 \pm 54$</td>
<td>$255 \pm 80$</td>
</tr>
<tr>
<td>VM</td>
<td>$224 \pm 37$</td>
<td>$269 \pm 76$</td>
<td>$226 \pm 74$</td>
<td>$280 \pm 95$</td>
</tr>
<tr>
<td>MG</td>
<td>$112 \pm 44$</td>
<td>$144 \pm 83$</td>
<td>$127 \pm 39$</td>
<td>$121 \pm 45$</td>
</tr>
<tr>
<td>BF</td>
<td>$236 \pm 100$</td>
<td>$258 \pm 61$</td>
<td>$266 \pm 89$</td>
<td>$292 \pm 179$</td>
</tr>
<tr>
<td>ST</td>
<td>$236 \pm 84$</td>
<td>$210 \pm 98$</td>
<td>$213 \pm 80$</td>
<td>$247 \pm 142$</td>
</tr>
</tbody>
</table>

* a negative value indicates that the muscle burst variable occurred before IC.

** a negative value indicates that the muscle burst variable occurred before the time of the peak FR.

The negative values noted in Table 4-8 for the muscle burst onset times indicated that the subjects activated their lower limb muscles before IC. That is, irrespective of the movement task or fatigue condition, the subjects used an anticipatory muscle activation strategy to stabilise their lower limbs at landing in anticipation of the loads to be generated. This preprogrammed strategy is consistent with previous landing studies in...
which subjects have activated their lower limb muscles prior to IC with the landing surface during drop jump movements (Edwards et al., 2001; Horita et al., 1996, 1999), after performing a leap to catch a ball (Cowling, 1998; Steele, 1997), and in a run and rapid stop movement (Nyland et al., 1994). Although each muscle group was activated before landing, the bursts were maintained until approximately 100 to 300 ms after IC, a pattern that again is consistent with these studies cited above.

Table 4-9: Means and standard deviations for the muscle burst peak activity displayed at initial contact (IC) and at the time of peak resultant ground reaction force (peak FR) by the subjects (n = 14) during landing in a spike jump movement (SJM) and a drop jump movement (DJM) when non-fatigued (NF) and fatigued (F).

<table>
<thead>
<tr>
<th>Muscle</th>
<th>SJM NF</th>
<th>DJM NF</th>
<th>SJM F</th>
<th>DJM F</th>
</tr>
</thead>
<tbody>
<tr>
<td>VL</td>
<td>74 ± 14</td>
<td>65 ± 30</td>
<td>94 ± 14</td>
<td>72 ± 41</td>
</tr>
<tr>
<td>RF</td>
<td>75 ± 25</td>
<td>70 ± 33</td>
<td>82 ± 22</td>
<td>92 ± 26</td>
</tr>
<tr>
<td>VM</td>
<td>74 ± 25</td>
<td>87 ± 32</td>
<td>80 ± 25</td>
<td>93 ± 49</td>
</tr>
<tr>
<td>MG</td>
<td>-3 ± 28</td>
<td>-5 ± 43</td>
<td>-13 ± 22</td>
<td>-18 ± 28</td>
</tr>
<tr>
<td>BF</td>
<td>73 ± 101</td>
<td>49 ± 64</td>
<td>70 ± 62</td>
<td>74 ± 72</td>
</tr>
<tr>
<td>ST</td>
<td>41 ± 88</td>
<td>39 ± 59</td>
<td>40 ± 62</td>
<td>44 ± 95</td>
</tr>
</tbody>
</table>

Muscle burst peak activity time to IC (ms)

Muscle burst peak activity time to peak FR (ms)

Muscle burst onset times relative to IC have previously been reported in the literature. For example, Edwards et al. (2001) reported that highly skilled male Beach Volleyball players displayed a MG muscle burst onset time relative to IC of -112 ± 60 ms during a DJM onto a sand surface. In the present study, the subjects recruited their MG earlier than the subjects noted by Edwards et al. (2001), even though the subjects in the present study and the subjects studied by Edwards et al. (2001), performed an identical DJM from the same drop jump bench height onto a similar compacted sand surface. The
between-study differences in muscle recruitment probably reflect the different subjects used in these studies. That is, in the present study, experienced male Beach and/or Indoor Volleyball players of a variety of different competition levels were recruited, whereas, Edwards et al. (2001) recruited only highly skilled Beach Volleyball players competing in the top local level.

Table 4-10: Means and standard deviations for the muscle burst intensity (% of SJM NF) displayed by the subjects (n = 14) during landing in a spike jump movement (SJM) and a drop jump movement (DJM) when non-fatigued (NF) and fatigued (F).

<table>
<thead>
<tr>
<th>Muscle</th>
<th>SJM NF</th>
<th>DJM NF</th>
<th>SJM F</th>
<th>DJM F</th>
</tr>
</thead>
<tbody>
<tr>
<td>VL</td>
<td>100 ± 0</td>
<td>106 ± 21</td>
<td>105 ± 32</td>
<td>114 ± 42</td>
</tr>
<tr>
<td>RF</td>
<td>100 ± 0</td>
<td>100 ± 26</td>
<td>109 ± 39</td>
<td>112 ± 47</td>
</tr>
<tr>
<td>VM</td>
<td>100 ± 0</td>
<td>107 ± 24</td>
<td>105 ± 26</td>
<td>116 ± 34</td>
</tr>
<tr>
<td>MG</td>
<td>100 ± 0</td>
<td>103 ± 31</td>
<td>97 ± 23</td>
<td>94 ± 26</td>
</tr>
<tr>
<td>BF</td>
<td>100 ± 0</td>
<td>100 ± 24</td>
<td>108 ± 48</td>
<td>106 ± 46</td>
</tr>
<tr>
<td>ST</td>
<td>100 ± 0</td>
<td>97 ± 34</td>
<td>99 ± 19</td>
<td>109 ± 54</td>
</tr>
</tbody>
</table>

Although the muscle onset patterns were similar to previous landing studies, the muscle recruitment order differed between both the movements in the present study and compared to the recruitment order reported for previous landing studies. That is, in a non-fatigued DJM, the MG was recruited first, followed by the quadriceps muscles (VM, VL and RF) and then the hamstring muscles (BF and ST). However, in a non-fatigued SJM, the MG was recruited first, followed by a medial hamstring muscle (ST), then the quadriceps muscles (VM, RF and VL) and finally the lateral hamstring muscle (BF). In a deceleration task involving a single-limb landing to catch a ball, Steele (1997) and Cowling (1998) both reported muscle recruitment patterns whereby the hamstrings muscles (BF and SM) were recruited first, then gastrocnemius and then lastly the quadriceps muscles (VM, VL, RF). The difference between the muscle recruitment patterns observed in the present study and those previously reported in the literature most probably reflect the different experimental task requirements between the studies. That is, the experimental task performed by subjects in the studies of both Steele (1997) and Cowling (1998) involved higher shear forces during landing, necessitating earlier activation of the hamstring muscles to stabilise the tibia against the
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anterior tibial drawer typically generated via quadriceps contractions at landing. In both the DJM and the SJM in the present study, the movement predominately occurred in the vertical plane, as confirmed by the negligible contribution of the $F_{AP}$ and $F_{ML}$ to the $F_R$ (see Section 3.4.2.). This more vertical landing motion required less need for the hamstring muscles to stabilise the knee joint against anterior translation during the landing, and therefore resulted in a later recruitment of both the hamstring muscles relative to the other muscle groups.

Similar data for the timing of peak muscle burst activity have previously been reported in the literature. For example, Edwards et al. (2001) reported that male Beach Volleyball players displayed peak RF activity time relative to IC of $82 \pm 21$ ms and at the time of the peak $F_R$ of $17 \pm 29$ ms during a DJM onto a sand surface. No literature examining the timing of peak muscle activity during a SJM was located. However, Steele (1997) reported that the time of peak muscle activity relative to IC occurred in a leap to catch a ball between $-49 \pm 32$ ms for BF to $10 \pm 40$ ms for gastrocnemius and $81 \pm 35$ ms for RF. In a similar single-limb deceleration task involving catching, Cowling (1998) reported that the time of the peak muscle burst activity relative to IC occurred between $-41 \pm 39$ ms for SM and $70 \pm 25$ ms for RF. These previously published quadriceps muscle and MG muscle burst peak activity times relative to IC are similar to those reported in the present study. However, the hamstring muscle burst peak activity time relative to IC was earlier in these leaping landings than during the more vertical DJM and SJM which were examined in this study. This between-study difference is again most likely due to the differences in the respective experimental tasks discussed in the previous paragraph.

