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Review Article

Adductor/Abductor Dysfunction Causing Patellofemoral Syndrome in Cyclists

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Abstract: Patellofemoral Pain Syndrome is the most common lower extremity musculoskeletal complaint reported by cyclists. Despite the prevalence of research, its pathogenesis and the pathophysiology responsible for this repetitive stress injury has yet to be identified. The most accepted and therefore researched theory involves patellar maltracking arising from the muscular imbalance of the vastus lateralis and vastus medialis. To date, research findings have been inconclusive and many times contradictory. Physical rehabilitation based on those findings has had mixed outcomes, both short and long term. There is a growing body of evidence linking patellofemoral pain syndrome and dysfunction of the hip musculature. This clinical commentary is to elucidate evidence correlating patellofemoral pain syndrome to the muscular dysfunction involving the femoral adductors and abductors in cyclists, thereby directly influencing the alignment and kinematics of the femur.

Keywords: Adductor Magnus, Muscle Imbalance, Lower Extremities, Repetitive Stress Injury, Vastus Medialis, Vastus Lateralis

1. Introduction

Patellofemoral Pain Syndrome (PFPS) is the most reported lower extremity musculoskeletal complaint incurred by cyclists (Holmes, 1991; Wanich et al., 2007; Clarsen, 2010; Bini, 2011). Multiple external factors have been examined such as: saddle position (Bini, 2014; Burke, 1986), foot/cleat position (Wheeler et al. 1995), training intensity (Burke, 1986; Pruitt, 2006). Internal factors such as; foot dysfunction (Ruby et al., 1992; Barton et al., 2009, 2010; Molgaard et al., 2011), knee alignment (Burke, 1986; Pruitt, 2006) and hip alignment (Ericson, 1986) have also been the subject of research. In spite of the extensive investigation. Its origin, pathogenesis and pathophysiology has not been clearly defined (Chrisman, 1986; Mirzabeigi et al., 1999; Powers et al., 2017). PFPS is among the most exasperating and challenging repetitive stress injuries (RSI) suffered by athletes and treated by medical professionals. Forty percent of the reported cases recur within 2 years (Winters et al., 2021). The general medical consensus cites PFPS resulting from an elevation in patellofemoral joint reactive forces contributing to soft tissue pathology (Goodfellow, 1976). Numerous periarticular structures (i.e. patellar tendon, subpatellar fat pad, patellar retinaculum, meniscus, subchondral bone and ligamentous structures) may contribute to the symptomatology. However, conclusive identification of the responsible structures also has not been forthcoming. Research of isolated risk factors (i.e. hip pathomechanics, patellar maltracking, patellar malalignment, Vastus Lateralis (VL)/Vastus Medialis (VM) imbalance, subtalar hyperpronation) has consistently been inconclusive and even contradictory, leading to theorize that the pathomechanics may be multifactorial in

nature, involving the possible simultaneous dysfunction of numerous structures (Powers, 2017).

The alignment and kinematics of the tibiofemoral joint may be largely dependent on the alignment and kinematics of the hip (Lee, 2003; Robinson, 2007; Powers, 2010; Nakagawa et al., 2013; Itoh et al, 2016) and foot (Tiberio, 1987; Powers, 2003b). This dual dependency may account for the knee being the most common lower extremity structure to become injured by repetitive stress (Callaghan, 2005; Wanich et al., 2007).

While Clarsen (2010) reported 36% of questioned cyclists stated they have experienced PFPS symptoms, Bini (2011) reported 50% of cyclists he questioned had suffered PFPS like symptoms at one time. Objective findings associated with PFPS may include; weak hip musculature (Clairbourne et al., 2006; Jacobs et al., 2007; Hollman et al., 2009; Winby et al., 2009), an increased Quadriceps Angle (QA) (Park, 2011; Pappas, 2012; Almeida et al, 2016), J-sign or maltracking of the patella (Heino, 2002), imbalance of the quadriceps femoris (QF) vs. hamstrings (HS) strength (Besier et al., 2009), alterations in muscle activation patterns of the VM and VL (Cowan et al., 2001; Pal et al., 2011, 2012; Dieter et al., 2014), atrophy of the VM (Pattyn et al., 2011), tight QF (Witvrouw et al., 2000; Piva, 2005), tight HS (Witvrouw et al, 2000; Piva, 2005), ankle pathomechanics (Waryasz, 2008; Paterson et al, 2016; Theisen, 2019) and foot dysfunction (Tiberio, 1987; Powers, 2003b; Barton et al., 2009, 2010; Telarolli et al., 2020; Nishizawa , 2022).

