2.1. Sports Injuries

Injuries are a common occurrence in people who perform sporting activities as a greater force is put on the person’s body than during normal everyday living. As a result of this the chance of injury is higher\(^1\).

Abnormalities within the body which may be compensated for or which may not be symptomatic in most people may cause problems for the sports person due to the increased load put on their musculoskeletal structures during training and competition.

Common injuries obtained by the sports person include traumatic injuries such as sprains, fractures, dislocation and muscle or ligament tears as well as overuse injuries including stress fractures and inflammation of soft tissue structures\(^2\).

2.2. Anatomy Of The Knee

The knee is a large synovial joint which is frequently injured during sporting activities. The knee joint comprises the tibiofemoral joint and the patellofemoral joint\(^3\). The knee acts like a modified hinge joint with movement mainly in the sagittal plane (flexion/extension) although some rotational movements in the

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transverse (medial/lateral rotation) do occur. The knee joint contains collateral ligaments, cruciate ligaments and menisci.

![Knee Anatomy](image)

**Figure 2.1** Diagram showing the muscles and ligaments around the knee joint

The ligaments and menisci in the knee all play an important role in the knee joints mechanism. The cruciates prevent excessive forward and backward movements of the femur in relation to the tibia during flexion and extension and also help control rotational movements. The collateral ligaments help to provide medial and lateral stability as well as resist rotational forces on the knee. The menisci protect the

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articulate surface of the knee joint by reducing joint stress, and contributing to joint lubrication\(^6\).

As well as these main structures around the knee, other structures also help stabilize the knee such as the joint capsule, bursae and muscles crossing the knee.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image.png}
\caption{Diagram to show the bursae around the knee joint}
\end{figure}

The patellofemoral joint is a gliding joint between the patella and the patella groove of the femur. It consists of the patella (a triangular bone) known as the kneecap which articulates with the patellofemoral groove between the femoral condyle.

During knee flexion the patella moves along the groove, it’s movement is controlled by the quadriceps muscles (in particular the vastus medialis obliquus). Different areas of the patella articular surface come into contact with the femur.7

Certain activities increase the load on the patellofemoral joint, for example; Activities like these which increase the load aggravate the condition causing inflammation of peri patella soft tissue.

<table>
<thead>
<tr>
<th>ACTIVITY</th>
<th>FORCE THROUGH PATELLOFEMORAL JOINT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level walking</td>
<td>0.5 times body weight</td>
</tr>
<tr>
<td>Going upstairs</td>
<td>3-4 times body weight</td>
</tr>
<tr>
<td>Squats</td>
<td>7-8 times body weight</td>
</tr>
</tbody>
</table>

The knee is a very important joint in the body, being involved in locomotion and stability of the lower extremity. The knee’s involvement in locomotion causes it to be a common site of injury. These injuries can be painful and often debilitating taking an athlete out of competition.

The knee joint, which appears like a simple hinge-joint, is one of the most complex joint. Moreover, knee is more likely to be injured than is any other joint in the body.

It is made of the two main joints: Tibiofemoral (the articulation between the tibia and femur) and patellofemoral (the articulation between the patella and femur).

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The patella is a unique structure that plays a central role in the normal biomechanics of the knee. The major functions of the patella are to increase the extensor moment of the quadriceps muscle and to minimize the concentration of stress by transmitting forces evenly to the underlying bone. In other words, the main biomechanical function of the patella (knee cap) is to improve the quadriceps efficiency by increasing the lever arm of the extensor mechanism. As the knee flexes (the tibia moving on the femur), it articulates with the trochlear region of the femur along the upper two third of its posterior surface. During knee flexion the patella moves downward.

The patellofemoral articulation consists of the facets of the patella in contact with the sulcus of the anterior femur. The patella surface can include up to seven facets, three on the medial and lateral surfaces and an extra (odd) facet on the medial side. The medial retinaculum and the vastus medialis obliquus stabilize the patella medially, while the lateral retinaculum, iliotibial band and the vastus lateralis muscle stabilizes the patella laterally. The surface anatomy of each side of the patellofemoral articulation, the overall rotational anatomy of the entire


lower limb and the relationship of the surrounding muscles affect the contact between the two surfaces\textsuperscript{16}.

According to Travell and Simons (1983)\textsuperscript{17}, the quadriceps femoris muscle group is comprised of four component muscles; the vastus lateralis (VL), vastus medialis (VM), vastus inter medialis (VI) and rectus femoris (RF); which are innervated by the posterior divisions of the femoral nerve(L2, L3, L4) . The anatomical origins:

- Vastus lateralis: Greater trochanter and the lateral lip of the linea aspera of the femur.
- Vastus medialis: Intertrochanteric line and medial lip of the linea aspera of the femur.
- Vastus inter medialis: anterior and lateral surfaces of the body of the femur.
- Rectus femoris: anterior inferior iliac spine and groove superior to the acetabulum.

These muscles insert into the patella proximally in a layered fashion\textsuperscript{18,19}. The common direction of pull of the muscle fibers\textsuperscript{20}:

- Vastus lateralis (VL): 12-15 laterally in the frontal plane
- Vastus medialis longus (VML): 15-18 medially in the frontal plane
- Vastus medialis obliquus (VMO): 50-55 medially in the frontal plane


• Rectus femoris (RF): 7-10 medially in the frontal plane

According to Voight & Wieder\(^\text{21}\) (1991), the pull of the VM and VL provides dynamic patella stability. Lieb and Perry (1968) and Felder and Leeson\(^\text{22}\) (2002) concluded that the function of the VM is to maintain patella alignment and stability, in congruence with Gilleard et al.\(^\text{23}\) (1998) who suggested that inadequate medial control from the VM muscle may result in lateral displacement of the patella. Two forces act on the patella during knee movement; the first is a patellofemoral compressive force, and the second is a quadriceps muscle tension force. The patellofemoral compressive force is also known as the patellofemoral joint reaction force (PFJRF) and is the measure of the compression of the patella against the femoral condyles and depends on the angle of flexion of the knee and the muscle tension\(^\text{24}\).

