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Tibial Rotation in Anterior Cruciate Ligament (ACL)-Deficient and ACL-Reconstructed Knees

Nikolaos Stergiou
University of Nebraska at Omaha, nstergiou@unomaha.edu

Stavros Ristanis
University of Ioannina

Constantina O. Moraiti
University of Ioannina

Anastasios D. Georgoulis
University of Ioannina

Tibial rotation in anterior cruciate ligament deficient and reconstructed knees: a theoretical proposition for the development of osteoarthritis.

Running Title: Tibial rotation in ACL deficient and reconstructed knees

Nicholas Stergiou, PhD, Isaacson Professor and Director of the HPER Biomechanics Laboratory, University of Nebraska at Omaha, Omaha, NE, USA

Stavros Ristanis, MD, Orthopaedic Sports Medicine Center of Ioannina, Department of Othopaedic Surgery, University of Ioannina Medical School, Ioannina, Greece

Constantina Moraiti, MD, Orthopaedic Sports Medicine Center of Ioannina, Department of Othopaedic Surgery, University of Ioannina Medical School, Ioannina, Greece

Anastasios D Georgoulis, MD, Professor of Orthopaedics, Director of the Orthopaedic Sports Medicine Center of Ioannina, Department of Othopaedic Surgery, University of Ioannina Medical School, Ioannina, Greece

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Please send all inquires to:

Anastasios D. Georgoulis, MD

Methodiou Anthrakitou 1

Ioannina 45221, P.O Box 1330

Greece

Tel/Fax: (+30) 26510- 64980

Email: oaki@cc.uoi.gr

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ABSTRACT

Excessive tibial rotation has been documented in ACL deficiency during walking. ACL reconstruction has been unable to correct this abnormality in activities that are more demanding than walking and involve both anterior and rotational loading of the knee. These findings persist regardless of graft selection for the ACL reconstruction [bone-patellar tendon-bone (BPTB) or semitendinosus-gracilis (ST/G)]. Based on this research work, we propose a theoretical perspective for the development of osteoarthritis in both the ACL deficient and the ACL reconstructed knee. We propose that excessive tibial rotation will lead to abnormal loading of the cartilage areas that are not commonly loaded in the healthy knee. Overtime this abnormal loading will lead to osteoarthritis. We hypothesize that the development of new surgical procedures and grafts, such as a more horizontally oriented femoral tunnel or a double-bundle ACL reconstruction could possibly restore tibial rotation to normal levels and prevent future knee pathology. However, *in-vivo* gait analysis studies are needed, that will examine the effects of these surgical procedures on tibial rotation. Prospective *in-vivo* and *in-vitro* studies are also necessary to verify or refute our theoretical proposition for the development of osteoarthritis.

Keywords: Osteoarthritis, gait analysis, anterior cruciate ligament, tibial rotation

1. INTRODUCTION

Approximately 250,000 anterior cruciate ligament (ACL) ruptures occur in the United States every year.[1] The majority of these injuries are sports related and they affect individuals aged from 15 to 44 years old.[2] It has been found that approximately one ACL rupture will occur for every 1500 player-hours spent practicing or competing in sports such as football, skiing, basketball and soccer.[3] Female athletes are the most likely targets. Epidemiological data suggest that females are 2 to 8 times more likely to sustain such an injury.[1] Thus, ACL rupture is a common sports related injury especially in female athletes.

Following an ACL rupture, the injured individual must make a decision either to have the ligament surgically repaired or to rehabilitate the injured knee joint without repairing the ligament. Younger individuals desiring to continue participation in competitive and recreational sports usually choose to have reconstructive ACL surgery. Individuals who do not wish to participate in high stress activities many times elect to forgo the surgical procedure or are advised to use conventional physical therapy interventions to heal their injured knee. [4-5]

However, living with a knee that is ACL deficient comes with a price. Several studies have shown that ACL deficiency leads to deterioration of the knee joint function, with development of a pathological anterior drawer, rotatory instability, poor control of muscle function, and muscle weakness.[6-9] Longitudinal follow-up studies have also shown that ACL deficiency is associated with the development of chondral injuries,[10] meniscal tears, degeneration of the articular cartilage, and eventually post-traumatic arthritis. [11-14] However, limited work has focused in identifying the exact causes for the

development of the above mentioned problems in the ACL deficient knee. Thus, the following important question remains unanswered: **is it possible that the absence of the ligament results in abnormal knee movements that eventually lead to cartilage damage?** If the answer to this question is yes, then the obvious solution to alleviate these problems it will be to proceed with an ACL reconstruction. However, this generates the following equally important question: **if abnormal knee movements exist in an ACL deficient knee, does an ACL reconstruction restore normal function?**