PFPS has been correlated to dysfunctions, strength imbalances and activation timing primarily in the QF musculature (Powers, 2003; Waryasz, 2008). Electromyograph (EMG) studies have focused on the VL and VM, the controllers of medial-lateral excursion of the patella (Powers, 2000, 2003, 2003b). It was theorized that this imbalance was responsible for the lateral deviation of the patella. The majority of these studies were performed with the test subject in a non-weight bearing position. Data from many of these studies reflected VL was stronger than the VM (Nunes et al., 2013; Giles et al., 2013; Miao et al., 2015). However, Hug (2015) found that EMG studies did not always provide an adequate representation of muscle force.

Muscle activation studies have also been performed testing the theory that a delay in VM activation may result in patellar maltracking (Miller, 1997; Laprade, 1998; Kooiker et al. 2014). Conflicting data was collected: Studies performed by Pal et al. (2011, 2012), Miao et al. (2015) and Dong et al. (2021) recorded a delay in VM activation in relation to the VL, while the studies conducted by Miller (1997), Laprade (1998) and Kooiker et al. (2014) did not. Researchers were then led to theorize that patellar maltracking may be a dynamic malalignment (Crossley et al., 2011; Petersen et al., 2010). The kinematics of patient's suffering from PFPS has been extensively researched and described (Powers, 1999, 2000; Shellock et al., 1989, 1993; Witonski, 1993). Using kinematic magnetic resonance imaging, with the subject weight-bearing, Powers (2003a) and Souza et al. (2010) were able to show the alteration in patellofemoral kinematics was likely due to the femur rotating beneath the patella. Additionally, in literature published by Almeida et al. (2016), he proposed the optimal method to analyze the QA was through a motion study.

With the lack of progress in resolving PFPS, researchers investigated alternative internal risk factors remote from the knee. Many studies provided substantial evidence correlating hip musculature weakness, specifically the abductors, to PFPS (Hruska, 1998; Fagan, 2008; Powers, 2010; Neumann, 2010; Miera, 2011; Barton et al., 2013; Witvrouw et al., 2013; Jellad et al., 2021). Powers (2010) reported that a dominant causative factor for PFPS may lay proximal to the knee joint involving hip pathomechanics. He made a convincing case for the importance of the abductors and external rotators in limiting excessive hip adduction and internal femoral rotation while weight bearing. Excessive adduction and internal rotation shifts the center of the knee joint medially in relation to the foot. Mascal (2003) and Robinson (2007) both correlated

pathomechanical hip movement as being responsible for excessive distal femoral movements in both the frontal and horizontal planes. Neumann (2010) reviewed the actions of the 21 muscles of the hip and the effects they have on the kinematics of the femur while their line of pull is anatomically correct. He further appreciated studying the actions muscles may have when in a pathological anatomical position and theorized the kinetic effects that may arise. He felt in depth investigation of their pathomechanics would provide knowledge benefiting the medical community in the diagnosis and treatment of lower extremity RSI.

2. Quadriceps Femoris

The rectus femoris (RF) along with the three muscles of the vastus group (VG), the medialis (VM), intermedius (VI) and lateralis (VL) form the quadriceps femoris (QF), the musculature responsible for providing the majority of the propulsive power in pedaling.

The RF has two origins, the anterior superior iliac spine and the supraacetabular groove. As it is known for its role as a knee extensor, it also contributes to hip flexion due to originating on the pelvis and crossing anterior to the hip joint, thus allowing it to contribute to hip flexion (Neumann, 2010).

