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Chapter

The Biomechanics of the Anterolateral Ligament

Paul Grimshaw, Christopher Jones, Merilyn Lock and Matthew Longbottom

Abstract

The anterolateral ligament (ALatL) of the knee is an under investigated soft tissue structure of the knee with its existence within the body mostly unknown to the layperson. It was determined that the ALatL has a femoral origin which is either anterior and distal, or posterior and proximal to the origin of the lateral collateral ligament, varying depending on the specimen being investigated. While there have been several studies recently conducted investigating the function of the ALatL of the knee through analysing a number of different factors including origin and insertion along with both physical and mechanical properties, there is still much mystery surrounding this ligament. Hence, further research should be conducted to accurately quantify the importance of the anterolateral ligament to internal tibial rotation stability, and the effect that a damaged anterolateral ligament can have on the stresses experienced by surrounding soft tissue structures of the knee.

Keywords: anterolateral ligament, knee, biomechanics, anterior cruciate ligament

1. Introduction

In late 2013, the world at large was told of a "new ligament" that had been discovered in the human knee, for example by ScienceDaily [1], the New York Times [2] and the British Broadcasting Corporation [3]. The subject of these stories was an anatomical paper by Claes and co-workers [4] that described the anterolateral ligament (ALatL) of the knee.

Looking at the interest generated by this paper does suggest some novelty in this description. At the time of writing, this paper had been cited by 172 other papers on PubMed. **Figure 1** shows the number of papers listed on PubMed, by year, that resulted from a search for the terms "anterolateral ligament" and "knee". The results of this search show a tremendous amount of interest by the scientific community for one small ligament.

Claes and colleagues' study was anatomical, and although it was hypothesised (not tested) that the ALatL is a restraint to internal rotation of the tibia, it could only point to the ligament's "suggested role" in anterior cruciate ligament (ACL) injury-associated instability, highlighting the need for further kinematic evidence.

Two of the reasons for this interest in the anterolateral ligament included (1) a "close association" of anterolateral and anterior cruciate ligament injuries, and (2) residual rotatory instability following anterior cruciate ligament reconstruction [5].

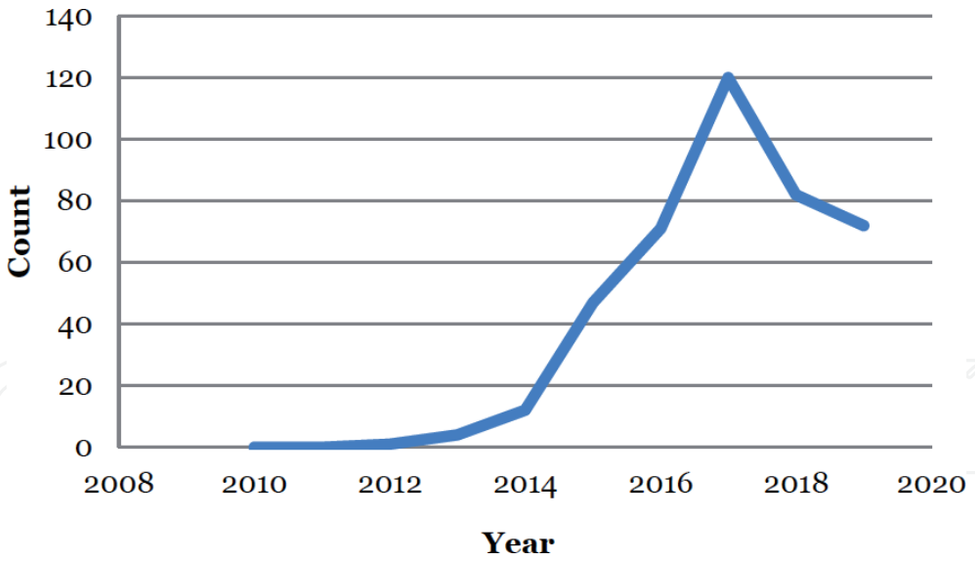

Figure 1.
Number of papers listed on PubMed, by year, that resulted from a search for the terms "anterolateral ligament" and "knee".