No normative data for lower limb muscle burst intensity displayed during a vertical landing task was located in the literature against which the present data can be compared. That is, in the present study muscle burst intensity was calculated by integrating $50$ ms either side of the peak muscle activity time and then normalising these raw values relative to the non-fatigued SJM value. A similar method of determining muscle burst intensity was used by Kovacs et al. (1999) who expressed the values in terms of the average levels attained during the eccentric phase of a heel-toe drop jump.
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4.4.1. Effect of Movement Task on the Muscle Activation Patterns

4.4.1.1. Muscle Burst Duration and Onset/Offset Times

The F-ratios and p-values derived for each source of variance for the muscle burst duration and onset/offset times are presented in Table 4-11. A significant main effect of movement task was noted on the muscle burst duration data for all the lower limb muscles, except VL and ST, when the data were pooled across the fatigue conditions (see Figure 4-8). That is, *post hoc* analysis indicated that the muscle burst durations in the DJM were longer for RF (338 ± 16 ms SEM; \( q = 3.774; p < 0.019 \)), VM (377 ± 18 ms SEM; \( q = 4.611; p < 0.006 \)) and MG (295 ± 14 ms SEM; \( q = 4.901; p < 0.004 \)) compared to a SJM (RF = 278 ± 16 ms SEM; VM = 295 ± 18 ms SEM; MG 224 ± 15 ms SEM; see Figure 4-8). Although a significant main effect of movement task was noted for the BF muscle burst duration, *post hoc* analysis revealed this difference was not significant between the movement tasks (\( q = 3.053; p < 0.052 \)). However, the subjects in general, displayed longer muscle burst durations during the landing phase of a DJM than when performing a SJM. The longer muscle burst durations during a DJM in the present study, may be a result of the longer time from the peak vertical jump height to IC with the sand (see Section 4.3.1.1.), allowing the subjects longer to recruit and activate their lower limb muscles in preparation to land compared to a SJM. Furthermore, longer muscle burst durations may provide greater stabilisation of the lower limb joints during landing. That is, in the DJM, the longer muscle burst durations displayed by the subjects may indicate that they required greater stabilisation of the lower limb joints during landing to withstand the higher impact loads compared to the SJM (see Section 4.2.1.). However, a potential negative effect of having longer muscle burst durations during landing, is that it may potentially cause the subjects to fatigue earlier (see Section 4.4.2.1.). The cause of the increased muscle burst duration becomes evident when observing the muscle onset and offset data results.
Table 4-11: F-ratios and p-values derived for each source of variance for the muscle burst durations and on/off times displayed by the subjects (n = 14) during landing.

<table>
<thead>
<tr>
<th>Muscle Movement</th>
<th>Condition</th>
<th>Movement ± Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>VL</td>
<td>3.094</td>
<td>0.099</td>
</tr>
<tr>
<td>RF</td>
<td>7.122</td>
<td>0.019*</td>
</tr>
<tr>
<td>VM</td>
<td>11.037</td>
<td>0.005*</td>
</tr>
<tr>
<td>MG</td>
<td>12.311</td>
<td>0.004*</td>
</tr>
<tr>
<td>BF</td>
<td>4.931</td>
<td>0.045*</td>
</tr>
<tr>
<td>ST</td>
<td>0.707</td>
<td>0.419</td>
</tr>
</tbody>
</table>

Muscle burst duration (ms)

- RF: df = 1,13; MG: df = 1,12; ST: df = 1,9.

Muscle burst onset time to IC (ms)

- RF: df = 1,13; ST: df = 1,10.

Muscle burst offset time to IC (ms)

- RF: df = 1,13; ST: df = 1,9.

Analysis of the muscle burst onset time to IC data also revealed a significant main effect of movement task for RF, VM and MG muscles when the data were pooled across the fatigue conditions. Post hoc analysis indicated that the subjects displayed an earlier muscle burst onset time relative to IC during the DJM for RF (-93 ± 8 ms SEM; q = 3.160; p < 0.044), VM (-100 ± 8 ms SEM; q = 3.438; p < 0.030), and MG (-159 ± 9 ms SEM; q = 4.872; p < 0.005) compared to during the SJM (RF = -69 ± 8 ms SEM; VM = -74 ± 8 ms; MG = -113 ± 9 ms SEM; see Table 4-11). Although not statistically significant, a trend for earlier ST, BF and VL muscle burst onset times were also evident during the DJM relative to the SJM. Therefore, it was evident that in general,
the subjects activated their lower limb muscles earlier relative to IC, particularly for the quadriceps muscles and the ankle joint plantar flexor muscles, during a DJM compared to a SJM. This earlier recruitment of these muscles partially accounts for the longer muscle burst durations displayed during the DJM which were discussed on the page 111.

Figure 4-8: Muscle burst duration (mean ± S.D.) during landing in a drop jump movement (DJM) and a spike jump movement (SJM) when non-fatigued (NF) and a fatigued (F; * indicates a significant difference between SJM and DJM).
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A primary function of the lower limb muscle pre-activation is to provide sufficient muscle tension at landing to assist in absorbing the high initial peak impact force and to trigger a segmental reflex activity to adjust muscle stiffness (Nicol et al., 1991). The level of force impact has been shown to correlate with the pre-activation of the gastrocnemius muscle (Arampatzis et al., 2001; Komi et al., 1987 cited in Nicol et al., 1991). This notion is consistent with the results of the present study in which an earlier pre-activation of MG was evident in the DJM, the movement task associated with a higher impact load relative to the SJM. The need to stabilise the lower limb to withstand the higher impact loads during the DJM may also explain why there was a significantly earlier muscle burst onset time relative to IC for RF and VL, combined with longer muscle burst durations. Furthermore, the smaller amount of knee flexion displayed by the subjects during the DJM (see Section 4.3.1.2.), may reflect the increased stiffness provided by the earlier recruitment of the quadriceps muscles.

The muscle burst offset times relative to IC indicated a significant main effect of movement task for RF and VM when the data were pooled across the fatigue conditions. Post hoc analysis indicated that the muscle burst offset times relative to IC occurred later during the DJM for RF (245 ± 9 ms SEM; \( q = 3.466; p < 0.029 \)) and VM (276 ± 15 ms SEM; \( q = 3.483; p < 0.029 \)) compared to during the SJM (RF = 213 ± 9 ms SEM; VM = 223 ± 15 ms SEM; see Table 4-11). The later muscle burst offset evident for all of the quadriceps muscles during the DJM, indicates that subjects activated their quadriceps muscles for longer when performing a DJM to control the deceleration of landing than when performing a SJM. This notion is supported by the longer RF and VM muscle burst durations displayed in the DJM compared to the SJM.

In the DJM the subjects sustained higher impact loads during landing (see Section 4.2.1.), as a consequence of stepping off a platform which was higher than the peak vertical jump height that they were able to achieve during a SJM (see Section 4.3.1.1.). These higher impact loads sustained by the subjects during landing, may have contributed to a greater need for stabilisation of the lower limb joints during landing compared to the SJM. The longer muscle burst durations displayed by the subjects in the DJM, caused by the earlier muscle burst onsets and later muscle burst offsets, reflects this greater stabilisation of the lower limb joints during landing compared to the
SJM. Furthermore, the longer time from the peak vertical jump height to IC with the sand in a DJM, may have also allowed more time for the subjects to recruit and activate their lower limb muscles in preparation to land during the DJM compared to the SJM, thereby contributing to this greater stabilisation.