The VG originates on the femoral shaft. It has two primary functions; first, is to act in conjunction with the rectus femoris in knee extension (Grob et al., 2018), second is to align and stabilize the patella within the intercondylar notch (Moore, 2017; Grob et al., 2018; Bordoni, 2022). All members of the VG originate on the shaft of the femur, however, the VM also has origins on the adductor longus and magnus (Moore, 2017; Grob et al., 2018; Bordoni, 2022). The VL is the strongest of the QF providing approximately 40% of the overall strength generated by the QF necessary for knee extension. The balance of the force is divided between the RF and VI working in conjunction for 35% and the remaining 25% being supplied by the VM (Farahmand, 1998). It has also been reported the VM participates more in the role of a patella stabilizer, then as an active knee extensor (Han, 2010; Koh, 2011). The musculature descends to have their tendons form a continuous aponeurosis, the patellar retinaculum (medial and lateral) and combine with the RF tendon to form the patellar tendon (Moore, 2017; Grob et al., 2018; Bordoni, 2022). The patellar tendon continues inferiorly inserting on the tibial tuberosity (Moore, 2017; Grob et al., 2018; Bordoni, 2022; Biondi, 2021). Other fiber from the VL and VM combine to form the retinacular ligament, which attaches to the tibial condyles and forms the anterior capsule of the knee. Essentially, the patella is suspended by the patellar retinaculum within the intercondylar notch (Moore, 2017; Rajput, 2017; Grob et al., 2018; Borndi, 2022).

Researchers have long accepted that the imbalance between the VL and VM was responsible for the development of PFPS. EMG studies performed on cyclists found the VL and VM are most activated during the propulsive phase (12 to 6 o'clock) (Dorel et al. 2008; Jorge, 1986; Ryan, 1992), peaking just before the 3 o'clock position (Savelberg, 2003). Data also reflected the rectus femoris activated prior to top dead center and terminated between 120 to 130 degrees, functioning longer than the VG (Jorge, 1986; Ryan, 1992). Multiple studies have shown muscle activation may be altered by changes in saddle positioning; height and fore/aft orientation (Gonzalez, 1989; Ericson, 1988; Heil et al., 1995; Price, 1997; Moura et al. 2017). Additional research has shown that the muscular activation patterns, primarily the interaction between the VL and VM is directly influenced by femoral alignment (Ferrari et al., 2014; Dieter et al., 2014), the 2 most investigated muscles pertaining to PFPS.

3. Femoral Alignment

The Quadriceps Angle (QA) has become the standard by which the biomechanics of the knee joint is evaluated. It is formed by the intersection of the ray connecting the anterior superior iliac spine and the mid-patella (delineating the line of pull of the rectus femoris) and the patellar tendon (Brattstroem, 1964). The measure of this

angle is an indication of the alignment of the femur and tibia. An increase in femoral adduction and/or femoral internal rotation increases the QA (Huberti, 1984; Lee, 2003), which Pappas (2012) theorized to be a contributing risk factor for PFPS.

Without anatomical deformity or malalignment, the positioning of the femur is largely dependent on the interaction of the 21 muscles of the hip (Dostal, 1981, 1986; Neumann, 2010), the same musculature contributing to the power production in the pedaling motion (Dorel et al. 2008; Jorge, 1986; Ryan, 1992).

Commonly, runners are used more often than cyclists as test subjects in PFPS studies. Many of these studies have reported an increase in hip adduction as a possible risk factor for PFPS (Van der Worp et al., 2012; Noehren, 2013; Esculier et al., 2020).

PFPS cycling research has focused primarily on external factors; such as saddle position (Burke, 1986; Pruitt, 2006; Bini, 2011), crank length (Burke, 1986; Pruitt, 2006) or cadance (Ericson, 1988; Mornieux et al., 2007; Bini, 2011) as being the primary influence on the kinematics of the knee.

As early as 1986, Francis using high speed cinematography, observed knee adduction through the power phase of the pedal stroke (12-6 o'clock). Bailey's (2003) study supported Francis's (1986) hypothesis of knee adduction being a factor in the etiology of PFPS. Ruby et al. (1992) reported cyclists with an increased QA had altered tibiofemoral joint kinetics. It was recommended and still is to correct this malalignment using wedges at the shoe/ pedal interface.

The reactive stresses at the articular surfaces of the patella (Loudon, 2016) and load distribution between the femoral condyles on the meniscus/tibial plateau become altered (Agneskirchner et al., 2007; Willinger et al., 2019). A study performed by Powers (2010) utilizing weight-bearing kinematic magnetic resonance imaging (KMRI) suggested that increased femoral internal rotation is responsible for the appearance of an increased lateral patellar displacement as the factor for the increase in patellofemoral stress and not a vastus lateralis/ vastus medialis dysfunction.