Even though the answers to these questions are not available, as we mentioned above, many injured athletes proceed with a reconstruction in order to return to sports and in hopes of avoiding the development of future pathology at their knees. However, a recent study with young female soccer athletes (mean age of 19 years at the time of the ACL injury) showed that these athletes eventually developed osteoarthritis 12 years after their injury, even though the ACL has been reconstructed.[15] In another study by Daniel et al,[16] with a mean follow-up of five years, an evaluation of bone scans and radiography from ACL reconstructed (91 individuals) and ACL deficient knees (145 individuals), showed that the ACL reconstructed individuals were not spared from post-traumatic pathology and they demonstrated a high prevalence of knee osteoarthritis. Asano et al[77] evaluated the articular cartilage in 105 ACL reconstructed patients and observed that all articular cartilage surfaces were significantly degenerated at a short time (15 months) after surgery. These findings suggested that ACL reconstruction can not prevent early degeneration of the knee. Thus, to the above two questions that we posed, we can practically add the following: **is it possible that cartilage damage can develop in the**

ACL reconstructed knee, because current reconstruction techniques do not fully restore normal function at the knee?

The purpose of this update is to address these questions by (1) reviewing the available literature on the *in-vivo* gait analysis research that has been conducted to assess knee joint movements (tibiofemoral kinematics) in ACL deficient and reconstructed knee; (2) to offer a theoretical perspective for the development of osteoarthritis in the ACL deficient and reconstructed knee; and (3) to discuss future directions for in-vivo gait analysis studies that will evaluate recent ACL surgical techniques which can possibly restore abnormal kinematics.

2. IN-VIVO RESEARCH TO QUANTIFY KNEE JOINT MOVEMENTS

2.1. Common Methods for Assessing Knee Joint Movement Patterns

The most widely accepted method for assessing joint movement patterns is gait analysis. Specifically, observational gait analysis is routinely performed by clinicians. However, in the present work we define gait analysis as the laboratory process by which present day electronics (i.e. video cameras, force platforms) are used to integrate information from a variety of inputs to demonstrate and analyze the dynamics of gait. Gait analysis can offer an in depth evaluation of movement patterns by providing information on each joint. Such information is necessary in domains where the effects of surgical procedures (i.e. joint arthroplasty, cerebral palsy) and rehabilitation of various movement disorders (i.e. stroke, aging) are evaluated to identify gains in mobility.[17-21]

The usage of this technology allows the development of normal joint movement profiles that can be used to identify abnormalities, helping in this way to improve diagnosis, treatment, design and performance of reconstructive surgery and rehabilitation programs. Presently, gait analysis using a 3D evaluation, can provide information regarding all six degrees of freedom of the knee joint and thus, rotational movements of the adjoining segments can be obtained (Figure 1). This is accomplished by using a number of cameras that obtain positional data from surface markers that are placed on specific anatomic bony landmarks (Figure 2), while the subject is performing a given motor task. The position of the markers in space is recorded and then, linear and angular joint kinematics and kinetics can be obtained.

INSERT FIGURES 1 AND 2 ABOUT HERE

However, gait analysis has a known drawback; the surface markers do not accurately represent the underlying bone motion,[32] since the markers are attached on the skin and not directly on the bone. As skin movement increases, the location of the marker and of the underlying bone differs. As a result, error is introduced [22, 31-35] and unfortunately this error is difficult to be removed with low-pass filtering, since its frequency content is close to that of the motion. According to Cappozzo et al,[34] the motion of the marker with respect to the underlying bone, due to skin movement ranges from a few mm up to 40 mm.

However these limitations can be partially addressed with careful experimentation procedures, such as the following: a) Minimize the inter-operator error by having the same clinician placing all markers and acquiring all anthropometric measurements. b) Incorporate a standing calibration procedure to correct for subtle misalignment of the

markers that define the local coordinate system and to provide with a definition of zero degrees for all movements in all planes. c) Maximize your control conditions to "tease" out true differences. For example, in our research work [36-37] we used as control conditions both the intact leg of the ACL reconstructed or deficient group and a completely healthy group of individuals. d) Always use the same instrumentation for all individuals to maintain the same level of measurement noise across all individuals. Thus, any differences could be attributed to changes within the system itself. e) Increase your statistical power.

Another method to obtain three-dimensional kinematics of the knee joint with accuracy is the Roentgen Stereophotogrammetric Analysis (RSA). [24-26] Recently, several researchers have tried to evaluate in-vivo kinematics during dynamic activities, using 3-D fluoroscopes to capture images [Li et al] or combine their data collected from high-speed biplane radiography with a static computed tomography (CT) [Tashman et al]. However, although RSA provides a more precise measurement of bony motion *in vivo*, it is limited by the exposure to radiation and the invasive nature of the procedure. Recently, an "open-access" magnetic resonance imaging (MRI) has been developed.[27] However, it is limited to the assessment of static activities (i.e. standing still).

Furthermore, an alternative to the usage of markers in 3D gait analysis is the employment of six-degrees-of-freedom electrogoniometers.[28-30] When using such electrogoniometers that mount on the leg surface, the accuracy of the measurement is affected by the skin and soft tissue movement and mostly by the precision by which the linkage is defined with respect to the internal bony structures of the knee joint.[22] In order to overcome these problems, researchers have used sensors attached to intra-cortical pins

inserted into the tibia and femur. However, the applicability of this method is limited, as the implantation of intracortical pins is a highly invasive procedure [31] that may cause discomfort or pain to the patient and result in restriction of movements. In addition, implantation of intracortical pins is a method that is limited by the sample size since an effective number of volunteers cannot be found.