4.4.1.2. Peak Muscle Activity Time
The F-ratios and p-values derived for each source of variance for the muscle burst peak activity times are presented in Table 4-12. A significant main effect of movement task was only found on the time of the peak VL activity relative to IC data when the data were pooled across the fatigue conditions. That is, post hoc analysis confirmed that there was an earlier peak VL muscle activity relative to IC during the DJM (69 ± 4 ms

**Table 4-12:** F-ratios and p-values derived for the source of variance for the muscle burst peak activity displayed at initial contact (IC) and at the time of peak resultant ground reaction force (peak FR) by the subjects (n = 14) during landing.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Movement</th>
<th>Condition</th>
<th>Movement x Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F(1,11)</td>
<td>p-value</td>
<td>F(1,11)</td>
</tr>
<tr>
<td><strong>Muscle bursts peak activity time to IC (ms)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VL</td>
<td>5.955</td>
<td>0.028*</td>
<td>6.860</td>
</tr>
<tr>
<td>RF</td>
<td>0.224</td>
<td>0.644</td>
<td>5.824</td>
</tr>
<tr>
<td>VM</td>
<td>1.727</td>
<td>0.210</td>
<td>0.619</td>
</tr>
<tr>
<td>MG</td>
<td>0.174</td>
<td>0.683</td>
<td>4.561</td>
</tr>
<tr>
<td>BF</td>
<td>0.003</td>
<td>0.959</td>
<td>1.132</td>
</tr>
<tr>
<td>ST</td>
<td>0.049</td>
<td>0.829</td>
<td>0.157</td>
</tr>
<tr>
<td><strong>Muscle bursts peak activity time to FR (ms)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VL</td>
<td>0.283</td>
<td>0.602</td>
<td>2.656</td>
</tr>
<tr>
<td>RF</td>
<td>11.851</td>
<td>0.004*</td>
<td>2.211</td>
</tr>
<tr>
<td>VM</td>
<td>9.713</td>
<td>0.008*</td>
<td>0.206</td>
</tr>
<tr>
<td>MG</td>
<td>2.497</td>
<td>0.138</td>
<td>7.371</td>
</tr>
<tr>
<td>BF</td>
<td>0.051</td>
<td>0.825</td>
<td>0.130</td>
</tr>
<tr>
<td>ST</td>
<td>0.243</td>
<td>0.631</td>
<td>0.041</td>
</tr>
</tbody>
</table>

* indicates a main effect or movement x condition interaction.

** Muscle burst peak activity time to IC RF df = 1,13; BF df = 1,10; ST df = 1,8.
** Muscle burst peak activity time to peak FR VL df = 1,10; RF df = 1,13; ST df = 1,8.
Results & Discussion

SEM; \( q = 3.463; \ p < 0.029 \) compared to the SJM (83 ± 4 ms SEM). Furthermore, a significant main effect of movement task was found on the time of the peak muscle activity relative to the time of the peak F\(_R\) data for RF and VM. Post hoc analysis indicated that the time of the peak muscle activity relative to the time of the peak F\(_R\) occurred later during the DJM for RF (38 ± 4 ms SEM; \( q = 4.868; \ p < 0.005 \)) and VM (47 ± 7 ms SEM; \( q = 4.344; \ p < 0.009 \)) compared to the SJM (RF = 20 ± 4 ms SEM; VM = 17 ± 7 ms SEM; see Figure 4-9).

![Figure 4-9](image)

**Figure 4-9:** The time of the peak muscle activity relative to the time of the peak resultant ground reaction force (time = 0) during landing (*indicates a significant difference between SJM and DJM; **indicates a significant difference between NF and F).
During knee joint flexion the patella shift laterally (see Section 2.4.1.). As a main
dynamic stabiliser of the knee joint, the VM is recruited to counteract this lateral patella
movement during activities involving dynamic knee joint flexion. Based on the present
findings, it is postulated that the subjects were better able to stabilise their knee joints,
against any lateral patella displacement during the DJM compared to the SJM. This is
evident in the VM having an earlier muscle burst onset time relative to IC, a later peak
muscle burst activity relative to the time of the peak FR, a later muscle burst offset time
relative to IC and a longer muscle burst duration during the DJM compared to during
the SJM. Furthermore, the RF had an earlier muscle burst onset time relative to IC, a
later peak muscle burst activity time relative to the time of the peak FR, and a longer
muscle burst duration in the DJM compared to a SJM. Whether this increased
stabilisation of the knee joint during landing in the DJM was necessitated by the higher
ground reaction forces generated at impact or merely more feasible due to the increased
time from the peak jump height to landing is difficult to ascertain. However, together
with the significantly different ground reaction force and segmental alignment variables,
the significantly different muscle activation patterns clearly supports the notion that the
two experimental movements, the DJM and the SJM, have two distinct landing patterns
and should therefore be considered as different skills.

4.4.1.3. Muscle Burst Intensity

The F-ratios and p-values derived for each source of variance for the muscle burst
intensity are presented in Table 4-13. There was no significant main effect of the
movement task on the muscle burst intensity on any of the muscles when the data were
pooled across fatigue conditions (see Figure 4-8). That is, when the lower limb muscles
were activated during landing, the muscles were recruited at the same intensity in both
experimental tasks. Therefore, although the pattern by which the subjects recruited their
lower limb muscles to stabilise their knee joints were affected by movement task, the
intensity at which the muscles was recruited was not. That is, the two experimental
tasks, the DJM and the SJM, did not significantly differ from each other with respect to
muscle burst intensity displayed during landing.
Table 4-13: F-ratios and p-values for each source of variance for the muscle burst intensity displayed by the subjects (n = 14) during landing in a spike jump movement (SJM) and a drop jump movement (DJM) when non-fatigued (NF) and fatigued (F).

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Movement</th>
<th>Condition</th>
<th>Movement x Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F(1,11)</td>
<td>p-value</td>
<td>F(1,11)</td>
</tr>
<tr>
<td>VL</td>
<td>2.139</td>
<td>0.169</td>
<td>0.311</td>
</tr>
<tr>
<td>RF</td>
<td>0.066</td>
<td>0.802</td>
<td>0.927</td>
</tr>
<tr>
<td>VM</td>
<td>2.539</td>
<td>0.135</td>
<td>0.842</td>
</tr>
<tr>
<td>MG</td>
<td>0.113</td>
<td>0.742</td>
<td>2.353</td>
</tr>
<tr>
<td>BF</td>
<td>0.018</td>
<td>0.895</td>
<td>0.459</td>
</tr>
<tr>
<td>ST</td>
<td>&lt; 0.001</td>
<td>0.991</td>
<td>1.606</td>
</tr>
</tbody>
</table>

* Muscle burst intensity VL df = 1,10; RF df = 1,13; MG df = 12; ST df = 1,8.

4.4.2. Effects of Fatigue on the Muscle Activation Patterns

4.4.2.1. Muscle Burst Duration and Onset/Offset Times

There was no significant main effect of fatigue on the muscle burst duration data for any of the six lower limb muscles when the data were pooled across movements (see Figure 4-8 and Table 4-11). That is, as a result of fatigue, the subjects did not increase or decrease the duration of their muscle bursts for any of the lower limb muscles during landing compared to the non-fatigued condition. This finding is in contrast to previous studies in which fatigue was shown to cause an increase in the muscle burst duration displayed by subjects performing drop jumps (Hortobagyi, 1991) and a decrease in the muscle burst duration for VL and ST during walking (Arendt-Nielsen, 1991). It should be noted, however, that there was relatively high variability in the muscle burst duration data in all conditions in the present study, but particularly for the hamstring muscles during the fatigued DJM. This variability suggests that the subjects adopted a variety of landing strategies in terms of the duration of their muscle burst activations when landing.

There was no significant main effect of fatigue condition on the muscle burst onset time relative to IC or the muscle burst offset times relative to IC when the data were pooled across movement tasks for any of the muscles (see Table 4-11). Therefore, consistent with the muscle burst duration data, fatigue did not cause the subjects to activate their lower limb muscles any earlier or later relative to IC compared to when non-fatigued.
This finding is again in contrast to the previous findings of Edwards et al. (2001) who noted a later MG onset relative to IC during a DJM onto a sand surface. Furthermore, Nyland et al. (1994) found that subjects displayed a significantly later muscle burst onset time relative to IC as a result of fatigue in a run and rapid stop task. Arendt-Nielsen (1991) noted that fatigue caused a delay in the onset of BF and ST in walking. However, Horita et al. (1996) noted an earlier muscle burst onset for VL during a drop jump landing when subjects were fatigued. This inconsistent effect of fatigue on muscle activation times evident in previous studies supports the notion that fatigue effects are specific to the movement task performed. Interestingly, in the present study, there was a trend for a later MG muscle burst onset time relative to IC in a fatigued condition (130 ± 6 ms) compared to a non-fatigued condition (143 ± 5 ms). The trend of a later MG onset when fatigued was supported by Edwards et al. (2001) who noted that, as a result of fatigue, male Beach Volleyball also players exhibited a later MG onset time relative to IC. This result is of particular importance as the MG acts eccentrically during landing to absorb and/or dissipate the impact forces by controlling ankle joint dorsiflexion (Kovacs et al., 1999; see Section 2.4.3.). By delaying MG activation when fatigued, the subjects may be less effective in controlling the rate of dorsiflexion during the impact of landing and, in turn, increasing the force that knee and hip joints must dissipate. This notion is supported by the fact that the subjects also displayed a greater ankle joint angular velocity during the DJM in a fatigued condition compared to a non-fatigued condition (see Section 4.3.). Therefore, in a DJM in a fatigued condition, the knee joint may sustain a higher load, most likely increasing the load sustained by the patellar tendon, and potentially increasing the risk of developing patellar tendinosis.