An increased QA is an indicator for greater lateralization of stress on the patella, resulting in an increase in retropatellar pressure between the lateral femoral condyle and the lateral facet of the patella (Heino, 2003; Lee, 2003; Loudon, 2016). The research of Huberti (1984) revealed a 10% increase in QA resulted in a 45% increase in stress in the patellofemoral joint. However, as a predictor of PFPS, an increased QA has come into controversy. There have been studies that have failed to correlate an alteration in the static QA to an alteration in knee kinematics or PFPS (Park, 2011; Freedman, 2014; Silva, 2015).

4. Adductor Musculature

The adductor muscle group includes; the magnus (AMag), longus (AL) and brevis (AB). The primary function of these muscles is to bring the femur toward the body's midline in an effort to stabilize the pelvis on the femurs while bipedal standing, and to balance the torso on the weight bearing lower extremity during the midstance phase of gait (Moore, 2010). The adductor muscles may also be classified as secondary hip extensors and flexors depending on the femur's anatomical positioning (Dostal, 1986; Nemeth, 1989; Neumann, 2010; Standring, 2015; Ransom, 2022) as it moves through its arc in the sagittal plane. When the hip exceeds 40 degrees to 70 degrees of flexion the musculature crosses the medial-lateral axis, from the anterior side to the posterior side, this alteration in line of pull results in their assuming the role of effective extensors (Dostal, 1981, 1986; Hoy,1990; Kendall et al., 1993; Neumann, 2010), except the adductor magnus (AMag) (Neumann, 2010). Conversely, when the femur is in extension, their line of pull crosses from the posterior side to the anterior side transforming them into secondary thigh flexors (Dostal, 1981, 1986; Hoy, 1990; Kendall et al., 1993; Neumann, 2010). This concept, switching between flexion and extension has not been thoroughly investigated, however, it has

been alluded to by both Dostal (1981) and Hoy (1990).

Jeno (2021) suggested the AMag may have three points of origin. The pubofemoral head arises where the ramus of the pubis and ischium meet. The muscle fibers are short and extend horizontally inserting on the proximal aspect linea aspera, just medial to the gluteus maximus. It functions in the adduction of the femur and assists in hip flexion (Benn et al., 2018; Jeno, 2021). Additionally, it has been theorized, depending on hip orientation, these fibers may assist in external rotation of the thigh (Neumann, 2010; Kendall et al., 1993). The fibers from the middle head arise from the ischial ramus immediately medial to the pubofemoral origin and fan lateral to vertical inserting on the linea aspera. These fibers are the primary adducting portion of the muscle. The posterior head, originating on the ischial tuberosity inserts on the adductor tubercle of the medial femoral condyle and supracondylar line. The posterior head is an effective hip extensor regardless of the hip's orientation (Kendall et al., 1993; Platzer, 2004; Benn et al., 2018; Takizawa, 2018; Jeno, 2021). The posterior fibers may assist in medial rotation (Kendall et al., 1993). The first two heads are considered to be part of the medial compartment musculature, while the ischiocondylar portion is considered to be part of the posterior compartment also containing the hamstrings.

Studies pertaining to the adductor's secondary roles as internal and external rotators of the thigh (Reimann, 1996; Arnold, 2001; Leighton, 2006) have been inconsistent, mostly due to their being based on EMG studies. This has led to on-going controversy. The research performed by Neumann (2010) may provide a plausible explanation resulting in both sides being correct. As Neumann (2010) explained, when the femur moves through its range of motion, from neutral to flexion or extension, the adductors' line of pull crosses the medial-lateral femoral axis. This mechanically changes the line of pull, from thigh extensor to flexor and vice versa. Similarly, the fibers functioning as an external and internal rotator may also be dependent on the orientation of the musculature to the femoral axis. When the femur is in flexion the line of pull is that of internal rotation. When in extension, the line of pull converts to that of external rotation. However, as Neumann (2010) also referenced, their attachment is on the frontal plane of the femur, thereby making it unlikely they could develop enough torque to be effective at either action.

Researchers may be overlooking the contribution the AMag makes to the power production in pedaling. Traditionally it has been primarily viewed as only a hip adductor. More current literature has been providing evidence suggesting the AMag may also be functioning as a significant femur extensor (Benn et al., 2018; Corcoran et al., 2023). Enough so Broski et al. (2016) has referred to the AMag as a "mini-hamstring".