It is unclear just how close the association is between the ALatL and ACL injuries [5], although this is not the forum for an in depth analysis. Magnetic Resonance Imaging (MRI) is used to diagnose ALatL injuries, e.g., Claes et al. [6], but MRI analysis has yet to consistently describe the complex anatomy of the lateral side of the knee [7]. Although identification of the ligament itself on MRI appears to be a reliable process, identifying an injury to the ALatL is much less reliable [8].

Thus, it remains to be seen whether it can be established how often the ALatL is injured at the same time as the ACL, or whether an ACL-deficient knee also has an ALatL deficiency.

1.1 Background

The knee of the human body is often considered the most complex joint in the body. Major functions of the knee joint are to provide movement of the lower limb and weight bearing, distributing forces from the femur to tibia. The knee joint provides a range of movements between the femur and tibia through a number of soft tissue structures that articulate motion about the medial and lateral condyles of the two bones. The knee is capable of providing movement in six degrees of freedom with the major movement being flexion/extension with a general range of motion of 0–140°. The joint is most stable when it is in the locked, extended position, however is not mobile. Therefore, this can be the cause of many soft tissue injuries when an external force acts on the knee while it is in this extended position.

Depending on the location, magnitude and direction of the external force applied, damage to different ligaments will occur. This generally occurs when they become taut and overloaded with force. There are essentially eight major ligaments that make up the knee including the anterior cruciate ligament (ACL), posterior cruciate ligament (PCL), medial collateral ligament (MCL), lateral collateral ligament (LCL), patellar ligament, oblique popliteal ligament, arcuate popliteal ligament and the "new" anterolateral ligament (ALatL). All of these ligaments provide stability to the knee depending on their attachment and insertion sites, along with their physical and mechanical properties.

1.2 Aim

This chapter will therefore investigate the properties of the anterolateral ligament of the knee to determine the main functionality it provides. The focus of the chapter will take a biomechanical approach to investigating the anterolateral ligament through the analysis of the physical and mechanical properties. The chapter will conclude with a discussion on its possible injury mechanism and its association with the anterior cruciate ligament.

2. Literature review

Initial research has been conducted to analyse the findings of research papers investigating a number of different features of the ALatL. Firstly, the presence of the ligament is discussed with existing speculation about the existence of the anterolateral ligament in all specimens. Research presenting data about both the physical dimensions of the ligament as well as the mechanical properties will be investigated to determine the functionality of the ligament.

2.1 Presence of the anterolateral ligament

The anterolateral ligament of the knee is a recently defined ligament in the scheme of anatomical history [4], with it only recently being fully recognised as a ligament [9]. There is still confusion surrounding the existence of the ALatL in all specimens with some studies reporting the ligament being found in all subjects, while others have reported discoveries in only as little as 50% of those investigated. The contradiction between studies continues with some demonstrating the ALatL is a capsular structure which has attachment below the lateral meniscus, while others have stated it is extra-capsular, with no such attachment at this site [9].

2.2 Origin and insertion

A study conducted by Claes et al. [4] investigated 41 specimens with an ALatL able to be located in 40 of the 41 samples. In all specimens the major origin of the ligament was located to be fixed to the lateral femoral epicondyle, anterior to the origin of the LCL and proximal and posterior to the insertion of the popliteus tendon. The insertion on the anterolateral tibia was most commonly located midway between Gerdy's tubercle and the fibular head. Within this investigation, all specimens with deficiencies were excluded and knees with a damaged ACL also disregarded [4].

Kennedy et al. [5] conducted an anatomic dissection of 15 non-paired fresh-frozen cadaveric knees to investigate the ALatL. Some major findings from the study included that the ALatL femoral attachment was consistently located posterior and proximal to the attachment of the LCL and the lateral femoral epicondyle. The anterolateral tibial attachment was located approximately midway between the centre of the Gerdy's tubercle and the anterior margin of the fibular head.