2.2. Knee Flexion-Extension Movement Patterns in ACL Deficient Athletes

ACL deficiency and its effects on knee joint movement patterns have been investigated extensively regarding flexion-extension using gait analysis and during walking. In most cases, to achieve a thorough evaluation of the joint function, kinematic data were combined with kinetic and electromyographic data that were collected simultaneously.[28,40-43, 96-98] The combination of kinetics and kinematics allows the calculation of joint moments or torques using inverse dynamics.[44]

Using these techniques, it has been found that loss of the ACL causes excessive anterior tibial translation relative to the femur.[45] It has also been shown that athletes with ACL deficiency use stronger contraction of the hamstrings to pull the tibia posteriorly [46] or they walk with weaker contraction of the quadriceps to avoid pulling the tibia anteriorly [40,47]. Berchuck et al[47] evaluated 16 ACL deficient individuals and found consistent abnormalities in their walking pattern. At mid-stance the ACL deficient individuals were found to exhibit an external knee extension moment, requiring internal flexing moments to maintain equilibrium. This was different from the controls who

exhibited external flexion moment. The authors concluded that the ACL deficient individuals exhibited an increased internal flexing moment as a reduction effect of the force generating the extending moment, i.e. the quadriceps force. They used the term "quadriceps avoidance gait" to characterize the walking pattern of these individuals.

The exact mechanism by which avoidance of the quadriceps contraction reduces anterior tibial translation has been investigated by examining the effects of strain on cadaveric knees with a transducer placed on the ACL.[48] The ACL strain depended upon whether the knee flexion angle was changed passively or after contraction of the quadriceps muscle. Simulated isometric quadriceps contraction increased significantly the antero-medial ACL strain above the normal resting level, and through the first 45 deg of knee flexion. From 60 deg of flexion and higher, the same contractions produced decreased ACL strain. This reduction in strain was significant at 105° of flexion and at 120° of flexion. It is obvious that excessive anterior translation of the tibia during gait would be avoided if the individuals were able, either to avoid the excessive activation of the quadriceps by walking with the knee in a more extended position, or avoid the quadriceps activation when the knee is near full extension.

Furthermore, Wexler et al [43] found that 7.5 years after injury, ACL deficient individuals walked with increased knee extension angles during the late stance period. This gait pattern with the knee in a more extended position, results in lower demands placed on the quadriceps. This finding can also be considered as an additional mechanism, which produces the quadriceps avoidance gait pattern in chronic ACL deficient knees as the nervous system adapts to the injury.[47] Patel et al[42] also reported that 72% of the individuals with a quadriceps avoidance gait walked with a significantly

reduced mid-stance knee flexion angle that allowed the patient to reduce the demand placed upon the quadriceps during the stance phase. Therefore, the anterior pull on the tibia was reduced, and the knee was more stable. In the remaining 28%, the authors found an increased peak external hip flexion moment. They hypothesized that a forward lean of the trunk by these individuals produced the increase in the hip flexion moments, thereby helping to decrease the strain placed upon the quadriceps during mid-stance.

In contrast, other in-vivo studies [49-50, 98] have not verified these findings and did not observe a quadriceps avoidance gait pattern in their examined patients. Ferber et al [98] examined 10 ACL chronic deficient patients and suggested that this gait pattern may not be as common as reported in the literature. In a study by Beard et al[41] that examined ACL deficient individuals approximately 2 years after the injury, found that their subjects walked with significantly greater terminal knee flexion angle. Additionally, these individuals demonstrated a prolonged period of average hamstrings activity during the stance phase, while the duration of their quadriceps activity was similar with the controls. The authors hypothesized that the ACL deficient subjects walk with hamstrings facilitation rather than displaying quadriceps avoidance during gait.

Electromyographic studies have also supported this interpretation. Lass et al[51] found earlier onset and longer duration of quadriceps activity, but no difference in the level of quadriceps activation in ACL deficient individuals during level and inclined walking. Similarly, Kalund et al[52] found no difference in quadriceps activation, but found earlier onset in hamstring activation in ACL deficient individuals during level and inclined walking. Cicotti et al[53] reported increased vastus lateralis activity and early

hamstring muscle onset during walking and during stair ascent/descent in ACL-deficient subjects.

Maybe these differences found the literature are due to the fact that some ACL deficient patients demonstrate excellent dynamic knee stability (copers), while others demonstrate poor dynamic knee stability (non-copers).[54] It is reasonable to expect that ACL-deficient subjects with poor dynamic stability would have different movement and muscle activity patterns than those with good knee stability. The results from a study by Snyder-Mackler et al[55] also showed that only in the non-copers, the reduced external knee flexion moment was related to quadriceps femoris weakness. It is also possible that the inconsistent findings reported in the literature, are due to the absence of carefully developed experimental conditions that can measure the degree of dynamic knee stability in the subjects tested. As a result, research has been unable to identify specific abnormal movements and mechanisms that can lead to future pathology in the ACL deficient knee.