What limited research that has been conducted on the adductor group's contribution to the pedaling force (Endo et al., 2007; Richardson, 1998) found high activity in the adductors during the pedaling motion. Saito (2015) reported not only did the AMag work synergistically with the QF during the pedaling motion, it continued to work synergistically with the hamstrings during hip extension. Confirming Saito's findings, Watanabe et al. (2009) recorded EMG activity in the AMag in both the propulsive and pulling phase of the pedaling motion. Using magnetic resonance imagining (MRI) Hug et al. (2006) researched the quantitative profile for the adductor group in cyclists. He reported the anatomical cross-sectional area of the AMag in professional cyclists was greater than that of students. Also using MRI, Ema et al. (2016) concluded the adductor group may be a major contributor to the pedaling motion as it showed signs of hypertrophy.

5. Abductor Musculature

The biomechanical function of the abductor and external rotator musculature is to work eccentrically with the adductor and internal rotator musculature to stabilize the pelvis in the frontal plane while standing and ambulating (Heller, 2003; Earl, 2005;

Presswood et al., 2008; Bogey, 2017). The abductors elevate contralateral hip/pelvis as the external rotators simultaneously rotate contralateral hip medially. This combined action maintains the body's center of gravity towards the midline when the contralateral leg is non-weight bearing, while simultaneously providing stability to the hip/pelvis on the ipsilateral leg preventing it from adducting and internal rotating (Winter, 1995), such as when in the single-leg support phase of the gait cycle (Kapandji, 1983; Taunton et al., 2002; Neumann, 2010).

The Gluteus Medius (GMed), the primary hip abductor, is the main pelvis stabilizer, making it instrumental in controlling frontal and transverse movement of the femur (Lee, 1999). It may also contribute to secondary functions depending on its orientation. The anterior portion may assist in internal rotation. While the posterior portion may assist in extension and external rotation (Arnold, 2001; Reiman, 2012). However, both Neumann (2010) and Ward (2010) reported the GMed torque diminishes with increasing hip flexion, possibly leading to lessening its effect to counter femoral adduction. Neuman (2010) recorded the gluteus maximus (GM) generating less external hip torque at angles above 60 degrees rendering it ineffective to counteract the internal hip torque of the adductor musculature.

Studies focusing on the GMed has linked its dysfunction to lower extremity RSI including; PFPS (Beckman, 1995; Tyson, 1998; Fredericson et al., 2000; Schmitz, 2002; Earl, 2005; Friel et al., 2006; Wilson, 2005); lower back pain (Janda, 1987) and gait pathomechanics (Marshall, 2004).

The gluteus mini is located immediately beneath the GMed and is the smallest of the glutei. It shares the characteristics of the GMed, depending on the positioning of the thigh it may act as an internal rotator or external rotator (Beck et al., 2000; Pratt, 2004; Neumann, 2010).

Abductor musculature studies have utilized runners predominantly as test subjects. There has been very limited research investigating the abductor musculature's function in the pedaling motion. McCulloch (2018) investigated the effect different pedal systems had on abductor activation. He reported the "locked" pedaling systems allow for only a few degrees of foot abduction and adduction, referred to as "float". This restriction in movement may inhibit activation of the hip abductor musculature, thereby possibly suppressing a potential increase in power output. He continued that the abductors were more actively recruited utilizing the "float" pedals then the RF for the same power output. Finally, McCulloch suggested cyclists who have overdeveloped flexor/extensor musculature (Hug et al., 2006) with underdeveloped adductor/abductor muscles would benefit from abductor strengthening, using this as a means to reduce the possible development of iliotibial band syndrome (ITBS), a common lower extremity RSI. Callaghan (2005) found ITBS to be a common RSI among cyclists and Nath (2015) reported ITBS to be the result of dysfunctional abductors and that ITB symptoms would respond to abductor strengthening.

6. Discussion

Historically, research and treatment related to PFPS has been based on the accepted premise patellar maltracking within the intercondylar notch arising from a vastus lateralis/vastus medialis imbalance (Souza, 1991; Cowan et al., 2002; Powers, 2003; McClinton et al., 2007; Pal et al., 2011, 2012; Jibri et al., 2019). The theories for this dysfunction includes; weakness or atrophy of the VM (Møller et al., 1987; Grabiner, 1994; Taskiran et al., 1998; Panagiotopoulos, 2006), alteration in activation timing between the VL and VM (Souza, 1991; Fulkerson, 2002; Santos, 2008; Pal et al., 2011, 2012). As many of these studies have been inconclusive or contradictory, continued research has been reporting a growing body of evidence which links a possible adductor/abductor dysfunction to PFPS (McConnell et al., 1996; Magalhaes et al. 2010; Meira, 2011).