A later study conducted by Kosy et al. [9] investigated the presence of the ALatL in 11 specimens to investigate its role within the knee structure. In this study they were able to identify the ALatL in 10 of the 11 specimens. The one specimen, which the ALatL was not able to be identified was also the only specimen which did not have the ACL intact, indicating the ALatL may have been damaged simultaneously in this example. Of the 10 specimens investigated, there was slight variation in the

femoral origin of the ALatL, with it found to be posterior and proximal to the LCL attachment in six knees, anterior and distal in three knees, and at the same site in one knee. The tibial attachment was more consistent in location, and was found to be a mean 17.7 (±2.95) mm from Gerdy's tubercle and 12.3 (±3.55) mm from the fibular head.

Helito et al. [10] stated that there is some controversy regarding the femoral attachment site of the ALatL around whether it is anterior and distal or posterior and proximal to the LCL attachment site. Despite this, the tibial attachment site is however consistently defined as being between the Gerdy's tubercle and the fibular head.

In conclusion, studies conducted by Claes et al. [4], Kennedy et al. [5] and Kosy et al. [9] found some discrepancy between the femoral origin of the ALatL and Helito et al. [10] stated that this was a common trait. Claes et al. [4] found the origin to be located anterior and distal to the origin of the LCL, compared to research conducted by Kennedy et al. [5] and Kosy et al. [9] who agreed that the most