Correct tracking of the patella during flexion and extension of the knee is influenced by the following forces\(^\text{25}\):

• The height of the femoral condyles and hence the depth of the femoral groove, keeping the patella “seated” and tracking correctly.


• The shape of the facets on the under surface of the patella determines the “fit” between the patella and the femoral groove.
• The medial and lateral retinaculae which keep the patella “centered” in the femoral sulcus.
• The composite angle of the pull of the quadriceps muscle group referred to as the Q-angle.
• The relative strength of the individual muscles comprising of the QF muscle.

2.3. Lateral And Medial Stabilizer Muscles Of Patella

Soft tissue structures provide both dynamic and static stabilization of the patellofemoral joint. As the VMO is an important dynamic medial stabilizer of the patella, the ITB and VL provide dynamic lateral stabilization of the patella. Despite of important role of ITB on the patella, most of studies considered only imbalance between the VMO and VL muscles.

Insall (1982) suggested that mechanism of abnormal lateral tracking of the patella is an imbalance in the activity of the VMO muscle relative to the VL. Habitual lateral tracking may produce adaptive changes, and in time the quadriceps tendon


comes to lie more to the lateral side of the knee. The VMO becomes stretched and the VL becomes contracted\textsuperscript{31}.

**Grabiner et al.** (1991) stated that the biomechanical balance between the VMO and VL is considered to be a factor influencing patellar tracking within the intercondylar notch. It is assumed that the medial tracking role of the VMO counteracts the laterally directed force of the VL on the patella\textsuperscript{32}. Lateral tracking of the patella may be due to inadequate medial control from the VMO in persons with PFPS. This inadequate control could be due to a reduction in the tension-producing capacity of the VMO or a problem with the timing of VMO activity\textsuperscript{33}.

**Souza and Gross** (1991) compared VMO/VL integrated electromyographic (IEMG) ratios of healthy subjects and patients with unilateral PFPS under isotonic and isometric quadriceps femoris muscle contractions. The results indicated VMO/VL ratios for isotonic stair climbing activities were significantly greater than VMO/VL ratios for isometric contractions and normalized VMO/VL ratios in healthy subjects were significantly greater than that of the patients. They stated that patients with PFPS may have abnormal VMO/VL activation patterns\textsuperscript{34}.

**Boucher** (1992) studied isometric maximum knee extension at 90°, 30° and 15° of knee flexion for recording the electrical activity of the VMO and VL in the subjects with and without PFPS. His results demonstrated that VMO/VL and VML/VL ratios showed no significant differences between groups and between


the three angles. They concluded that in advanced cases of patellofemoral pain syndrome the VM may even be less active relative to the VL in the last degrees of extension compared to 90°.\(^{35}\)

**Karst and Willett** (1995) also considered the onset timing of EMG activity of the VMO and VL in asymptomatic subjects and subjects with PFPS during reflex knee extension, active knee extension in non-weight-bearing and weight-bearing situations. They showed no differences between the two groups with respect to the relative timing of initial VMO and VL activity under any of three conditions tested\(^{36}\).

**Cowan et al.** (2001) considered the electromyographic (EMG) onset of the VMO and VL in the patients and control group during the functional task of stair stepping. The results showed that in the PFPS population, the EMG onset of the VL occurred before that of VMO in both the step up and step down phases of the stair-stepping task\(^{37}\).

**Owings and Grabiner** (2002) studied the activation timing and amplitude of the VMO and VL in the health subjects and patients with PFPS during maximum voluntary knee extension contractions initiated from a flexed and an extended position. Their results showed that there were no between group differences in activation timing. The activation amplitude of the VMO and VL muscles of the


PFPS subjects was altered to the greatest extent during eccentric contractions and differed significantly from that of the control group\textsuperscript{38}.

**Mellor and Hodges** (2005) demonstrated that coordination of motor units between the medial and lateral vastus muscles in people with anterior knee pain is reduced compared to people without knee pain. They believed that it confirms that motor control dysfunction is a factor in this condition and has implicated for selection of rehabilitation\textsuperscript{39}.

**Mc Clinton et al.** (2007) in a case control study compared the onset timing and activation of the VMO and VL between subjects with and without PFPS at various step heights. The result of their study demonstrated that quadriceps onset timing and activation magnitude during stair ascent was similar between two groups, regardless of step height\textsuperscript{40}.

**Moraes Santo et al.** (2007) determined difference between the VMO/VLL muscles activation during treadmill gait level and ascending to 5% degree between patients and healthy subjects. They showed no significant difference in the VMO/VL ratio between the two groups, regardless the condition. They stated that although there was no significant difference, the subjects of the control group


showed higher values in the VMO/VL ratio in two tested conditions than the subjects of the PFPS group\textsuperscript{41}.

**Santos et al** (2008) suggests that there is an imbalance in the electric activity and abnormal recruitment patterns among the VMO, VLL and VLO muscles in individuals with PFPS, with greater delay and lower amplitude of activation of the VMO in this group\textsuperscript{42}.

**Van Tiggelen et al.** (2009) showed that delayed onset of electromyographic activity of the VMO-VL is one of the contributing risk factors to the development of PFPS\textsuperscript{43}.