Knee adduction was detected in the early studies performed by Francis (1986,

1988). Using high speed cinematography, he was able to capture and analyze pedaling biomechanics, where he was able to visualize knee adduction during the power phase (12-6 o'clock). Later, Bailey (2003) also using video, linked excessive coronal plane knee deviation to cyclists that exhibited PFPS symptoms.

The medical and cycling communities continue to address PFPS through changing the cyclist's positioning. Primarily an alteration in saddle positioning is made, thereby changing knee kinematics (Burke, 1986; Pruitt, 2006; Zinn, 2004; Bini, 2011, 2014). However, there have been numerous studies linking a change in saddle positioning to an alteration in muscular activity. The findings of Jorge (1986) reflected an increase in saddle height may have a detrimental effect on rider performance, as the quadriceps femoris activity was shown to diminish. MacAuley (1995) reported the rectus femoris was more active at lower seat heights, while the sartorius became more active with an increase in saddle height. Similarly, Diefenthaeler et al. (2008) reported that a 1 centimeter change in seat height may produce significant adaptations in kinematics and muscle properties.

The weakness/atrophy paradigm was founded on kinematic studies performed under non-weight bearing (open kinetic chain) protocols which also included the femur being aligned and femoral movement was inhibited (Brossmann et al., 1994; Laprade, 2003; MacIntyre et al., 2006; Wittstein, 2006). Other studies which drew similar conclusions were based on data collected using cadavers (Huberti, 1984; Garg, 1990; Heegaard, 1995).

Grabiner (1994) theorized, to correct patellar maltracking involved reestablishing the VL/VM strength ratio. He advocated utilizing rehabilitation exercises which attempted to isolate or prioritize the VM from the other quadriceps musculature. Subsequent studies brought this concept into question, revealing the exercises' ineffectiveness (Cerny, 1995; Herrington, 2006; Livecchil, 2002; Lee et al., 2002). Their studies revealed the VM was not capable of being biomechanically isolated or enhanced, as the vastus muscles, in conjunction with the rectus femoris contracted in unison (Cerny, 1995; Malone, 2002; Boling et al., 2006). Further discrediting this theory, Powers (2003) performed studies utilizing kinetic magnetic resonance imaging (KMRI) with a weight bearing protocol. He reported dynamic internal rotation of the femur beneath the patella was observed and may be responsible for the "appearance" the patella was tracking abnormally. Souza et al. (2010) performed similar studies confirming Powers findings.

Ample literature correlates aberrant hip biomechanics to pathomechanics at the knee joint (Jacobs et al., 2007; Sigward, 2008; Powers, 2010; Howard et al., 2011). Accordingly, in cycling, dynamic control of the hip may be paramount to knee alignment and stability. Since the foot is stabilized by the cleat/pedal interface, when the hip adducts and/or medially rotates, the knee must respond (Imawalle et al., 2009). Patients with PFPS exhibit an increase in transverse (Souza, 2009) and frontal plane knee joint excursion (Wilson, 2008). This increase in movement may reflect a dysfunction in the adductor/abductor musculature balance. Researchers (Ireland et al., 2002; Powers, 2003, 2010; Leetun et al., 2004; Willson, 2005) have suggested that weakness in the hip abductors may be a risk factor for increased hip adduction and internal femur rotation predisposing the knee to PFPS. Leetun et al. (2004) recommends the stabilization of proximal joints as a means of injury prevention for distal joints. His study revealed hip external rotation strength was the primary predictor of lower extremity injury status.

A recent concept in rehabilitation protocols recommends patients with PFPS to have the abductor hip musculature strength addressed. The rationale for this treatment arises from research linking excessive hip movement to dysfunction of the hip musculature (Ireland et al., 2003; Robinson, 2007; Cichanowski et al., 2007; Bolgla et al., 2008; Powers, 2010; Barton et al., 2013). There is a growing body of evidence strongly

supporting strengthening the hip abductors in conjunction with the QF has a greater effect in the resolution of PFPS then, strengthening the QF alone (Dolak et al., 2011; Khayambashi et al, 2012, 2014). Other researchers suggest increasing hip abductor strength may reduce the knee adduction moment (Chang et al., 2005; Mundermann et al., 2005, 2008). In another study, Chiu et al. (2012) reported patellar tilt was not reduced with quadriceps strengthening.