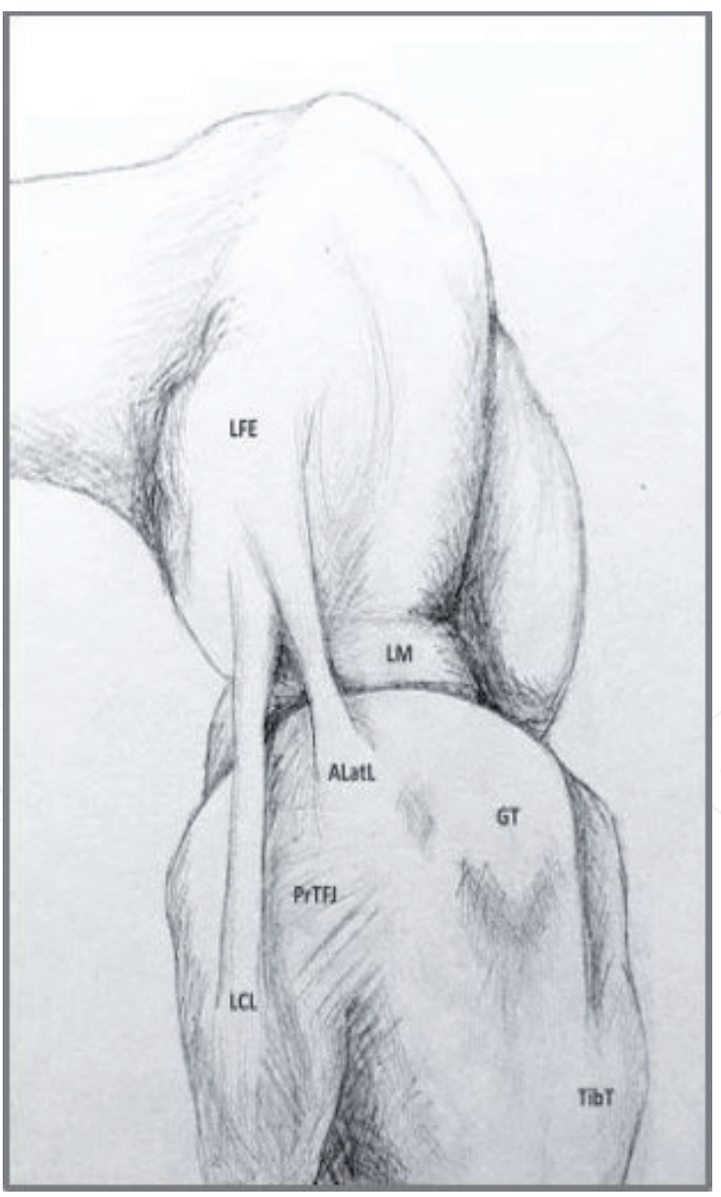

Figure 2.
Sketch of the anterolateral aspect of the knee showing the anterolateral ligament relative to other soft tissue structures. ALatL = anterolateral ligament; GT = Gerdy's tubercle; LCL = lateral collateral ligament; LFE = lateral femoral epicondyle; LM = lateral meniscus; PrTFJ = proximal tibiofibular joint; and TibT = tibial tubercle.

common origin site was posterior and proximal to the LCL attachment. Kosy et al. [9] did however define the origin, in three of the 10 specimens, to be anterior and distal to the ALatL, which agreed with the Claes et al. [4] findings. Hence, it must therefore be concluded that there is variance in the origin of the ALatL in different knee specimens. All four sources however agreed the anterolateral tibial insertion of the ALatL was roughly midway between Gerdy's tubercle and the fibular head [4, 5, 9, 10]. **Figure 2** shows an image that highlights the position of the ALatL on the lateral side of the knee.

2.3 Physical dimensions

Claes et al. [4] found that the measured mean length of the ALatL while in neutral rotation at a flexion angle of 90° was 41.5 (±6.7) mm and in extension was 38.5 (±6.1) mm. This therefore shows that there is some tension in the ALatL during flexion of the knee. Further testing found that the ALatL was mostly in tension while the knee was in a flexed position with an internal rotation applied. The mean width of the ALatLs' investigated by Claes et al. [4], was 6.7 (±3.0) mm and thickness 1.3 (±0.6) mm. Kosy et al. [9] found that of the 10 specimens that the ALatL was able to be located and for a knee flexion angle of 30° and neutral rotation, the mean dimensions were length 40.1 (± 5.53) mm, width 4.63 (±1.39) mm and thickness 0.87 (±0.18) mm. Hence, the values found by Claes et al. [4] and Kosy et al. [9] were similar while also taking into consideration the knee was positioned differently during measurement.

3. Mechanical properties

Zens et al. [11] conducted a biomechanical analysis of the ALatL to determine its mechanical properties allowing for a better understand of its role. Four specimens were investigated in the study. When a load was applied, it was found that all four specimens showed an inter-ligamentous failure at approximately one third of the ALatL's length, distal from the femoral insertion site. The mean ultimate load to failure was 49.90 (±14.62) N, ultimate tension of 32.78 N/mm^2 and the mean ultimate extension distance of 11.89 (±1.56) mm, hence, resulting in a mean extensional stiffness of 2.60 (±0.93) N.

Results from a study conducted in 2015 discovered that the average maximum load that the ALatL was able to handle during a pull-to-failure test was 175 N with a mean stiffness of 20 N/mm [5]. Failure of the ligament occurred through several mechanisms including ligamentous tear at the femoral or tibial origin, mid-substance tear and bony avulsion of the tibial attachment.

Helito et al. [10] also investigated the strength and stiffness of the ALatL of the knee through completing a biomechanical study. The methodology of testing involved 14 knee specimens of which the ALatL was tested for its tensile strength. Throughout testing the strength at the maximum resistance limit, deformation and stiffness were all measured. The mean maximum strength of the ALatL was found to be 204.8 (±114.9) N. The stiffness was 41.9 (±25.7) N/mm and the deformation of the ALatL was 10.3 (±3.5) mm [10]. **Table 1** shows a summary of the findings from the various studies discussed.