2.3. Knee Flexion-Extension Movement Patterns in ACL Reconstructed Athletes

Besides ACL deficiency, ACL reconstruction has also been investigated regarding its effects on knee flexion-extension movement patterns using gait analysis. Similarly, for a thorough evaluation of the joint function, kinematic data were combined with kinetic (i.e. joint moments) and electromyographic data that were collected simultaneously.

In ACL reconstructed individuals, it has been suggested that the time elapsed since surgery may play an important role in the return to normal gait patterns.[56-59] Devita

et al[58] examined the gait of ACL reconstructed individuals 3 weeks and 6 months post-operatively. They found a reduced but prolonged hip extensor moment pattern and a sustained knee extensor moment 3 weeks post-operatively. However, at 6 months after surgery, the ACL reconstructed individuals demonstrated knee and hip moment patterns closely resembling those of the control group, suggesting that ACL reconstructed individuals can regain pre-injury gait characteristics over time. In addition, Bush-Joseph et al[57] studied ACL reconstructed individuals 8 months post-operatively and reported only slight reductions in the peak knee extensor moment during gait. These findings are in contradiction with the work by Timoney et al[59] who reported that 10 months after surgery, ACL reconstructed individuals walked with a significantly reduced knee extensor moment as compared to controls. They suggested that not all individuals demonstrate a time-related return of normal gait patterns during the first year following ACL reconstructive surgery. Maybe the solution to this debate is the work by Bulgheroni et al[56] who examined ACL reconstructed individuals 2 years post-operatively and reported no significant differences in the flexion-extension knee or hip moments. Thus, it seems that given time, ACL reconstructed individuals can eventually regain normal knee gait patterns regarding knee flexion-extension. However, this is exactly where confusion sets in and new questions are raised. **If normal gait patterns eventually emerge in ACL reconstructed individuals, then why do they still develop cartilage damage and osteoarthritis? Is it possible that the answer may lie in the secondary planes of the joint's movement?**

2.4. Knee Movement Patterns in Secondary Planes in ACL Deficient and Reconstructed Athletes

Even though the knee flexion-extension movement patterns during gait have been extensively studied in both ACL deficient and reconstructed individuals, little is known regarding the transverse and the frontal plane movements of the tibia with respect to the femur. This is probably due to the complexity and past technical limitations of gait analysis. However, it is possible that the answers to the questions raised above, lie in this area. This speculation is enforced by a recent study from McLean et al.[60] They used subject-specific forward dynamic musculoskeletal models to identify if sagittal plane knee loading during sidestep cutting could - in isolation- injure the ACL. They reported that sagittal plane knee joint forces cannot rupture the ACL during sidestep cutting. Valgus and rotational loading is the most likely injury mechanism.

The abduction-adduction movement patterns of the knee in ACL deficient and reconstructed individuals have been examined by several investigators.[48,56,61-62] Their work has shown that increased adduction moments are usually present in such populations. Furthermore, they have linked their findings with the development of osteoarthritis in the medial compartment of the tibial plateau.[62] Our experimental work has focused in quantifying knee joint rotational movement patterns where *in-vivo* research work is scanty.

Therefore, to answer the above raised questions, our investigations have examined knee joint rotational movement patterns during high and low demanding activities in both ACL deficient and reconstructed individuals using gait analysis. In our first study, we collected kinematics from ACL deficient and reconstructed individuals during a low

demanding activity such as walking.[36] We examined 13 individuals with unilateral ACL deficiency (time from injury 7.6 ± 4.3 weeks), 21 individuals who had undergone ACL reconstruction (time from reconstruction 30 ± 16.9 weeks) and 10 healthy controls. ACL reconstruction was performed arthroscopically using a bone-patellar tendon-bone (BPTB) autograft.[63] We found that the ACL deficient group exhibited significantly increased tibial rotation range of motion, during the initial swing phase of the gait cycle, when compared with the ACL reconstructed and the control group. Thus, our results demonstrated that ACL deficiency produced rotational differences at the knee, during walking. These differences did not exist when we compared the ACL reconstructed group and the control. Therefore, the surgical reconstruction restored tibial rotation to normal levels during walking.

Next, we wanted to identify if this is also the case in a higher demanding activity that can apply increased rotational loading at the knee. Therefore, we examined 18 ACL reconstructed individuals and 15 controls during such an activity, descending stairs and subsequent pivoting.[37] The ACL reconstruction was again performed arthroscopically using a BPTB autograft. The evaluation was conducted at an average of 12 months after the reconstruction. The individuals were asked to descend 3 steps and then, immediately, pivot on the landing leg at 90 degrees and walk away from the stairway, while kinematics were collected. The tibial rotation range of motion during the pivoting period was found significantly larger in the ACL reconstructed leg as compared to the contralateral intact leg and the healthy control. No significant differences were found between the healthy control leg and the intact leg of the ACL reconstructed group. Therefore, our results demonstrated that tibial rotation remained abnormal and

significantly increased one year after ACL reconstruction during high demanding activities, such as pivoting after descending from stairs.