The muscle recruitment order displayed by subjects in the present study differed between movements (see Section 4.4.), and changed as a result of fatigue. That is, in a DJM in a fatigued condition, although the MG was still recruited first, it was followed by the hamstring muscles (BF and ST), and then the quadriceps muscles (VM, RF and VL) as opposed to the reversed order evident in the non-fatigued condition. In a SJM in a fatigued condition, MG activation was followed by activation of ST, VL, VM and BF followed lastly by RF, an order that differed when non-fatigued (see Section 4.4.). This recruitment order change is not consistent with the findings of Wojtys et al. (1996) who observed that fatigue did not change the order of muscle recruitment, even though they
found a slowing of muscle responses in the plantar flexor muscles, hamstring muscles and quadriceps muscles during maximal knee extension. This between-study difference may again be due to the different movement tasks performed in each study, with the dynamic SJM and DJM landings placing greater stress on the lower limb compared to knee extensions, necessitating the muscle recruitment order change and further supporting the notion that fatigue effects are specific to the movement task.

4.4.2.2. Peak Muscle Activity Time

A significant main effect of fatigue was observed on the muscle burst peak activity time relative to IC for VL and RF when the results were pooled across movement tasks (see Table 4-12). Post hoc analysis indicated that there was a later muscle burst peak activity relative to IC in the fatigued condition for VL (83 ± 4 ms SEM; $q = 3.771; p < 0.020$) and RF (87 ± 4 ms SEM; $q = 3.413; p < 0.031$) compared to the non-fatigued condition (VL = 69 ± 4 ms SEM; RF = 73 ± 4 ms SEM; see Table 4-12). Furthermore, a significant main effect of fatigue was noted on the time of the peak MG activity relative to the time of the peak FR. That is, when fatigued, there was a later peak MG activity (68 ± 4 ms SEM; $q = 3.840; p < 0.018$) compared to when not fatigued (54 ± 4 ms SEM).

As was discussed in the previous section, MG acts eccentrically during a landing to absorb and/or dissipate the impact forces by controlling ankle joint dorsiflexion. The delay in the time of the peak MG activity relative to the time of the peak FR when fatigued, may further compound the problem associated with the delayed MG activation noted in the fatigued state and discussed in Section 4.4.2.1. That is, the subjects may be less effective in dissipating the impact of landing and, in turn, increasing the force that the ankle, knee and hip joints must dissipate. This notion is supported by the fact that when fatigued, the subjects also displayed greater ankle joint angular velocity at the time of the peak FR during a DJM, less ankle joint dorsiflexion at the time of the peak FR during a SJM, and a trend for a later MG onset relative to IC than compared to when non-fatigued (see Section 4.3.). Therefore, when fatigued, MG may be less efficient in dissipating the impact loads associated with landing and, in turn, exposing the joints superior to the ankle joint, to increased loading. However, how the changes in MG muscle activation during landing as a result of fatigue impact on patellar tendon loading remain unclear.
Although not significant, there was a trend during the DJM for a later peak RF activity relative to the time of the peak FR when fatigued (50 ± 6 ms SEM) compared to non-fatigued (27 ± 6 ms SEM). As the statistical power of the ANOVA was very low for both this fatigue effect (16.6% at \( p = 0.05 \)) and for the movement x condition interaction (31.4% at \( p = 0.05 \)), these data need to be interpreted cautiously. For this reason, further research is recommended into the effects of fatigue on the time of peak activity relative to the time of the peak FR generated during these volleyball skills. Although requiring further investigation, the trend in the present study for a later peak RF activity relative to the time of the peak FR when fatigued was supported by Edwards et al. (2001). The authors noted that fatigue resulted in a later peak RF activity time relative to IC and to the time of the peak FR during a DJM onto a sand surface. Edwards et al. (2001) postulated that the delayed RF muscle activity indicated that the subjects may be less efficient in using their knee extensors to control deceleration of their body mass during landing. This notion should be investigated as part of the recommended further research.

4.4.2.3. Muscle Burst Intensity

No significant main effect of fatigue was found on the intensity of any of the lower limb muscles when the results were pooled across movement tasks (see Table 4-13). That is, as a result of fatigue, the subjects did not increase or decrease the intensity of the muscle activity in any of the lower limb muscles during landing compared to a non-fatigued condition. Although not significant, there was a trend towards a change in the muscle intensity data, with an increase for the quadriceps muscles (VL, RF, VM) and the hamstring muscles (BF, ST), but a decrease for the plantar flexor muscle (MG) in the fatigued condition compared to the non-fatigued condition. As the statistical power of the ANOVA was very low when testing the effect of fatigue for these muscles (5% to 18.1% at \( p = 0.05 \)), these data need to be interpreted cautiously. For this reason, further research is recommended into the effects of fatigue on the muscle intensity activity generated during these volleyball skills. Although again requiring further investigation, the lack of change with respect to the muscle burst intensity data noted in the present study was consistent with the data of Streepey et al. (2000), who observed that fatigue resulted in a decrease in maximum muscle force with no change in the VL level of EMG activity when subjects performed a basketball task on both a wooden and a
Results & Discussion

composite floor. However, in contrast, the authors noted an increase in the peak muscle activity of soleus with an accompanying decrease in the maximum muscle force when landing on a wooden floor, suggesting that the type of surface may influence the muscle activation patterns and muscle loading. Similarly, other studies have reported changes in the muscle burst intensity as a result of fatigue that included a time dependent increase in integrated EMG during cycle ergometry (Housh et al., 1995), an increase in EMG amplitude in sledge jumps (Strojnik & Komi, 1998), and an increase in average EMG during 34 maximal isokinetic knee extensions (Kellis & Baltzopoulos, 1999) and during landing (Nummela et al., 1994; Viitasalo et al., 1993), and an increase in motor unit recruitment (Kellis & Baltzopoulos, 1999a; Nummela et al., 1994; Viitasalo et al., 1993). In contrast, other studies have reported a decrease in integrated EMG in tasks such as 50 repeated maximum voluntary contractions (Kouzaki et al., 1999), drop jump landings (Horita et al., 1999), maximal strength loading (Linnamo et al., 1998), explosive strength loading (Linnamo et al., 1998), and hurdle jumping (Viitasalo et al., 1993). These differences in changes to the intensity level of the muscle burst in response to fatigue further confirms the notion that fatigue response are specific to the experimental task.

In the present study, despite causing significant changes to the vertical jump height in the SJM, and some lower limb segmental motion and alignment, fatigue did not significantly alter either the duration, intensity or the recruitment timing of the lower limb muscles. However, as a result of fatigue, the peak muscle activity occurred later for VL, RF and MG and the muscle recruitment order changed. How these changes influence the loading of the knee joint during either a DJM or A SJM remain to be determined.
Chapter 5
Summary & Conclusions

5.1. SUMMARY OF RESULTS

Excessive and repetitive loading of the body and, in turn, loading of the patellar tendon, occurs in many sports, particularly those sports that involve numerous jumping and landing tasks. It is these excessive loads that have been identified as a primary extrinsic factor in the development of patellar tendinosis. It has been suggested that neuromuscular fatigue may contribute to lower limb injuries such as patellar tendinosis. However, it is not known how neuromuscular fatigue affects the landing technique of a typical Beach Volleyball movement which may, in turn, affect the ability of the patellar tendon to sustain repetitive loading or to efficiently dissipate the external loads generated during landing. The only previous study located within the literature investigating the effects of fatigue on landing in Beach Volleyball was a study by Edwards et al. (2001). Although the results of this study indicated that fatigue could affect muscle recruitment strategies during a drop jump movement, the task investigated was performed by subjects stepping from a constant height bench. That is, the movement did not include a take-off component and only examined the landing phase of the task in isolation. Further investigation is therefore warranted to investigate the effects of fatigue on landing performance during a typical Beach Volleyball movement in which the peak jump height is not held constant so that we can gain a better understanding of the relationships among fatigue, lower limb landing mechanics and possible extrinsic factors that contribute to developing patellar tendinosis. Therefore, the purpose of the study was to establish if the landing phase of a drop jump movement (DJM) performed from a bench and a spike jump movement (SJM) performed from ground level differed with respect to landing mechanics, and if fatigue induced by repetitive standing vertical jumps altered the landing mechanics of either movement task.