The other soft tissue, which influences the patella, is TFL. To my knowledge a few studies considered the electrical activity of the TFL **Banovetz et al.** (1996) suggested that the VL, TFL and rectus femoris (RF) all produce a lateral force moment on the patella and the VMO counteracts this lateral force moment\textsuperscript{44}.

**McConnel** (1996) expressed that the decrease in activity of the TFL will result in a decreased lateral pull on the patella which, because the patella is not being displaced laterally, will enhance VMO activity\textsuperscript{45}.


Smith (1997) believed that ITB tightness results in over-activity in the TFL and diminished activity in the VMO. Wheatly and Jahnke (1951) did an electromyographic study of the thigh and hip muscles. They used essentially isokinetic movements and found the vasti fire later than the rectus femoris (RF) and tensor fascia lata (TFL) in active extension in the patients.

Gregersen et al. (2006) determined whether activation patterns of the VMO, VL and TFL were affected by changes in the varus/valgus in the cyclists without a history of overuse knee injury. Their results revealed that the VMO/ VL activation ratio increased significantly and the TFL activation decreased significantly as the varus moment decreased. They suggested that everything the foot may be beneficial towards or ameliorating PFPS in cycling.

Some electromyographic studies investigated the VMO and VL muscles ratio in different exercises, such as in closed kinetic chain (ascending and descending stairs; squatting and leg press) as well as in open kinetic chain (extension isotonic and isometric contractions and knee flexion). Souza and Gross (1991) compared the electromyographic ratio (EMG) of the VMO/VL muscles between healthy and with unilateral PFPS subjects in isotonic contractions of ascending and descending stairs and maximal and submaximal ones of the femoral

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quadriceps. The healthy subjects showed significantly higher values in the VMO/VL ratio than the ones with PFPS during the isometric contractions. These authors concluded that patients with PFPS have abnormal activation patterns in the VMO/VL ratios. Boucher et al. (1992) evaluated the VMO/VL ratio in 18 subjects with and without PFPS during maximal isometric contractions in three different knee flexion angles (15°; 30° and 90°). These authors also found a significant decrease in the VMO/ VL ratio in the subjects with PFPS, especially in those with higher values of Q angle, suggesting hence, a reduction in the VMO activity. Moreover, it was reported that exercises close to the final extension could increase the muscular imbalance in the VMO/VL ratio. Conversely, Cerny did not find significant difference between healthy and with PFPS individuals when analyzing the VMO/VL ratio during a series of exercises in open and closed kinetic chain.

The author concluded that none of the exercises used selectively recruited the VMO muscle. Sheehy et al. (1998) did not find difference in the VMO/VL activation peaks ratio between healthy and with PFPS individuals during exercises of ascending and descending a step, suggesting that there is no imbalance between these muscles in both studied groups. Thus, some authors deny the idea of

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muscular imbalance between VMO and VL, questioning VMO selective strengthening programs as well as the cause of the abnormal patellar dislocation.

Jean P. Boucher et al.\textsuperscript{55} (1992) showed that no significant differences between groups or between the three angles, suggesting that all vasti measured were consistently active throughout the studied range of motion. This suggests that the neural drive was not affected in the patellofemoral pain syndrome patients. However, when the five patients showing the largest Q angles were isolated, they revealed a significantly smaller vastus medialis oblique/vastus lateralis ratio when compared to the other group. The same ratio was also significantly smaller at 15° compared to 90°. These results did demonstrate that in advanced cases of patellofemoral pain syndrome the vastus medialis may even be less active relative to the vastus lateralis in the last degrees of extension compared to 90°. Furthermore, one may suggest that in patellofemoral pain syndrome the mechanical disturbances are exhibited first, at which time the vastus medialis atrophy, if present, would have a mechanical origin.

Anatomical studies have shown that the inferior portion of the vastus medialis has muscle fibres orientated obliquely, the vastus medialis oblique (VMO) of Lieb and Perry\textsuperscript{56}. Bose et al. (1980) found at operation that patients with recurrent dislocations of the patella had a deficient VMO and Ahmed\textsuperscript{57}. Ahmed and et al.

\begin{itemize}
\end{itemize}
(1983) showed in cadaver knees that removal of VMO tension shifted the pressure zone from the centre to the lateral facet of the patella\textsuperscript{58}.

**Hanten and et al.**\textsuperscript{59} (1990) found that, during hip adduction, the electrical activity of the VMO was significantly greater than that of the VL. Differences noted during medial rotation were not significant. The results suggested that the VMO may be selectively strengthened by performing hip adduction exercises.

**Cerny**\textsuperscript{60} (1995) showed that with PFPS or without PFPS, only one exercise resulted in a higher VMO/VL activity ratio over similar exercises. The KEMR (Knee extension - Hip maximally medially rotated) showed a higher VMO/VL ratio than the KELR (Knee extension - Hip maximally laterally rotated). Exercises more commonly prescribed to enhance VMO activity over that of the VL, however, failed to selectively activate the VMO.

### 2.4. Effect Of Fatigue On Muscles

Muscle fatigue is frequently defined as a temporary loss in force- or torque-generating ability because of recent, repetitive muscle contraction\textsuperscript{61}. The development of this temporary loss of force is a complex process and results from the failure of a number of processes, including motor unit recruitment and firing rate, chemical transmission across the neuromuscular junction, propagation of the action potential along the muscle membrane and T tubules, Ca2+ release from the

\begin{itemize}
\item \textsuperscript{59} Hanten WP, Schulthies SS. (1990) Exercise effect on electromyographic activity of the vastus medialis oblique and vastus lateralis muscles. *Phys Ther*.; 70:561-5.
\end{itemize}
sarcoplasmic reticulum (SR), Ca2+ binding to troponin C, and cross-bridge cycling\textsuperscript{62}.