To verify our findings, we performed an additional experiment where we evaluated another high demanding activity.[64] Kinematics were collected while the subjects jumped off a 40cm platform and landed on the ground; following foot contact, they immediately pivoted at 90 degrees and walked away from the platform. We chose this activity as landing from a jump is a task that places higher demands on the knee, than walking or even stepping down.[65-66] We combined landing with a subsequent pivoting in order to create high rotational loads on the knee. The subjects were 11 patients, all ACL reconstructed with the same arthroscopic technique using a BPTB autograft, 1 year after the surgery, 11 ACL deficient subjects that had sustained the injury more than 1 year prior to testing, and 11 controls. The same dependent variable was evaluated as in the previous study.[37] Both the reconstructed leg of the ACL group and the deficient leg of the ACL deficient group had significantly larger tibial rotation values than the healthy control. We also found no significant differences between the deficient leg of the ACL deficient group and the reconstructed leg of the ACL reconstructed group. We concluded that current ACL reconstruction using the BPTB autograft is inadequate to restore excessive tibial rotation during an activity like landing and subsequent pivoting that practically simulates sport activities.

Next, we wanted to identify if tibial rotation remains excessive after a longer time period; two years following the reconstruction. We speculated that after a longer time period adaptations will eventually occur and the patients will compensate. Thus, we performed a follow-up evaluation[67] in 9 ACL reconstructed subjects that participated in

our previous study.[64] We examined them with the same methodology and for both activities that we used in our previous work.[37,64] We also incorporated a control group of 10 patients. We found that tibial rotation remained significantly excessive even two years after the reconstruction. This result was verified with comparisons conducted with both the intact contralateral knees of our patients and with the healthy controls. Furthermore, we found that tibial rotation of the intact knee of our patients was similar with those recorded from the control healthy group.

In all of our previous work, ACL reconstruction was performed with a BPTB autograft. Thus, it was logical to question if tibial rotation will remain excessive if an alternative autograft will be used. Such an autograft is the quadrupled hamstrings tendon (semitendinous and gracilis; ST/G). Originally, we hypothesized that the ST/G autograft will be able to restore tibial rotation during our experimental protocols, due to its superiority in strength and linear stiffness[68-71] and because it is closer morphologically to the anatomy of the natural ACL.[68-70] We examined 11 individuals, ACL reconstructed with an ST/G autograft, 11 individuals, ACL reconstructed with a BPTB autograft, and 11 healthy controls.[72] The experimental protocol was identical with our previous studies. Tibial rotation was found to be significantly larger in both ACL reconstructed groups when compared with the healthy controls and their intact contralateral knees. Therefore, our hypothesis was refuted. We concluded that ACL reconstruction using the ST/G autograft is as inadequate, in restoring excessive tibial rotation, as the one using the BPTB autograft,

The results of our studies were also supported by *in-vitro* research work, where the biomechanical efficiency of the ACL reconstruction has also been questioned.[74-76]

These studies showed that ACL reconstruction was successful in limiting anterior tibial translation in response to an anterior tibial load, but was insufficient to control a combined rotatory load of internal and valgus torque. Furthermore, our tibial rotational values were in close agreement with the *in-vitro* study by Loh et al.[74]

In summary, our research work showed that ACL deficiency results in abnormal movement patterns, such as excessive tibial rotation. ACL reconstruction seems to restore ACL function regarding tibial rotation in low demanding activities such as walking. However, this is not the case in higher loading activities such as during pivoting, immediately following a step-down or a landing from a jump. These types of activities can reveal differences that are masked during low demanding activities. In the next section we will provide with an intriguing theoretical proposition regarding the relationship of this work and cartilage damage.

3. A THEORETICAL PERSPECTIVE FOR THE DEVELOPMENT OF OSTEOARTHRITIS IN ACL DEFICIENT AND RECONSTRUCTED KNEES

Degeneration of the knee joint and eventually development of osteoarthritis has been associated with ACL deficiency. Longitudinal follow-up studies have shown that ACL deficiency leads to the development of chondral injuries,[10] meniscal tears, degeneration of the articular cartilage and eventually post-traumatic arthritis.[11-14] However, similar problems have been also found longitudinally in the ACL reconstructed knee.[16] And even more disturbingly, such findings have been seen shortly after the reconstruction as well.[77] Specifically, Asano et al[77] evaluated the articular cartilage in 105 ACL reconstructed patients and observed that all articular cartilage surfaces were

significantly degenerated at a short time (15 months) after surgery. These results showed that even ACL reconstruction cannot protect the knee from progression to degenerative change.