In contrast with Hypothesis 1, the two experimental tasks, the DJM and the SJM, significantly differed from each other with respect to the biomechanics displayed by the
Summary & Conclusions

subjects at landing. That is, during the DJM, subjects generated a significantly higher peak FR, a shorter time to the peak FR, and a faster rate of loading of the ground reaction forces during landing than when performing a SJM. Furthermore, subjects displayed significantly different segmental motion and alignment during landing between the two experimental tasks. That is, during a DJM the subjects displayed a significantly higher peak vertical jump height, less knee joint flexion, a higher knee joint angular velocity, and a more vertically aligned tibia both at IC and at the time of the peak FR compared to when performing a SJM. The subjects also displayed less tibial angular displacement during landing, more plantar flexion at the ankle joint at the time of the peak FR, a higher ankle joint angular velocity at the time of the peak FR, a more horizontally aligned foot relative to the ground at IC, and a greater foot angular velocity at the peak FR during a DJM compared to a SJM. Furthermore, differences in the synchrony of the muscle activation patterns were also evident between the two experimental movements. That is, the subjects exhibited a significantly longer muscle burst duration for RF, VM, MG and BF; an earlier muscle burst onset time relative to IC for RF and VM; an earlier peak muscle burst activity time relative to IC for VL; a later peak muscle burst activity time relative to the time of the peak FR for RF and VM; and a later muscle burst offset time relative to IC for RF and VM. Despite these between-task differences, partial support for Hypothesis 1(c) was found in that the muscle burst intensity displayed during landing did not significantly differ between the two experimental tasks. Furthermore, the muscle recruitment patterns in both experimental tasks differed from the patterns reported in previous landing studies. This reflected the different experimental task requirements between the studies, with the DJM and the SJM acting predominately in the vertical plane, in turn, requiring less need for the hamstring muscles to stabilise the knee joint during the landing compared to the more horizontal landing tasks, leading to the later recruitment of both hamstring muscles.

Overall, it is evident that the results of this study do not support the study’s initial hypothesis that the two experimental tasks, the DJM and the SJM, would not significantly differ from each other with respect to the ground reaction forces generated at landing, lower limb motion and alignment during landing in the sagittal plane, or the synchrony of the lower limb muscle activation patterns. This is of particular importance, as a modified landing task in which the take-off component of the
movement is removed, has different landing mechanics relative to a landing task that incorporates a take-off component. This clearly indicates that the two experimental tasks involve different landing mechanics and, therefore, using a DJM to replicate the landing phase of a SJM is not considered valid.

Following the fatigue protocol, the subjects in the present study were truly fatigued as indicated by a significant decrease in their standing vertical jump height and an increase in the post-fatigue blood lactate concentration. It was originally hypothesised that this fatigue induced by a series of standing vertical jumps would not significantly alter the ground reaction forces generated at landing (Hypothesis 2(a)), an hypothesis which was supported by the results of the present study. It was cautioned however, that as the statistical power of the tests examining the effects of fatigue on the ground reaction force data were low, these results should be further investigated.

Hypothesis 2(b), that fatigue would change the lower limb motion and alignment in the sagittal plane, was also partially supported by evidence of a significantly lower knee joint angular velocity at IC and a more vertically aligned tibia both at IC and at the time of the peak F_R in a fatigued compared to a non-fatigued condition. However, many of the fatigue effects were task specific. For example, when fatigued, the subjects displayed a higher foot angular velocity at the time of the peak F_R during a DJM, a higher ankle joint angular velocity at the time of the peak F_R during a DJM, less ankle joint dorsiflexion at the time of the peak F_R during a SJM, and a more vertically aligned tibia both at IC and at the time of the peak F_R during a SJM. Of particular importance was the finding that in the SJM, the subjects displayed a lower peak vertical height of the greater trochanter when fatigued, indicating a significant decline in their jumping performance compared to when non-fatigued. However, only minor changes were observed in the synchrony of the muscle activation patterns following fatigue with a later RF and VM peak activity relative to IC and a later MG peak activity relative to the time of the peak F_R. These findings offer only partial support for Hypothesis 2(c), particularly as muscle burst intensity was not significantly altered as a consequence of fatigue during landing in either experimental task.
5.2. CONCLUSIONS

Based on the findings of the present study it is concluded that the landing phases of the DJM and SJM are significantly different from each other with respect to the ground reaction forces generated at landing, segmental motion and alignment, and the synchrony of the muscle activation patterns displayed during landing. Therefore, the landing mechanics of a specific movement skill that involves a take-off component should not be evaluated in isolation. Modifying the task by removing the take-off component creates a different movement skill with a distinct landing pattern that differs from the landing phase of the task as a whole. Therefore, using a DJM to replicate the landing phase of a SJM is not valid.

It was also concluded that fatigue induced by a series of standing vertical jumps did not significantly alter the ground reaction forces generated at landing, or the duration or activation times of lower limb muscles. However, fatigue did alter lower limb motion and alignment, and the synchrony of the lower limb muscle activation patterns displayed by the subjects during landing, although most of these were task specific. In particular, fatigue resulted in a significant reduction in their peak vertical jump height achieved by subjects during the SJM. This has direct implications to both competition and training in Beach Volleyball, in which the players who repetitively perform a SJM may become fatigued, resulting in a decline in their SJM performance. Furthermore, the decrease in the subject’s peak vertical jump height provides them with less time to position their segments to control the deceleration of landing and dissipate the same high impact loads that are experienced when not fatigued. Consequently, this may expose the joints of the lower limb, particularly those superior to the ankle joint such as the knee, to increased loads. How these changes to the lower limb motion and alignment and the synchrony of lower limb muscle activation patterns during landing as a result of fatigue would impact on patellar tendon loading remains unclear and requires further investigation. Until then, an effective injury prevention and/or injury rehabilitation program cannot be designed and implemented.
5.3. RECOMMENDATIONS FOR FURTHER RESEARCH

The following recommendations for further research have been based on the findings of this present study:

(1) Further research is recommended, using an inverse dynamics approach, in an attempt to predict the patellar tendon forces during the landing phase of a SJM. This will provide more specific evidence of the loads sustained by the patellar tendon during landing and the relationship between lower limb motion, muscle recruitment strategies and patellar tendon loading during this volleyball skill.

(2) In an effort to understand factors that contribute to the development of patellar tendinosis, a prospective study following a large number of Beach Volleyball players, or other athletes at risk of developing the injury, is required to identify the predominant extrinsic and intrinsic risk factors for patellar tendinosis.
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18 April 2001

Ms S. Edwards
Department of Biomedical Science
University of Wollongong

Dear Ms Edwards,

Thank you for your response to the Ethics Committee’s requirements for your Human Research Ethics application HEOO/201 “Can we predict patellar tendon forces during dynamic landings?”.

Your response and amendments meet with the requirements of the Committee and your application was formally approved on 13/04/2001.

Yours sincerely,

Karen McRae
Secretary to the
Human Research Ethics Committee
Subject Information Package

A. PROJECT TITLE
Can we predict patellar tendon forces during dynamic landings?*

B. BACKGROUND INFORMATION
Overuse knee injuries in volleyball, predominantly patellar tendinosis, have increased dramatically over the past 10 years. During beach volleyball, players perform repetitive jumping movements which, in turn, create high forces in the patellar tendon. These high tendon forces have been associated with the development of patellar tendinitis. Spike jump movements and fatigue have also been shown to be potential risk factors for overuse injuries, particularly patellar tendinitis. During beach volleyball players frequently perform jumping movements and the way that they land may be affected as they get tired as a match progresses. However, it is not known what factors directly affect the forces generated during a dynamic landing movement in beach volleyball.

C. PROJECT OBJECTIVES
The aim of this study is to attempt to predict patellar tendon forces generated during dynamic landings on sand and to determine the effects of fatigue on these forces. The results of the study will have implications in the design of injury prevention and rehabilitation programs for Beach Volleyball players, particularly those susceptible to patellar tendinitis.