The mean maximum strength of the ALatL varied quite significantly between studies with Zens et al. [11] recording a much smaller strength value compared to other sources. Helito et al. [10] had a large variance in strength values from 89.9–319.7 N outlining how much it can vary between specimens. Zens et al. [11] however detailed that the ACL has been proven to have a higher ultimate tensile strength in

	Max. Strength (N)	Stiffness (N/mm)	Deformation (mm)	Ultimate Tension (N/mm^2)
Zens et al. [11]	49.90 (±14.62)	—	11.89 (±1.56)	32.78 N/mm^2
Kennedy et al. [5]	175	20	—	—
Helito et al. [10]	204.8 (±114.9)	41.9 (±25.7)	10.3 (±3.5)	—

Table 1.
Comparison between mechanical properties measured.

younger patients by a factor of 2.5 times. Considering that the previously presented loads of failure are based on specimens of mean age 86.5 ± 1.7 years, there is the possibility that the ALatL ultimate tension may be around 125 N in younger specimens if the same trend follows. The size of the ALatL is expected to reduce with age, hence the possibility for higher ultimate tension in younger specimens. This is more like the range of maximum strength found by other sources. The stiffness of the ligament was found to also differ between Kennedy et al. [5] and Helito et al. [10]. Zens et al. [11] and Helito et al. [10] agreed on the deformation of the ALatL to be around 10–12 mm. Hence, it can therefore be concluded from these studies that the ALatL has approximately the following mechanical properties: maximum strength of 50–200 N, stiffness of 20–42 N/mm, deformation of 30% of its length and tension of 33 N/mm^2. These values and ranges for the mechanical properties of the ALatL were also supported by Patel and Brophy [12].

Drews et al. [13] conducted an investigation to assess the function of the ALatL. This research tested the response of the ALatL through recording the ligaments activity while completing flexion of the knee and a number of rotations and translations. The testing involved eight specimens with the three conditions of: a normal knee with both ACL and ALatL intact, ACL resected (ACL_{res}) and ACL and ALatL resected ($ALatL_{res}$) to be compared. During the flexion testing under unloaded conditions there was no significant difference in internal rotation between the ACL_{res} and $ALatL_{res}$ specimens. With an internal tibial torque of 1–4 Nm applied, internal rotation significantly increased between 60 and 120° in the $ALatL_{res}$ specimen. Anterior tibial translation was also significantly higher at 30° in the $ALatL_{res}$ specimen. When unloaded there were no ALatL strains, however adding different internal tibial torques led to strain of the ALatL starting at an angle of 60° of flexion with a 1 Nm internal torque applied and 15° flexion with 4 Nm internal torque in the intact ligaments. It was also found that the ACL_{res} specimen also had significantly greater ALatL strains under low flexion angles than the intact specimen [13].

4. Analysis of the anterolateral ligament

4.1 Function

Due to the findings of these insertion and origin sites, Claes et al. [4] concluded that given its anatomical location at the anterolateral edge of the knee, it is hypothesised that the ALatL functions as a stabiliser for internal rotation. Dissections found the ALatL to become tense with forced internal rotation between 30 and 90° of knee flexion, as seen in **Figure 3**, however further kinematic analysis is needed to confirm this hypothesis formally. Therefore, research is needed to establish the function of the ALatL and to determine its role in clinical knee joint injuries [4].

Kosy et al. [9] concluded from their study that given the orientation of fibres in the ALatL and the tightening of this structure during internal rotation of the tibia as pictured in **Figure 4**, the structure plays a role in restraining this abnormal movement. Conclusions from the study of Drews et al. [13] were that the ALatL does not have a function under passive motion as there was no change in tibial rotation when the ALatL was removed from the specimen. When extrinsic loads are applied, the ALatL had a slight stabilising effect against anterior tibial shear at low flexion angles. Therefore, it is suggested that the ALatL is supporting the ACL