Here, we would like to propose a hypothesis for the development of future knee pathology not only for the ACL deficient but also the ACL reconstructed knee. Based on the results from our studies, excessive tibial rotation may be an abnormal movement mechanism that degenerates soft tissues (i.e. cartilage) resulting in osteoarthritis. We propose that since current ACL reconstruction procedures cannot replicate exactly the normal ACL anatomic complexity, they cannot restore normal tibiofemoral kinematics at the knee joint, leading this way to pathological movement patterns. These patterns also exist in the ACL deficient knees. The abnormal rotational movements of the articulating bones at the knee could result in the applications of loads at areas of the cartilage and are not commonly loaded in a healthy knee. It has been shown that normal functional loading results in increase of the resistance of the cartilage by improving the mechanical stiffness and the proteoglycan content of the tissue.[78-81] Furthermore, in joints that are prone to arthrosis it has been found that the best-preserved cartilage areas are those of higher loading.[82] Therefore, in a healthy knee there are areas that are commonly loaded and others that are not. These latter areas due to lack of sufficient cartilage may not be able to withstand the newly introduced loading which is the result of the abnormal rotational movements of the articulating bones. Over time this could lead to knee osteoarthritis.

Our hypothesis is partially supported by *in-vitro* research work. Logan et al[27] has found that in ACL deficient knees the absence of the ligament leads to anterior subluxation of the lateral plateau, while the medial tibiofemoral relationship remains

unchanged. This results in abnormal internal rotation of the tibia. In addition, Logan et al[83] has found that ACL reconstruction with an ST/G autograft did not improve the weight-bearing kinematics of the lateral compartment of the knee. The findings from these two papers are of great importance, if we will also consider which areas of the tibial plateau receive normal functional loading and thus, have healthy and well-preserved cartilage, and which areas receive sporadic loading and thus, have insufficient cartilage. Highly loaded areas at the knee are located underneath the menisci at the tibial plateau, while areas that receive little or sporadic loading are located at the lateral portion of the tibial plateau.[78] Therefore, using the information from the Logan et al research work, ACL deficiency and reconstruction will result in abnormal rotational loading of areas at the lateral compartment of the lateral plateau that were not previously loaded and thus have insufficient cartilage. Our research work showed that excessive tibial rotation is actually present *in-vivo* at the ACL deficient and reconstructed knees and thus, could result in loading of these same areas as were identified in Logan et al research work. Over time, the end result could be osteoarthritis.

With our theoretical proposition, we do not claim that the medial compartment of the tibial plateau will not be affected due to ACL deficiency and reconstruction. Changes in knee rotational movement patterns should affect all joint soft tissues. Actually, mechanisms that lead to osteoarthritis of the medial compartment, especially in cases with a later rupture of the medial meniscus, have been identified in the literature. However, our proposition is focused on the development of the osteoarthritis that is found in the lateral compartment. Noyes et al[14] has even mention that in patients without damage to the medial meniscus the development of osteoarthritis was found mostly in the

lateral compartment. Thus, our theoretical model provides an explanation for these initial observations by Noyes et al.[14]

4. RECOMMENDATIONS FOR FUTURE WORK

The ACL, except from being the primary restraint to anterior tibial translation, has also been shown to be a secondary restraint to rotational instability of the knee joint.[84] Nevertheless, only during the last few years, the rotational role of the ACL has been studied more thoroughly. Recent anatomic cadaveric studies have shown that the ACL consists of two major major bundles, the anteromedial (AM) and the posterolateral (PL) (Figure 3). The two bundles seem to exhibit different tension patterns and they seem to be susceptible to different forces. When the knee is extended, the PL bundle is tight and the AM bundle is moderately lax. As the knee is flexed, the femoral attachment of the ACL takes a more horizontal orientation, causing the AM bundle to tighten and the PM bundle to loosen up.[85] However, it seems that this structural complexity of the ACL cannot be restored with the commonly used ACL reconstruction techniques. Therefore, recent techniques have been developed to approximate better the ACL anatomy. The two most promising techniques are the two-bundle ACL reconstruction and the more oblique femoral tunnel placement.

The two-bundle reconstruction can replicate better the function of the natural ACL, due to the actual reinstatement of the true two-bundle anatomy of the ligament.[86] There is general agreement that current ACL reconstruction techniques using BTPB or ST/G grafts, anchored in one femoral and one tibial tunnel, achieve this goal partially,

because they replicate mostly the AM bundle of the ACL. The role of this bundle has been well documented as resisting anterior translational loads.[87] However, the PL bundle has received limited attention. A recent *in-vitro* study by Woo et al[88] has revealed that the PL bundle is important for the stabilization of the knee against rotational loads. So, it is possible that the lack of restoration of tibial rotation after an ACL reconstruction is related with the lack of proper replication of the two ACL bundles and specifically of the PL bundle. Recent studies in both human and animals have demonstrated similar results upon the two-bundle reconstruction technique. Radford et al[89] used an *in-vitro* sheep model and reported that the knee function after a reconstruction with a double-bundle was closer to that of the intact knee than after the single-bundle. Human clinical trials also reported similar results.[90-91] Muneta et al[90] reported the clinical results after a two-year follow-up with a two-bundle procedure in 54 patients and demonstrated good anterior stability with no serious complications. In addition, several studies[86,92-93] have evaluated the two-bundle technique using cadaveric specimens. Their conclusion was that the two-bundle technique is superior to the single-bundle in restoring tibial rotational instability. However, further investigation using *in-vivo* gait analysis, as described in our research work, is required to clearly establish this conclusion.