D. STUDY REQUIREMENTS
Before testing you will be required to sign the Informed Consent Form, after you have read the Subject Information Package and had any of your questions answered. Then your height, weight, lower limb strength and lower limb dimensions (lengths and girths) will be recorded. This will be followed by preparing and placing surface electrodes on the skin of your dominant lower limb at selected muscle sites. These electrodes will be used to measure muscle activity during the landing, using standard procedures that are non-invasive. Special markers will be placed on the skin of the same lower limb on specific anatomical locations. These makers allow us to record your movement during the landing action with special cameras. Video and digital photographs will be taken and viewed by the researchers to analyse each player's landing technique.

After warming up, you will be asked to jump from a box (approximately 50 cm high) onto wet compacted sand followed by a spike jump movement also onto the sand. The sand will be placed over a special platform (force platform) to enable us to measure the forces generated on your lower limbs during landing. Jumping from the box simulates the landing phase after typical beach volleyball movements. During each movement your lower limb muscle activity, landing forces, and

*The initial intention of the study was to attempt to predict patellar tendon forces during dynamic landings. However, scrutiny of the data indicated that the errors inherent in estimating the magnitude and location of the ground reaction forces at the foot during a dynamic landing on a sand surface was too high to enable calculation of meaningful data. Therefore, the original purpose of the study was modified to exclude prediction of forces at landing.
movement pattern will be recorded. Each movement type will be repeated about 5 times to get representative results.

After the second 5 trials, you will then be fatigued (made tired) by performing repeated beach volleyball type jumps. To make sure that you are tired enough, you will have blood samples taken to test your lactate levels. To do this, the tip of your index finger will be cleansed with an alcohol swab. The surface of your finger will then be pricked using a disposable auto-lancet (sharp instrument to take blood samples). A droplet of blood will be collected using a capillary tube (very small vessel) and expressed immediately onto a reagent strip to analyse lactate levels. After the fatigue tasks you will then perform 5 jumps from a box again onto the sand or 5 spike jump landing following the same procedures described previously.

Part of the fatigue procedure will then be repeated again, after which you will perform the opposite movement type to that performed after the first fatigue effort. The opposite movement will be repeated again 5 times. After the last movement is performed, you will then have a second blood lactate sample taken to measure the post-fatigue lactate level. A total of 2 blood lactate samples will be taken. The total testing time will be approximately 3 hours.

E. RISKS, INCONVENIENCES AND DISCOMFORTS
As this study requires you to perform typical Beach Volleyball movements, minimal risks are involved. However, you will not be required to perform any movement with which you feel uncomfortable.

F. BENEFITS
This study will provide us with information on the way that fatigue effects how players land in Beach Volleyball and what factors contribute to the high patellar tendon forces sustained during landing. This information will lead to a better understanding of factors that may contribute to patellar tendinitis in Beach Volleyball and, in turn, ways that we may be able to prevent this injury.

G. FREEDOM OF CONSENT
Participation in this study is entirely voluntary. You are free to deny consent before or during the experiment. Your participation and/or withdrawal of consent will not influence your present and/or future involvement with the University of Wollongong or the Beach Volleyball associations. You have the right to withdraw from any experiment, and this right shall be preserved over and above the goals of the experiment.

H. CONFIDENTIALITY
All questions, answers, and results of this study will be treated with absolute confidentiality. Subjects will be identified in the resultant manuscripts, reports or publications by use of the subject codes only.

I. DATA AND RESULTS
All data collected during this study will be retained in a secure place after completing this study for at least five years so as to comply with the University's Code of Practice – Research.
J. ENQUIRES
Please feel free to ask any questions you have concerning the procedures used in this study. Initial contact can be made to the Chief Investigators of the study, Suzi Edwards (Master of Science Candidate, Department of Biomedical Science, University of Wollongong (Ph: (02) 4221 3881) or to Dr Julie Steele (Senior Lecturer, Department of Biomedical Science, University of Wollongong (Ph: (02) 4221 3881). For any concerns or complaints regarding the way in which the research is or has been conduct, you should contact the Secretary of the University of Wollongong Human Research Ethics Committee on (02) 4221 4457.
Appendix 3.3

UNIVERSITY OF WOLLONGONG
CONSENT FORM
CAN WE PREDICT PATELLAR TENDON FORCES DURING DYNAMIC LANDINGS?
Suzi Edwards, Dr Julie Steele & Dr Klaus Peikenkamp

I have been given information about “Can we predict patellar tendon forces during dynamic landings?” and I have discussed the research project with Suzi Edwards who is conducting this research as part of a Master of Science (Honours) thesis, supervised by Dr Julie Steele in the Department of Biomedical Science, University of Wollongong and Dr Klaus Peikenkamp, University of Munster, Germany.

I understand that, if I consent to participate in this project I will be asked to:

- have my height, weight and lower limb dimensions measured.
- perform a typical Beach Volleyball movement onto a box of sand (i.e. a spike jump) and a modified spike jump landing from a box called a drop jump landing. During both movements the researchers will measure my muscle activity, the forces that I generate upon landing, and my technique. I will repeat both movements about 5 times.
- I will then do numerous jumps and bending/extending activities of my lower limbs to make me very tired.
- the researchers will take two blood samples from the tip of my index finger to ensure that I am very tired. After these tests I will repeat the landing activities but performing the other movement (box jump landing or spike jump landing).
- the researchers will video and digitally photographed me during the project to enable my landing technique to be quantified. These images will only be viewed by the researchers.

I have been advised of the potential risks and burdens associated with this research, which include minimal risk from landing activity, although it is similar to a movement that I typically perform in a game. I may also experience slight discomfort when the blood sample is taken. I have had an opportunity to ask Suzi Edwards any questions I may have about the research and my participation. I understand that my participation in this research is voluntary, I am free to refuse to participate and I am free to withdraw from the research at any time. My refusal to participate or withdrawal of consent will not affect my relationship with the Department of Biomedical Science or my relationship with the University of Wollongong.

If I have any enquiries about the research, I can contact Suzi Edwards, 4221 3881 or Dr Julie Steele, 4221 3881. If I have any concerns or complaints regarding the way the research is or has been conducted, I can contact the Complaints Officer, Human Research Ethics Committee, University of Wollongong on (02) 4221 4457.

By signing below I am indicating my consent to participate in the research entitled “Can we predict patellar tendon forces during dynamic landings?”, conducted by Suzi Edwards as it has been described to me in the information sheet and in discussion with Suzi Edwards. I understand that the data collected from my participation will be used for a thesis, journal publication, and presentations at conferences, and I consent for it to be used in that manner.

Name (please print)

Contact Phone Number

Address

Signed

Date

Witness Name

Witness Signature

Date

Name and phone number of contact person in case of an emergency:

Name: __________________________ Phone number: __________________________
INJURY HISTORY QUESTIONNAIRE

CAN WE PREDICT PATELLAR TENDON FORCES DURING DYNAMIC LANDINGS?

The following information will be collected for purposes of the research project. All information will remain confidential.

Subject Code:_____________ Date of Birth:_____________ Sex: M or F

Occupation:_____________

DETAILS OF LEVEL OF COMPETITION/TRAINING

1. What level of beach volleyball do you compete in (circle answers)?
   AAA       AA       A       Other

2. What level of indoor volleyball do you compete in (circle answers)?
   AAA       AA       A       Other

3. How often do you compete in games/tournaments in beach volleyball per month?
   1-2       3-4       5-6       >6     Other (specify):_____________

4. How often do you compete in games/tournaments in indoor volleyball per month?
   1-2       3-4       5-6       >6     Other (specify):_____________

5. How many hours per week do you train for beach volleyball?
   1 2 3 4 5 6 7 8 Other (specify):_____

6. How many hours per week do you train for indoor volleyball?
   1 2 3 4 5 6 7 8 Other (specify):_____

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7. Do you do any other type of training specifically for beach or indoor volleyball (e.g. weight, plyometric etc)?

YES/NO

If so, lists the types of training and hours of training.

8. List any other physical activity(s) that you are currently involved in on a regular basis (more than once per week)

HISTORY OF INJURIES

1. Have you sustained any major ankle or knee injuries that required medical attention or disturbance of normal activities for two or more days? YES/NO

If yes, what injuries.

<table>
<thead>
<tr>
<th>Leg</th>
<th>Right</th>
<th>Left</th>
<th>Right</th>
<th>Left</th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type of Injury</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sport or Activity Occurred</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Level of Sport</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 5 years</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-5 years</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 12 months</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
2. Have you experienced any other lower limb or back injuries?