Muscular fatigue is considered a viable culprit to interrupt the compensatory stabilizing mechanism, as research has demonstrated its deleterious effects on knee joint laxity\textsuperscript{63,64} as well as both the afferent and efferent neuromuscular pathways\textsuperscript{65,66,67}.

In response to various exercise protocols designed to induce muscular fatigue, increases in knee joint laxity have been documented in male and female athletes\textsuperscript{68,69,70}.

Even though researchers have studied the sexes in the investigation of muscular fatigue's effects on joint laxity, exercise protocols between the sexes have varied, making comparisons by sex virtually impossible. Knee joint proprioception,


determined by examining joint kinesthesia (the ability to sense joint motion) and joint position sense (the perception of joint position), appears to be both directly and indirectly affected by muscular fatigue. Directly, muscular fatigue appears to worsen or impair expected learning improvements in joint position sense\textsuperscript{71,72} while having no apparent effect on joint kinesthesia\textsuperscript{73,74}.

Indirectly, muscular fatigue affects proprioception in that alterations in kinesthesia and joint position sense have been demonstrated secondary to increased knee joint laxity\textsuperscript{75}.

The efferent pathway, assessed by measuring balance or by examining muscle activity with EMG, appears to also be affected by muscular fatigue. After an isokinetic exercise program designed to induce muscular fatigue, healthy individuals have demonstrated decreased balance ability, suggesting that fatigue results in motor control deficits\textsuperscript{76}. EMG studies suggest that, in addition to balance, muscular fatigue affects muscle activity by extending the latency of muscle firing\textsuperscript{35} and by resulting in less efficient muscular processes\textsuperscript{77}.


In a state of fatigue the muscles surrounding the knee joint may lose the ability to protect the joint. Altered neuromuscular control due to fatigue may be reflected by changes in electromyographic (EMG) activity, joint kinematics, and vertical ground reaction force magnitudes, and these changes also may be observed as an altered lower extremity stiffness during landing. Altered stiffness during landing has been linked to a decrease in stored elastic energy and possibly a change in the sensitivity and activation of the stretch reflex. Alteration of the stiffness during drop landing due to fatigue may negatively affect neuromuscular control of the knee joint, thereby, making it more susceptible to injury.

Attarzadeh, R (2003) investigated about the effect of fatigue on the ratio of electrical activity of oblique vastus medialis and vastus lateralis in closed kinetic chain before and after training program and the results of this study showed that there is no significant change in the ratio of electrical activity of oblique vastus medialis and vastus lateralis after fatigue either before and after the training program.


Enoka (2002) defined muscle fatigue as an exercise induced reduction in the maximum force that a muscle can exert. During a fatiguing muscle contraction the EMG decreases in amplitude and the frequency at which motor units fire decreases. This is caused by the fast twitch muscle fibers fatiguing more rapidly and slow twitch fibers being recruited to compensate.

Rodacki et al. (2001) investigated the effects of fatigue on the lower extremity coordination. It was reported that knee stiffness was significantly greater in the presence of fatigue when compared to the pre-fatigue conditions. The type of fatigue seemed to be more localized than general and therefore supports the findings of Miura. Miura (2004) states that fatigue of a local joint will still allow the activation of the higher motor centers, which will allow the muscle reflexes to fire around a joint thereby increasing the stiffness and protecting the joint from injury. However, if an individual’s entire system is fatigued (general fatigue), those reflex pathways to the higher motor control centers may become compromised and make an individual more susceptible to injury.

Fatigue has been implicated in altering knee joint stiffness during landing activities. Altered stiffness during landing has been linked to a decreased in

stored elastic energy and possibly a change in the sensitivity of the activation of the stretch reflex. Alteration of neuromuscular control during landing, due to fatigue, will negatively affect the stiffness of the knee joint, thereby, making it more susceptible to injury.

Studies have suggested that differences in relative muscle forces exerted on the patella between the vastus medialis (VM) and vastus lateralis (VL) contribute to lateral patellar tracking and malalignment, which have been linked to PFPS. The lateral tracking and malalignment occurs when the lateral pull of VL is not adequately balanced by pull of the vastus medialis longus (VML) and vastus medialis oblique (VMO). This imbalance can lead to altered patellofemoral contact forces and pressures that may lead to PFP. The VMO is deemed the primary medial patellar stabilizer due to the anatomical differences between the VML and VMO.

Therefore, much effort has been directed towards identifying techniques to selectively strengthen the VMO to improve the force balance on the patella.
although the effectiveness and feasibility of selective VMO strengthening have been widely questioned\textsuperscript{97,98,99,100}.

Much of the controversy regarding the effectiveness of VMO strengthening is caused by the difficulty of isolating the treatment effect in patients. Electrical stimulation may be the only way to strengthen the VMO selectively\textsuperscript{101}, but should not be recommended unless its effect on patellofemoral joint mechanics is shown to be significant. Therefore, a theoretical analysis is needed to quantify the effect of VMO strengthening on patellofemoral joint loads to justify further studies seeking to identify techniques to selectively strengthen the VMO.

From a neuromechanical perspective, selective VMO activation can also reduce the lateral dominance of VL by either increasing the intensity of VMO relative to VL, or by initiating VMO activity prior to VL\textsuperscript{102}. In patients with PFPS, it is


speculated that VL is activated earlier than VMO\textsuperscript{103} and at a higher relative intensity\textsuperscript{104}.

But studies examining this dynamic imbalance theory have provided conflicting results\textsuperscript{105,106,107}, and the functional significance of the identified timing differences, on the order of 5 ms, has been questioned\textsuperscript{108,109}.