A more oblique placement of the femoral tunnel can also affect rotational stability.[74,94-95] This technique is not as surgically demanding as others (i.e. a two-bundle reconstruction) and the only difference from the current techniques is the setting of the femoral tunnel in a more oblique location (between 9 and 10 o'clock for a right knee). Current techniques use a vertical orientation approximately at 11 o'clock (Figure 4). Several studies used *in-vitro* methodology to examine the more obligue placement of the

femoral tunnel using either the BPTB[74,94] or the ST/G autograft.[95] They found that the more oblique placement of the femoral tunnel more effectively resisted rotational loads. This can be attributed to the fact that the PL bundle of the ACL is located more horizontally and towards the 9 o'clock of the femur (for the right leg) and is important for the stabilization of the knee against rotational loads. Thus, it is possible that a more oblique placement can better replicate the PL bundle and result in increased resistive ability to rotational forces. In our studies, the femoral tunnel was placed at the 11 o'clock position. Therefore, additional investigations using in-vivo gait analysis are required to examine tibial rotation after ACL reconstructions with both the BPTB and the ST/G graft in which the femoral tunnel is placed in a more oblique location and at 9 o'clock.

INSERT FIGURES 3 AND 4 ABOUT HERE

In summary, excessive tibial rotation has been documented in ACL deficiency. After ACL reconstruction, it has also been found in activities that are more demanding than walking and they involve both anterior and rotational loading of the knee. These findings persist regardless of graft selection. Based on this research work, we propose a theoretical perspective for the development of osteoarthritis in both ACL deficient and ACL reconstructed knees. Specifically, we propose that excessive tibial rotation will lead to abnormal loading of cartilage areas located in the lateral compartment of the tibial plateau which are not commonly loaded in the healthy knee. Overtime this abnormal loading will lead to osteoarthritis.

The present review also demonstrated the need for the improvement and development of new surgical procedures and grafts that could restore not only the pathological anterior drawer, but also the increased tibial rotation. Attempts to achieve

this, include a more horizontally oriented femoral tunnel or a double-bundle ACL reconstruction. However, *in-vivo* gait analysis studies are needed to examine the functional outcome of these different surgical procedures. Prospective *in-vivo* and *in-vitro* studies are also needed to verify or refute our theoretical proposition for the development of osteoarthritis.