<table>
<thead>
<tr>
<th>Leg</th>
<th>&gt; 5 years</th>
<th>1-5 years</th>
<th>&lt; 12 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Right</td>
<td>Left</td>
<td>Right</td>
</tr>
<tr>
<td>Type of Injury</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sport or Activity Occurred</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Level of Sport</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3. Do you have any current injury or illness which may impede your participation in the task described in the Subject Information Package?


Thank You.
Force & EMG PROG

[PROGRAMSTART] emg&force_bv.prg
//look for *** to know where input needs to be changed - ie- file name/directory

[READ]

*** INPUT THE FORCE FILE NAME AND CORRECT DIRECTORIES HERE
filename="c:\suzi\bl l\emgll\Emg$#011.b11" // filename where data resides

***
filetype="TXT" // file TYPE TXT=ASCII text file
Rowstart=2
//Rowend=5001
//EveryNthRow=1
columnLabels="T","Z1","Z2","Z3","Z4","Y1","Y2","X1","X2","VL","RF","VM","S1","BF","ST","S2","G"
// T = time, then force channels, muscles - need to check order that optotrak has put channels in - prog wanting Y1-Y4, X1-X2, Z1-Z2 order??
//LabelsInRow=1 // default ==0 means no labels in row 1 of file //1==there is

[DELETEALL]
//everything will be deleted from memory except what is below in the exclude statement.
//ie. only keep in what you need to analyse in this section!
exclude="T","Z1","Z2","Z3","Z4","Y1","Y2","X1","X2"
//basically get rid of all channels except for force channels and time

[TRANSLATESCALE]

columns="Z1","Z2","Z3","Z4"
//transmode is the way to relate the data to baseline data
transmode=3
//0=translate from the mean of the original data set
//1=above but the mean of 'row' data points from START
//2=above but the mean of 'row' data points from END
//3=constant
//4=from another field eg
//if transmode= 1 or 2 then row must be defined and it
//takes on the meaning for either mode

//row=10
//row means how many samples to average from sample 1, in called in data
//to work out offset from baseline
order=1 // 0 = translates first then scales
// 1 = scales first then translates
//you can change the order in which things are done
Appendix 3.5

//***input scaling factor to convert volts from OptoTrak to Newtons so prog understands
scale=1305.483029 //scaling occurs before translation
//scaling factor Force (N) = Voltage [V]/System Sensitivity [V/N]
// Amplifier Range [pC/10V]
// = 3.83/(50,000/10)
//Force = Voltage [V] X 1/System Sensitivity
//Force = Voltage [V] x 1305.483029
sign=-1
//inverts data if negative
//sign can be positive or negative depending on whether you want to add or subtract

//you can only define transconst or transcol NOT both !!!
transconst=-1 // use a constant translation value
//transcol="refX" // use data from the column label defined here

//deleteOriginal=1 // 0=default 1 = delete the raw data from memory after
//deleteOriginal deletes the raw data from memory after you performed a task on it. //ie. the raw data will be replaced by data with zero offset etc but this will not //delete it from disk!
tag=0 // default=0 posttag applies to new data column
//1 = apply posttag to original data and use the old label on the
//new data set
posttag="_TRS"

[TRANSLATESCALE]
columns="Y1","Y2","X1","X2"
//transmode is the way to relate the data to baseline data
transmode=3
//0=translate from the mean of the original data set
//1=above but the mean of 'row' data points from START
//2=above but the mean of 'row' data points from END
//3=constant
//4=from another field eg
//if transmode= 1 or 2 then row must be defined and it
//takes on the meaning for either mode
//row=10
//row means how many samples to average from sample 1, in called in data
//to work out offset from baseline
order=1 // 0 = translates first then scales
// 1 = scales first then translates
//you can change the order in which things are done
//***input scaling factor to convert volts to Newtons so prog understands
scale=641.025641 //scaling occurs before translation
//scaling factor Force (N) = Voltage [V]/System Sensitivity [V/N]
 // Amplifier Ranger [pC/10V]
 // = 7.8/(50,000/10)
//Force = Voltage [V] x 1/System Sensitivity
//Force = Voltage [V] x 641.025641
sign=-1
//inverts data if negative
//sign can be positive or negative depending on whether you want to add or subtract

//you can only define transconst or transcol NOT both !!!
transconst=-1 // use a constant translation value
//transcol="refX" // use data from the column label defined here

//deleteOriginal=1 // 0=default 1 = delete the raw data from memory after
//deleteOriginal deletes the raw data from memory after you performed a task on it. //ie. the raw data will be replaced by data with zero offset etc but this will not //delete it from disk!
tag=0 // default=0 posttag applies to new data column
//I = apply posttag to original data and use the old label on the
//new data set
posttag="_TRS"

[KISTLER]
label="KF" // pres tag label to summed Z Y X and <label>copX <label>copY
//posttag="_X","_Y"
//NA means different things to different filetypes
columns="Z1_TRS","Z2_TRS","Z3_TRS","Z4_TRS","Y1_TRS","Y2_TRS","X1_TRS","X2_TRS"
//CentreOfPressure=1 // default is no 1== yes
//Xwidth=420 //check X = X etc
//Ywidth=700 //
//Zdepth=-61 // mm from top of working plane of the plate to the Resultant=5// gives resultant Force
//Z is the vertical component down into the plate. The 4 Z's
//Y is the long axis of the plate
//X is the narrower width of the plate
//0 = none default
//1 = YZ resultant Force
//2 = XZ
//3 = XY
//4 = all the above
//5 = XYZ + all the above
Appendix 3.5

[DELETEALL]
#include
//exclude means dont delete these labels defined here
exclude="T","Z1_TRS","Z2_TRS","Z3_TRS","Z4_TRS","Y1_TRS","Y2_TRS"
,  "X1_TRS","X2_TRS","KFZ","KFY","KFX","KFXYZf"

[WRITE]
// *** CHANGE THE FILE NAME THAT YOU WANT TO SAVE IT TO
filename="c:suzi\bl\emgl\force011.txt" // write TO File ***
//path="c:subApr\force"
filetype="TXT" // output file type = ASCII TEXT comma seperated
delimiterFormat=""," // N/A
dataFormat="12.2" //
//ext=""

[READ]
// *** INPUT THE EMG FILE NAME AND CORRECT DIRECTORIES HERE
filename="c:suzi\bl\emgl\Emg$#011.bl11" // filename where data resides

filetype="TXT" // file TYPE TXT=ASCII text file
columnLabels="T","Z1","Z2","Z3","Z4","Y1","Y2","X1","X2","VL","RF","VM","G","BF","ST","S2","G"
Rowstart=2
//LabelsInRow=1 // default ==0 means no labels in row 1 off file 1==there is

[DELETEALL]
//everything will be deleted from memory except what is below in the exclude
//statement.
//ie. only keep in what you need to analyse in this section!
exclude="Z1_TRS","Z2_TRS","Z3_TRS","Z4_TRS","VL","RF","VM","G","BF","ST" //Z = vertical force

[FILTERHIGH]
frequency=1000 // hertz default == 1
cutoff=15 // hertz
deleteOriginal=0 // 0=default 1 = delete the raw data from memory after
calculating leaving the filtered data with the old label name
tag=0
posttag="_HP"
columns="VL","RF","VM","G","BF","ST"

[RECTIFY]
columns="VL_HP","RF_HP","VM_HP","G_HP","BF_HP","ST_HP"
//exclude=
deleteOriginal=0 // 0=default 1 = delete the raw data from memory after
tag=0 // default=0 posttag applies to new data column
//1 = apply posttag to original data and use the old label on the
// new data set
Appendix 3.5

posttag="_RFY"

[FILTER]
//low pass filter
frequency=1000 //hertz default == 1
cutoff=20 // hertz
deleteOriginal=0 // 0=default 1 = delete the raw data from memory after
// calculation leaving the filtered data with the old label name
tag=0
posttag="_ENV"
columns="VL_HP_RFY","RF_HP_RFY","VM_HP_RFY","G_HP_RFY","BF_HP_RFY","ST_HP_RFY"

[RECTIFY]
columns="VL_HP_RFY_ENV","RF_HP_RFY_ENV","VM_HP_RFY_ENV","G_HP_RFY_ENV","BF_HP_RFY_ENV","ST_HP_RFY_ENV"
//exclude=
deleteOriginal=0 // 0=default 1 = delete the raw data from memory after
tag=0 // default=0 posttag applies to new data column
// 1 = apply posttag to original data and use the old label on the
// new data set
posttag="_RFY"