Methodological differences between studies and the inherent difficulty identifying muscle excitation onset and offset make reconciling differences difficult, but these studies suggest a theoretical investigation into the influence of vasti timing and intensity is warranted.

McConnel (2002) stated that the patellofemoral joint is largely a soft-tissue joint. In the first 20°, the patella is under the influence of the surrounding soft-tissue structures so that it is particularly vulnerable and susceptible to problems. After


20° of knee flexion, the bony architecture is increasingly responsible for controlling the position of the patella\textsuperscript{110}.

Stability of the patellofemoral joint involves dynamic and static stabilizers, which control movement of the patella within the trochlea, referred to as patellar tracking\textsuperscript{111}. Fibers from the iliotibial band (ITB) and vastus lateralis (VL) stabilize the patella laterally and fibers of the vastus medialis oblique (VMO) stabilize the patella medially\textsuperscript{112}.

The vastus medialis (VM) is divided into two distinct parts the longus (VML) originating from the shaft of the femur\textsuperscript{113} and the VMO originates from the adductor tubercle of the distal medial femur and inserts into the medial retinaculum and superomedial portion of the patella\textsuperscript{114}. The longus, where the fibers are oriented 15-18 degrees medially to the frontal plane and the obliquus, where the fibers are oriented 50-55 degrees medially in the frontal plane. The VML acts with the rest of the quadriceps to extend the knee. Although the VMO does not extend the knee, it is active throughout knee extension to keep the patella centered in the trochlea of the femur\textsuperscript{115}.


The VL, which is a lateral stabilizer, originates in outer surface of the greater trochanter of the femur and insert to lateral border of the patella\textsuperscript{116}. The fibers of the VL are oriented 12-15 degrees laterally in the frontal plane\textsuperscript{117}.

The ITB, which is a tendinous portion of the Tensor fascia lata (TFL) and gluteus maximus, provides dynamic lateral stabilization of the patella\textsuperscript{118}. Since the TFL muscle is a lateral stabilizer of patella and an internal rotator of hip\textsuperscript{119}, and TFL/ITB complex tightness may contribute to the development of PFPS\textsuperscript{120}, in the current study we considered to vastus lateralis (VL) and vastus medialis oblique (VMO) muscles.

\textbf{2.5. Origin And Development Of Plyometric Training}

Plyometrics is the term now applied to exercises that have their origins in Europe and were first known as ‘jump training’\textsuperscript{121}. It is widely accepted that plyometric training has its origin in the former Soviet Union as far as the early 1960’s with the scientific formalization of the training system, ‘shock training’ by Dr. Yuri Verkhoshansky\textsuperscript{122}. In the West, a certain mystique surrounded plyometrics in the early 1970’s, as it was thought that plyometrics were responsible for the Eastern bloc countries’ rapid competitiveness and growing supremacy in international


track and field athletic events\textsuperscript{123}. The term, ‘plyometrics’, was first used in 1975 by American track and field coach, Fred Wilt. The development of the term is confusing; Plyo- is derived from the Greek word pleythein, which means to increase. Plio is the Greek word for “ore”, while metric means “to measure”\textsuperscript{124}.

Dr. Verkhoshansky preferred the term ‘shock method’ instead of the more widely used term of ‘plyometric’, to differentiate between the naturally occurring plyometric actions in sport and the formal discipline he devised as a training system to develop speed-strength\textsuperscript{125}.

Plyometrics grew rapidly in popularity with coaches and athletes as exercise or drills focused on linking strength with speed of movement to produce power\textsuperscript{126}.

\textbf{2.6. The Physiology Of Plyometric Training}

Plyometric exercise are quick, powerful movements that enable a muscle to reach maximal force in the shortest possible time\textsuperscript{127}. This is achieved by using a prestretch, or countermovement, that involves the stretch-shortening cycle (SSC)\textsuperscript{128},\textsuperscript{129}. The purpose of plyometric exercises is to increase the power of

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subsequent movements by using both the natural elastic components of muscle and tendon and the reflex\textsuperscript{130}.

Peak performance in sport requires technical skill and power, where success is dependent upon the speed at which muscular force or power can be generated\textsuperscript{131}. Power combines strength and speed\textsuperscript{132}. It can be improved by increasing the amount of work or force that is produced by the muscle or by decreasing the amount of time required to produce force. The amount of time required to produce muscular force is an important variable for increasing power output. The training method which combines the speed of movement with strength is plyometrics\textsuperscript{133}.

According to Coetzee (2007), plyometric training (PT), or the combination of PT with a sport-specific training programme, have acute and chronic training responses. The acute effects of plyometric programmes include a significant increase in the 1RM leg strength and the delayed onset of muscle soreness. Chronic improvements include increases in explosive power, flight time and maximal isotonic and isometric leg muscle strength, average leg muscle endurance, isokinetic peak torque of the legs and shoulder, range of ankle motion, speed and frequency of muscle stimulation. PT programmes also seem to


significantly decrease ground contact time during sprinting activities and the amortization time during execution of plyometric exercises\(^{134}\).

Models of plyometric training according to Coetzee (2007) and Potach and Chu (2008), the production of muscular power is best explained by three proposed models: mechanical, neurophysiological and the stretch-shortening cycle.