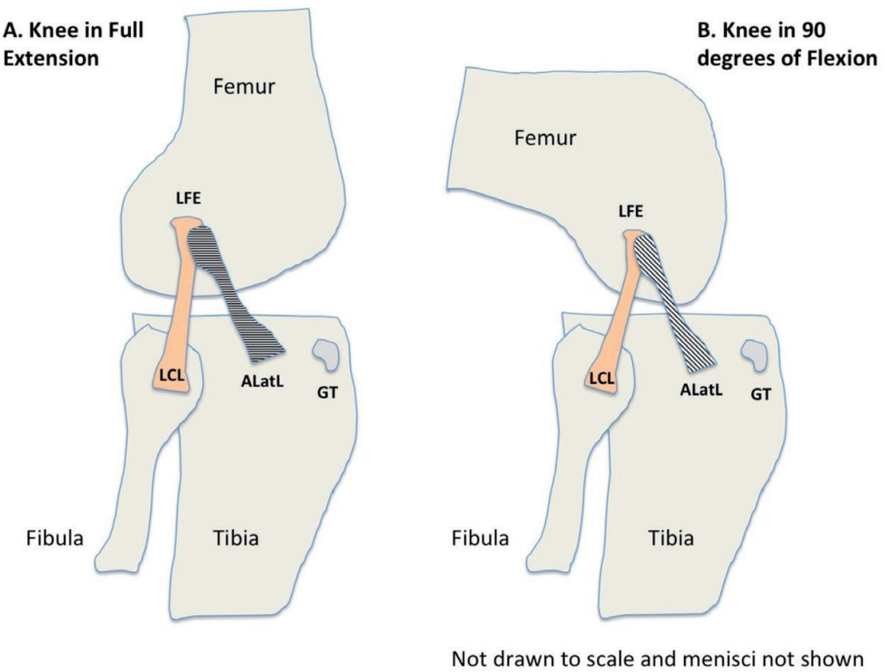

Figure 3.
Anatomical drawing of the anterolateral ligament on the lateral aspect of the human knee. (A) Knee in full extension. (B) Knee in 90° of flexion. ALatL = anterolateral ligament; GT = Gerdy's tubercle; LCL = lateral collateral ligament; and LFE = lateral femoral epicondyle.

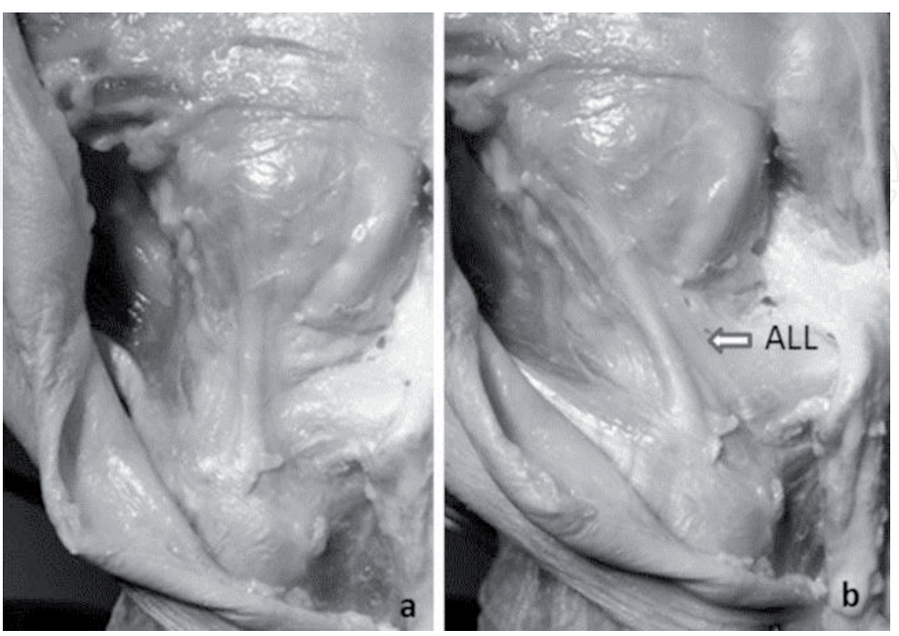

Figure 4.
Photographs demonstrating the tightening of the anterolateral ligament (labelled ALL) between a - a neutral position and b - with internal rotation of the tibia reprinted from, Kosy et al. [9]. Please see the following link for the creative commons licence: http://creativecommons.org/licenses/by/4.0/.

against internal tibial loads to a slight degree. Hence all three sources were in support that the ALatL plays a role in restricting internal tibial rotations [4, 9, 13].