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FIGURES

Figure 1

EXTERNAL – INTERNAL ROTATION

Figure 2

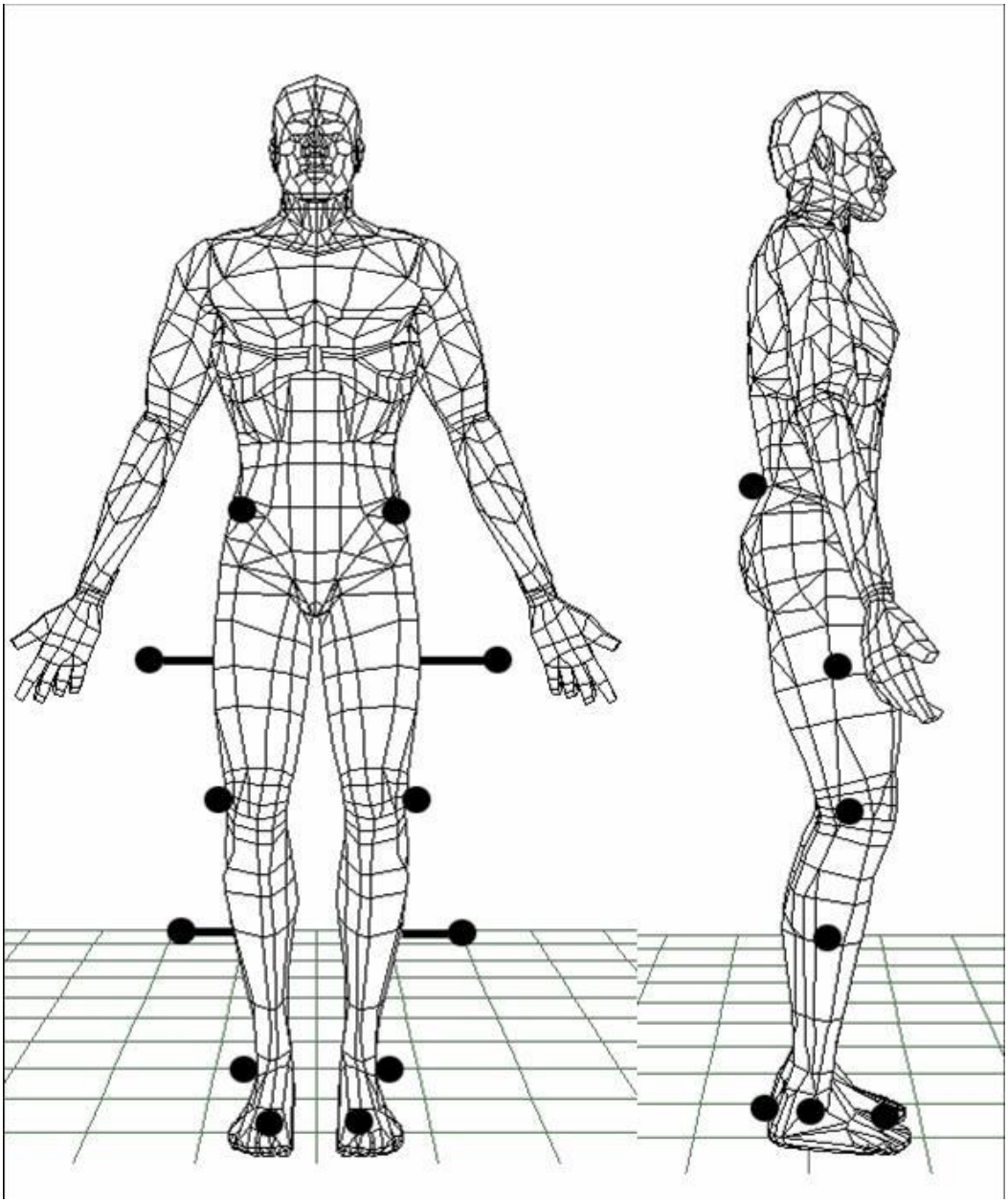

Figure 3

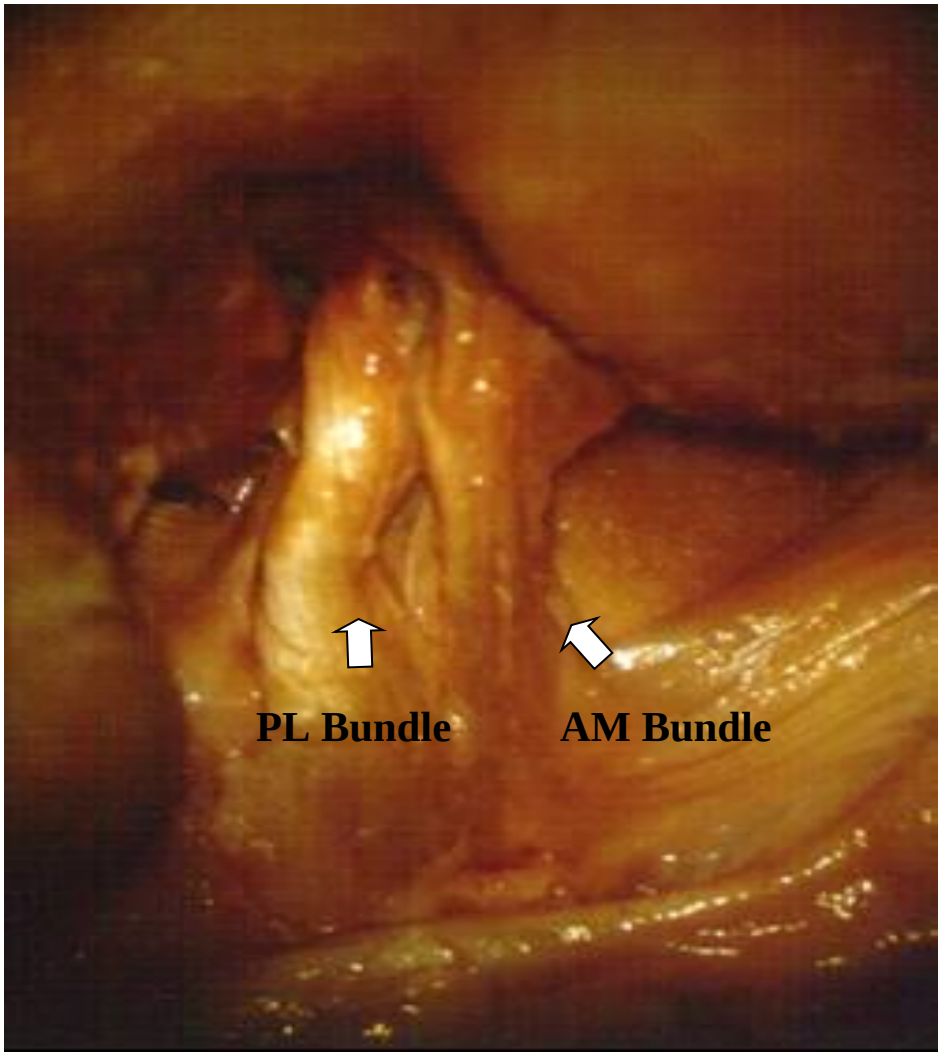

Figure 4