A theory for the development of the adductor/abductor dysfunction may be found in data presented by Neumann (2010). He stated, when the hip is flexed above 60 degrees, the position the hips are in a majority of the time when pedaling, the abductors are less effective against the adductor torque (Neumann, 2010), lessening their effectiveness as knee joint stabilizers.

Utilizing surface EMG, Bini (2014) recorded AL activation in conjunction with the RF prior to top dead center (TDC) and continuing through the propulsive phase of the pedal stroke. Watanabe et al. (2009) reported both the AL and AMag became more actively involved in thigh extension as the other musculature responsible for thigh extension fatigued. Saito (2015) reported the AMag contraction coincided with the contractions of the QF and hamstrings during thigh extension.

Cycling studies investigating adductor usage have reported their being highly activated. Using MRI, the thigh musculature of cyclists of various levels of experience was measured (Hug et al., 2006; Ema, 2016). They reported the more experienced cyclists had hypertrophied adductors and this hypertrophied state may be an indication that it is highly likely the adductors could be participating as major hip extensors in the pedaling motion. Other studies have investigated their contribution to pedaling through an increase in oxygen consumption. (Richardson, 1998; Endo, 2007). Thus, the potential for strengthening the adductor musculature thereby creating an imbalance with the abductors may exist. Neumann (2010) observed the adductors are in constant usage as the muscles move through the multiplanar mechanical demand of pedaling. Only 2 studies were found directly examining adductor muscular development. They were evaluated in conjunction with hamstring development in cyclists (Hug et al., 2006; Ema, 2016). Hug et al. (2006) utilizing MRI compared AMag and hamstring development in professional cyclists to control subjects. He reported the AMag and biceps femoris showed greater development in cyclists. Ema (2016) also utilizing MRI found the adductor musculature to be hypertrophied among the experienced cyclists. These findings led Ema to state the adductors are major hip extensors during pedaling. Endo (2007) measuring oxygen consumption (VO2) and Gondha (2009) measuring glucose uptake reported the adductors to be considerably activated. However, the adductor musculature adaptations to pedaling have yet to be thoroughly investigated.

The hip abductors play an integral role in ambulation. They are responsible for pelvis on femur stabilization and maintaining the body's center of gravity toward the midline (Kapandji, 1983; Taunton et al., 2002; Heller, 2003; Earl, 2005).

Literature has been published linking weak femoral abductors and external rotators as potential risk factors responsible for PFPS (Cichanowski et al., 2007; Ireland et al., 2007; Dierks et al., 2008; Robinson, 2007; Bolgla et al., 2008; Boling et al., 2009; Baldon et al., 2009; Na et al., 2021). Their dysfunction has also been linked to femoroacetabular pain syndrome (Casartelli, 2011) and iliotibial band syndrome (Ferber et al., 2010; Fredericson et al., 2000). Witvrouw et al. (2000) reported adduction and internal rotation of the knee arises from weak hip abductors. Ireland et al. (2003) found PFPS subjects had a diminished abduction strength of 26% and diminished external rotation strength of 36% when compared to the control subjects. Prins (2009) reported patients with PFPS consistently tested 21% to 29% weaker in their abductor muscles than non-PFPS subjects. Hertel (2005) specifically correlated GMed dysfunction to PFPS.

Literature has been published linking hip flexion and abduction strength. Delp et al. (1999) reported; as the hip increased in flexion, the moment arm of the gluteal region musculature decreased, diminishing their contribution to abduction power. After researching effective ways to rehabilitate the gluteus maximus and gluteus medius, Distefano et al. (2009) reported the abduction strength of the GM decreased with an increase in hip flexion. And, Neumann (2010) found diminished function of the GM in angles above 60 degrees.

7. Conclusion

PFPS continues to be the most predominant lower extremity complaint among cyclists. While research has linked an adductor/abductor dysfunction in runners to this syndrome, the cycling community has yet to investigate an adductor/abductor dysfunction as a risk factor for riders.

Ample evidence, both biomechanical and clinical studies have been cited in this clinical commentary supporting the proposed theory; PFPS in cyclists may develop as a result of an adductor/abductor imbalance adversely impacting the kinematics at the knee joint. Further research should be conducted examining the role the adductors and abductors play in the pedaling mechanics.

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