### 2.7. The Mechanical Model

The mechanical model explains that during an eccentric muscle action, elastic energy in the musculotendinous components is increased with a rapid stretch and then stored\(^{135}\). Significant increases in concentric muscle production occur when immediately preceded by an eccentric contraction. This increase might be partly due to this storage of elastic potential energy, since the muscles are able to utilize the force produced by the series-elastic component (SEC)\(^{136}\). SEC in the muscle plays an important role in this model\(^{137}\). Even though all components of the SEC (actin and myosin filaments and tendon) are stretched when a joint is loaded, the tendon is the main contributor to muscle-tendon unit length changes and the storage of elastic potential energy\(^{138}\). To maximize the power output of the muscle, the eccentric muscle action must be followed immediately by a concentric muscle

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action\textsuperscript{139,140}. If a concentric muscle action does not occur, or if the eccentric phase is too long or requires too great a motion about the given joint, the stored elastic energy is lost as heat, and stretch reflex is not activated\textsuperscript{141,142}. For example, greater vertical jump height has been attained when the movement was preceded by a countermovement as opposed to a static jump\textsuperscript{143}.

2.8. The Neurophysiological Model

The neurophysiological model involves the potentiation (force-velocity characteristics of the contractile components change with a stretch) of the concentric muscle action by use of the myotatic or stretch reflex. The stretch reflex is the body’s involuntary response to an external stimulus that stretches the muscle\textsuperscript{144}.


Muscle spindles are amongst the special receptors that play a permanent role in the appearance of the myostatic stretch reflex. These proprioceptive organs are sensitive to the rate and magnitude of a stretch\textsuperscript{145}.

During plyometric exercise, or when the muscle is rapidly stretched, the stimulated muscle spindles cause a reflexive muscle action. The more rapidly the load is applied to the muscle, the greater the firing frequency of the spindle and resultant reflexive muscle contraction\textsuperscript{146}. This reflexive response increases the activity of the agonist muscle, and increases the amount of force for the resultant concentric muscle action\textsuperscript{147}. The rapid lengthening phase in the stretch-shortening cycle produces a more powerful subsequent movement. This is due to a higher active muscle state (greater potential energy) being reached before the concentric, shortening action, and the stretch-induced evocation of segmental reflexes that potentiate subsequent muscle activation\textsuperscript{148}.

\section*{2.9. Stretch-Shortening Cycle Model}

The repeated sequence of eccentric (lengthening) contractions followed by a concentric, explosive, powerful muscular contraction is known as the stretch-shortening cycle (SSC)\textsuperscript{149}. The SSC uses the energy-storing capacity, the


SEC and stimulation of the stretch reflex to facilitate a maximal increase in muscle recruitment over a minimal amount of time\textsuperscript{150}. An effective SSC can only be achieved if the following basic conditions are met: first, a timed preactivation of the muscles before the eccentric phase occurs; secondly, a short and fast eccentric phase; and finally, an immediate transition (minimal delay) from the eccentric to the concentric phase\textsuperscript{151}.

The SSC involves three distinct phases: the eccentric or loading phase, amortization or coupling phase, and the concentric or unloading phase. Phase One, the eccentric phase, involves preloading the agonist muscle group(s). Eccentric loading will place load upon the elastic components of the muscle fibers\textsuperscript{152}. The SEC stores elastic energy and muscle spindles are stimulated. As the muscle spindles are stretched, they send a signal to the ventral root of the spinal cord via the Type 1a afferent nerve fibers. Phase Two, the amortization phase, is the electromechanical delay between the first (eccentric) phase and third (concentric) phase where alpha motor neurons then transmit signals to the agonist muscle group.

Muscles must switch from overcoming work to acceleration in the opposite direction.

The shorter the amortization phase, the greater the amount of force production. Phase Three, the concentric phase, is the body’s response to the eccentric and


amortization phases. When the alpha neurons stimulate the agonist muscles, they produce a reflexive concentric muscle action\textsuperscript{153}. Most of the force that is produced comes from the fiber filaments sliding over each other\textsuperscript{154}. The stored elastic energy in the SEC during the eccentric phase is used to increase the force of the subsequent isolated concentric muscle action\textsuperscript{155}.

Plyometric exercises stimulate proprioceptive feedback to fine-tune for specific muscle-activation patterns. These exercises utilize the SSC, train the neuromuscular system by exposing it to increased strength loads and improve the stretch reflex\textsuperscript{156}. Increased speed of the stretch reflex and increased intensity of the subsequent muscle contraction will amount to better recruitment of additional motorunits.

The force-velocity relationship postulates that the faster a muscle is loaded or lengthened eccentrically, the greater the resultant force output will be\textsuperscript{157}.


2.10. Land-Based Plyometric Training

2.10.1. Explosive Leg Power

‘Plyometric training’ is a colloquial term used to describe quick, powerful movements using a pre-stretch, or countermovement, that involves the SSC\(^{158}\). Plyometric training (PT) is a common modality to enhance lower-extremity strength, power and stretch-shortening cycle (SSC) muscle function in healthy individuals\(^{159}\). The ability to produce force rapidly is vital to athletic performance. Increases in power output are likely to contribute to improvements in athletic performance\(^{160}\). The transfer of these plyometric effects for athletic performance is most likely dependent upon the specificity of the plyometric exercises performed. Specific plyometric exercises can be used to train the slow or fast SSC. Examples of slow SSC plyometrics include vertical jumps and box jumps. Bounding, repeated hurdle hops, and depth jumps, typically, are regarded as fast SSC movement\(^{161}\). Athletes who require power for moving in the horizontal plane (e.g. sprinters and long jumpers) mainly engage in bounding plyometric exercises, as opposed to high jumpers, basketball or volleyball players who require power to be

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exerted in a vertical direction and who perform mainly vertical jump (VJ) exercises.\textsuperscript{162}

These training adaptations are in accordance with the principle of specificity\textsuperscript{163}. In the literature appropriate plyometric training programs have been shown to increase power output\textsuperscript{164}, agility\textsuperscript{165} running velocity\textsuperscript{166} and also running economy\textsuperscript{167}.