[TRANSLATESCALE]
columns="VL_HP_RFY_ENV_RFY","RF_HP_RFY_ENV_RFY","VM_HP_RFY_ENV_RFY","G_HP_RFY_ENV_RFY","BF_HP_RFY_ENV_RFY","ST_HP_RFY_ENV_RFY"
//transmode is the way to relate the data to baseline data
transmode=3
// 0=translate from the mean of the original data set
// 1=above but the mean of 'row' data points from START
// 2=above but the mean of 'row' data points from END
// 3=constant
// 4=from another field eg
// if transmode= 1 or 2 then row must be defined and it
// takes on the meaning for either mode

//row=10
//row means how many samples to average from sample 1, in called in data
//to work out offset from baseline
order=1 // 0 = translates first then scales
// 1 = scales first then translates
//you can change the order in which things are done

//***input scaling factor to convert volts to Newtons so prog understands
scale=1000 //scaling occurs before translation
//In DSP the number are very small so all peaks are stated as the same even
thought //they are not.
//Therefore scale all EMG data
sign=-1
//inverts data if negative
//sign can be positive or negative depending on whether you want to add or subtract

//you can only define transconst or transcol NOT both !!!
transconst=-1 // use a constant translation value
//transcol="refX" // use data from the column label defined here

//deleteOriginal=1 // 0=default 1 = delete the raw data from memory after
//deleteOriginal deletes the raw data from memory after you performed a task on it. //ie. the raw data will be replaced by data with zero offset etc but this will not //delete it from disk!
tag=0 // default=0 posttag applies to new data column
//1 = apply posttag to original data and use the old label on the
//new data set
posttag="_SCL"

[DELETEALL]
//everything will be deleted except what is in the exclude statement
exclude="VL_HP","RF_HP","VM_HP","G_HP","BF_HP","ST_HP","VL_HP_RFY_ENV_RFY_SCL","RF_HP_RFY_ENV_RFY_SCL","VM_HP_RFY_ENV_RFY_SCL","G_HP_RFY_ENV_RFY_SCL","BF_HP_RFY_ENV_RFY_SCL","ST_HP_RFY_ENV_RFY_SCL"

[READ]
// *** CHANGE THE FILE NAME THAT YOU WANT TO SAVE IT TO
filename="c:\suzi\bl\force011\force011.txt" // dummy file
filetype="TXT"
columnLabels="T","Z1","Z2","Z3","Z4","Y1","Y2","X1","X2","KFZ","KFY","KFX","KFXYZf"
LabelsInRow=1 // default ==0 means no labels in row 1 of file

[DELETEALL]
exclude="VL_HP","RF_HP","VM_HP","G_HP","BF_HP","ST_HP","VL_HP_RFY_ENV_RFY_SCL","RF_HP_RFY_ENV_RFY_SCL","VM_HP_RFY_ENV_RFY_SCL","G_HP_RFY_ENV_RFY_SCL","BF_HP_RFY_ENV_RFY_SCL","ST_HP_RFY_ENV_RFY_SCL"

[WRITE]
// *** CHANGE THE FILE NAME THAT YOU WANT TO SAVE IT TO
filename="c:\suzi\bl\progll\emg011.txt" // write TO File ***
//path="c:\subApr\emg"
filetype="TXT" // output file type = ASCII TEXT comma seperated
delimiterFormat=""," // N/A
dataFormat="8.2" //
//ext=""

[PROGRAMEND] emg&force bv.prg // terminates program execution at this point
Residual Analysis PROG

[PROGRAMSTART] residual.prg
  // calculates cut-off frequencies for kinematic data. Output goes to Excel to
  // calculate y-intercept of regression line and to pick off cut-off

[READ]
  // *** INPUT DIGITISED .DIG FILE IN HERE
  filename="c:\analysis\control\c1\optotrak\resid.csv" // filename where data
  resides
  filetype="TXT" // file TYPE DB=ParaDox
  //LabelsInRow=1
  //RowStart=2

[RESIDUAL]
  // calculates residual analysis following the protocol in Winter
  FREQUENCY=100 // Data Collection Frequency (camera speed)
  // Define the range of Frequencies to LowPass on - all variables as integers
  FREQLOWER=1
  FREQUPPER=30
  FREQGAP=1

  // [DELETEALL]
  // exclude="t_XRA","h_XRA","a_XRA","l1_XRA","l2_XRA",
  //"k_XRA","t1_XRA","t2_XRA","gt_XRA"

  [WRITE]
    // *** CHANGE THE FILE NAME THAT YOU WANT TO SAVE IT TO
    filename="c:\analysis\control\c1\optotrak\residd.txt" // write TO File
    filetype="TXT" // output file type = ASCII TEXT comma seperated
    delimiterFormat="", // N/A
    dataFormat="8.4" //

[PROGRAMEND] residual.prg /terminates program here.
Integrate PROG

[PROGRAMSTART] emg&force bv.prg
   // look for *** to know where input needs to be changed - ie- file name/directory

[READ]
   // ***INPUT THE FORCE FILE NAME AND CORRECT DIRECTORIES HERE
   filename="d:\progl\emg012.txt" // filename where data resides ***
   filetype="TXT" // file TYPE TXT=ASCII text file
   Rowstart=2
   //Rowend=5001
   //EveryNthRow=1
   columnLabels="VL_HP_RFY_ENV_RFY_SCL","RF_HP_RFY_ENV_RFY_SCL","VM_HP_RFY_ENV_RFY_SCL","G_HP_RFY_ENV_RFY_SCL","BF_HP_RFY_ENV_RFY_SCL","ST_HP_RFY_ENV_RFY_SCL"
   //LabelsInRow=1 // default ==0 means no labels in row 1 of file //1==there is

[INTEGRATE]
   column="VL"
   frequency =1000 //(Frequency of data collection)
   Row=2343,2443
   posttag="_INT"

[INTEGRATE]
   frequency =1000 //(Frequency of data collection)
   Row=2357,2457
   column="VL_HP_RFY_ENV_RFY_SCL"
   stepwidth=0.1 //(length of number of rows integrated e.g. 0.001 for 1 ms
   posttag="_INT"

[INTEGRATE]
   frequency =1000 //(Frequency of data collection)
   Row=2357,2457
   column="RF_HP_RFY_ENV_RFY_SCL"
   stepwidth=0.1 //(length of number of rows integrated e.g. 0.001 for 1 ms
   posttag="_INT"

[INTEGRATE]
   frequency =1000 //(Frequency of data collection)
   Row=2357,2457
   column="VM_HP_RFY_ENV_RFY_SCL"
   stepwidth=0.1 //(length of number of rows integrated e.g. 0.001 for 1 ms
   posttag="_INT"

// [INTEGRATE]
   //frequency =1000 //(Frequency of data collection)
   //Row=2343,2443
   //column="G_HP_RFY_ENV_RFY_SCL"
Appendix 3.7

//stepwidth=0.1 //(length of number of rows integrated e.g. 0.001 for 1 ms
//posttag="_INT"

[INTEGRATE]
frequency =1000 //(Frequency of data collection)
Row=2387,2487
column="BF_HP_RFY_ENV_RFY_SCL"
stepwidth=0.1 //(length of number of rows integrated e.g. 0.001 for 1 ms
posttag="_INT"

[INTEGRATE]
frequency =1000 //(Frequency of data collection)
Row=2306,2406
column="ST_HP_RFY_ENV_RFY_SCL"
stepwidth=0.1 //(length of number of rows integrated e.g. 0.001 for 1 ms
posttag="_INT"

[DELETEALL]
exclude="VL_HP_RFY_ENV_RFY_SCL_INT'', "RF_HP_RFY_ENV_RFY_SCL_INT'', "VM_HP_RFY_ENV_RFY_SCL_INT'', "G_HP_RFY_ENV_RFY_SCL_INT'', "BF_HP_RFY_ENV_RFY_SCL_INT'', "ST_HP_RFY_ENV_RFY_SCL_INT''

[WRITE]
// *** CHANGE THE FILE NAME THAT YOU WANT TO SAVE IT TO
filename="e:\b01\int012.txt" // write TO File ***
//path=" c :\sub Apr\emg"
filetype="TXT" // output file type = ASCII TEXT comma seperated
delimiterFormat="," // N/A
dataFormat="8.2" //
//ext=""

[PROGRAMEND] emg&force_bv.prg // terminates program execution at this point.