\textbf{2.10.2. Neuromuscular Changes For Power Development}

Current literature suggests that plyometric training (PT), either alone or in combination with other typical training modalities (e.g. weight training [WT] or electromyostimulation), elicits many positive changes in the neural and musculoskeletal systems, muscle function and athletic performance of healthy individuals\textsuperscript{168}. The ability of the neuromuscular system to produce power at the highest exercise intensity, often referred as ‘muscular power’ is an important

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determinant of athletic performance. Markovic and Mikulic (2010) summarized as follows: “the adaptive changes in neuromuscular function due to PT are likely to be the result of: (I) an increased neural drive to the agonist muscles; (II) changes in the muscle activation strategies (i.e. improved inter muscular coordination); (III) changes in the mechanical characteristics of the muscle-tendon complex of plantar flexors; (IV) changes in muscle size and/or architecture; and (V) changes in single-fiber mechanics”.

Portage et al. (1999) showed that a plyometric training (PT) program could bring about significant increases in leg extensor muscle power and whole muscle fiber hypertrophy. In an eight-week, three day per week plyometric and aerobic exercise program, changes in muscle power output and fiber characteristics following this intervention were examined. A group of 19-physically active men aged 21.3 ± 1.8 years were randomly selected to either a plyometric-group or combination-group of PT and aerobic exercise. The PT consisted of vertical jumps (VJ), bounding, and 40- centimetres (cm) depth jumps. The aerobic exercise was performed at 70 percent (%) heart-rate maximum (HRmax) for 20-minutes immediately following the plyometric workouts. Muscle biopsy specimens were taken from the vastus lateralis (VL) muscle before and after training. Type I (slow twitch) and Type II (fast twitch) muscle fibers were identified and cross-sectional areas (CSA) calculated.

Peak and average muscle power output were measured using countermovement vertical jump (CMJ). No significant differences were found between the groups following training for either peak or average power. Both groups displayed


significant increases from pre-testing to post-testing for both peak and average leg extensor muscle power. The plyometric-group increased by 2.8% and 5.5%, for peak power and average power, respectively. The combination-group increased by 2.5% in peak power and 4.8% average power, respectively. VJ height improved in each group from pre-training to post-training. The plyometric-group increased peak power and average power by 2.8% and 5.5%, respectively. Each group demonstrated a significant increase in muscle fiber CSA from pre-training to post-training for Type I (plyometric-group, 4.4%; combination-group 2, 6.1%) and Type II (plyometric-group 7.8%; combination-group 2, 6.8%) fibers, with no differences between the groups. The improved CMJ and increased power output following the PT were most likely due to a combination of the enhanced motor unit recruitment patterns and increased muscle fiber CSA, caused by fiber hypertrophy in both slow twitch and fast twitch fibers.

Malisoux et al.\textsuperscript{171} (2006), on the other hand, focused on the contractile properties of single fibers of VL muscle of recreationally active men (n= 8; age: 23 ± 1 year). After eight weeks of PT induced significant increases in peak force and maximal shortening velocity in the myosin heavy chain (MHC) isoforms Type I, IIa and hybrid IIa/IIx fibers, while peak power increased significantly in all fiber types. PT significantly increased maximal leg extensor muscle force, and VJ performance was also improved 12% (p<0.01) and 13% (p<0.001), respectively. Peak force increased 19% in Type I (p<0.01), 15% in Type IIa (p<0.001), and 16% in Type IIa/IIx fibers (p<0.001). Maximal shortening velocity increased 18, 29, and 22% in Type I, IIa, and hybrid IIa/IIx fibers, respectively (p<0.001). Single-fiber CSA increased 23% in Type I (p <0.01), 22% in Type IIa (p<0.001), and 30% in Type IIa/IIx fibers (p<0.001), in VL muscle following the PT-

intervention. Potteiger et al. (1999) also reported significant increases in Type I and type II fiber CSA of the VL muscle, but these effects were of lesser magnitude (6–8%). Malisoux et al. (2006b) also found a significant increase in the proportion of type IIa fibers of the VL muscle. In contrast, Potteiger et al. (1999) did not observe any significant changes in fiber-type composition of the VL muscles.

Contradictory to the above research, Kyröläinen et al.172 (2005) found that 15-weeks of maximal-effort PT performed by recreationally active men (n=23; age 24 ± 4 years) showed no significant changes in muscle fiber type or size. Plantar flexor strength did improve with significant increases in muscle activity, but not the rate of force development (RFD) and without any changes in either the muscle fiber distributions or CSA. Although no changes were found in the maximal strength or muscle activation for knee extensor muscles, the enhancements in jumping performance were due to improved joint control and increased RFD at the knee joint.

In contrast, Kubo et al.173 (2007) showed in a 12-week comparative study of PT and WT upon untrained male participants (n=10; age: 22 ± 2 years), PT induced changes in the strength of plantar flexors, but not in that of the knee extensors. Plantar flexors showed significant hypertrophy and significant increases in maximal voluntary contraction with increased muscular activation.

Plyometric training (PT) exercises require a high level of eccentric force to stabilize and control the knee and hip joint. A high level of concentric quadriceps and hamstring muscle force development is also needed for perpetuation and

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momentum during PT movements. To determine the effect of PT on the knee extensor and flexor muscles, Wilkerson et al.\textsuperscript{174} (2004) studied the neuromuscular changes in 19-university women basketball players (age: 19 ± 1.4 years). A six-week plyometric jump training programme was completed as part of their pre-season conditioning. Concentric isokinetic peak torque of the hamstrings and quadriceps were measured before and after the intervention at 60°·s\textsuperscript{-1} and 300°·s\textsuperscript{-1}. The experimental group (n=11) completed stretching, isotonic WT and structured PT under the supervision of the researcher. The control-group (n=8) also participated in stretching, isotonic WT and a periodic performance of unstructured PT under the supervision of the team’s basketball coaches. Data was also collected from the quadriceps and hamstring muscles during a forward lunge test, called the unilateral step-down test. Results showed a significant increase for hamstrings’ peak torque at 60°·s\textsuperscript{-1} (p=0.008) in the experimental group, while only three of the eight participants in the control-group showed an increase. The hamstrings did not show a significant increase at 300°·s\textsuperscript{-1} for the experimental group. There were no significant increases in quadriceps muscle torque at either 60°·s\textsuperscript{-1} and 300°·s\textsuperscript{-1} isokinetic test velocities. Therefore, PT increased the performance capabilities of the hamstring muscles, but not the quadriceps muscles. An improvement in the hamstring muscle strength stabilizes and controls the eccentric movement through the hip and knee whilst the body is in motion.

Maffiuletti NA, et al. (2002) investigated the Effect of combined electro stimulation and plyometric training on vertical jump height. It was reported that At

week 2. MVC significantly increased (+20% knee extensors, +13% plantar flexors) as compared to baseline (< 0.05).\(^{175}\)

**Shankar, R, et al.** (2008) Studied the effect of high intensity and low intensity plyometric on vertical jump height and maximum voluntary isometric contraction in football players. Their results demonstrated that that High Intensity Plyometric training has significant effect on Vertical Jump Height and Maximum Voluntary Isometric Contraction as compared to Low Intensity Plyometric.\(^{176}\)

**Vissing et al.**\(^{177}\) (2008) showed that weight training (WT) and PT seemed to lead to similar gains in maximal strength, whereas PT induced far greater gains in muscle power. The study compared the changes in muscle strength, power, and morphology induced by WT versus PT. Young, untrained male participants (age: 25.1 ± 3.9 years) performed 12-weeks of progressive WT (n=8) or PT (n=7). Tests included 1RM incline leg press, 3RM knee extension, and 1RM knee flexion, countermovement jumping (CMJ), and ballistic incline leg press. Muscle strength increased by approximately 20–30% (1–3RM tests) (p<0.001), with WT showing a 50% greater improvement in hamstring strength than PT (p<0.01). For the 1RM inclined leg press, the WT-group increased leg strength by 29 ± 3% (p< 0.001) and PT group improved by 22 ± 5% (p <0.01) with no significant differences present between the groups. In the 3RM isolated knee extension, WT increased by 27 ±

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2% (p<0.001) and PT increased by 26 ± 5% (p<0.001). In the 1RM hamstring curl, WT increased by 33 ± 3% (p<0.001), which was larger than the 18 ± 4% improvement in PT (p<0.05). PT increased maximum CMJ height 10% and maximal power by 9% (p<0.01). PT increased maximal power in the ballistic leg press 17% (p<0.001) versus WT 4% (p<0.05); this was significantly greater than WT (p<0.01). Gains in maximal muscle strength were essentially similar between the PT and WT groups, whereas muscle power increased almost exclusively with PT-training.

Fatouros et al.\textsuperscript{178} (2000) found that athletic training combining both PT with traditional and Olympic-style weightlifting exercises showed significantly greater improvement (p<0.05) in 1RM back squat and 1RM leg press when compared with PT alone. In a 12-week intervention of three training sessions per week (3d-wk-1), 41-untrained men (age: 20.7 ± 1.96 years) were assigned to one of the four-groups: PT (n=11), WT (n=10), plyometric plus weight training (n=10), and control (n=10). WT showed greater improvements than PT in maximal leg strength measured by the leg press, whereas maximal strength measured by the back squat showed equal increases by both groups. These findings were attributed to the nature and specificity of the plyometric and weight-training exercises prescribed during the 12-week intervention.

Márk V, et al.\textsuperscript{179} (2013) investigated the effects of a short-term in-season plyometric training program on power, agility and knee extensor strength. Male soccer players from a third league team were assigned into an experimental and a


control group. The experimental group, beside its regular soccer training sessions, performed a periodized plyometric training program for six weeks. The program included two training sessions per week, and maximal intensity unilateral and bilateral plyometric exercises (total of 40 – 100 foot contacts/session) were executed. Controls participated only in the same soccer training routine, and did not perform plyometrics. Depth vertical jump height, agility (Illinois Agility Test, T Agility Test) and maximal voluntary isometric torque in knee extensors using Multicont II dynamometer were evaluated before and after the experiment. In the experimental group small but significant improvements were found in both agility tests, while depth jump height and isometric torque increments were greater. The control group did not improve in any of the measures. Results of the study indicate that plyometric training consisting of high impact unilateral and bilateral exercises induced remarkable improvements in lower extremity power and maximal knee extensor strength, and smaller improvements in soccer-specific agility. Therefore, it is concluded that short-term plyometric training should be incorporated in the in-season preparation of lower level players to improve specific performance in soccer.

In the above literature, PT induced significant improvements in neuromuscular function for power development. PT appears to enhance motor unit recruitment patterns, with increases in muscle fiber hypertrophy for optimal maximal power output. PT significantly increased maximal leg extensor muscle force, with improved CMJ performance and increased RFD at the knee joint in recreationally active males.

These changes were accompanied with increased muscle fiber CSA in whole muscle and in single fiber studies. PT has also significantly improved maximal shortening velocities of leg extensor muscles. Plyometric exercises can too optimize performance and assist with injury prevention by improving hamstring strength, eccentric control and stability of the pelvis and knee.