4.2 Kinematics of the anterior cruciate (ACL) and anterolateral (ALatL) ligaments

4.2.1 The anterior cruciate ligament

The largest kinematic difference resulting from ACL deficiency is a substantial increase in anterior tibial translation on application of anterior tibial force [14], which has led to the clinical tests that employ anterior tibial force, for example the Lachman test, where pulling the leg forwards at the knee can result in an increase in translation in the horizontal plane [15].

Surgical replacement of the ACL, or reconstruction, reduces anterior tibial translation to (near-) normal levels, but in some knees there is a rotational instability remaining [16]. This residual instability is thought to lead to the accelerated osteoarthritis seen in the years following ACL rupture and reconstruction [17], and residual rotational instability, diagnosed through the pivot shift test, is associated with poorer outcomes following surgery [18].

In vivo studies of knee joint kinematics following ACL rupture and reconstruction can give a partial view into the ligament's function and its functional loss, but with the potential confounding influence of concurrent injuries to other structures. Further information can be drawn from in vitro laboratory experiments, where damage can be limited to the individual structures. Anterior tibial translation clearly is increased [19–22], but there is contention around rotation in the same plane. Internal tibial rotation as a response to internal tibial torque, defined to occur about a centrally-located axis [23], has been found to be unchanged [24, 25], or slightly increased [22, 26] following ACL ablation, but there has been doubt expressed as to whether this is sufficient to cause clinical problems [22].

The residual rotational instability that is sometimes seen following reconstruction is found during the pivot shift test [27], which involves coupling of internal rotation torque with abduction torque in the frontal plane. Application of these combined loads in the ACL deficient knee leads to normal [19] or slightly increased [28, 29] internal rotation but increased anterior translation [19, 30–32]. This finding has led some researchers to recommend that rotational instability be observed by increased translation [19].

4.2.2 The anterolateral ligament

Injury to the ALatL may cause residual instability in the knee after the ACL has been reconstructed. In a retrospective study of ACL reconstruction patients, those who were diagnosed as also suffering an ALatL tear, by MRI, showed a greater tendency to instability by the pivot shift test following reconstruction [33]. But the picture in relation to the pivot shift test, or in combined internal/valgus torque, is far from clear. What clarity studies of the ALatL provide will therefore be of great interest. Kennedy et al. [34] claimed that "residual rotatory laxity that may be seen clinically following ACL reconstruction may be attributable to an associated anterolateral structure injury". Considering this proposition when looking at ALatL-related kinematics will help to shed light on this issue.

Studies have mostly investigated the ALatL's contribution to kinematics in the ACL-deficient or -reconstructed knee, reflecting the ligament's role as a secondary stabiliser, i.e., having a lesser role than the ACL, and serving that function in the ACL-deficient knee. As knee flexion angle can affect the results of these

investigations, the findings of the following studies relate to results found at 30° of flexion.

Application of an 88 N anterior tibial force produced an additional increase in anterior translation after cutting the ALatL in the ACL-deficient knee [35]. This was supported by Tavlo and co-workers [36]. Knees with ACL reconstruction showed no change in anterior translation with an 88 N anterior force after cutting the ALatL, or after reconstructing the ALatL [37]. Thus the role of the ALatL in restraining anterior translation in the presence of either an intact or a reconstructed ACL appears not significant.

ALatL-deficient knees showed an increase in internal rotation on application of 5 Nm internal rotation torque to ACL-deficient knees [35]. Knees with ACL reconstruction showed an increase in internal rotation, compared with the intact state, on internal torque of 5 Nm, and this was increased on cutting the ALatL; rotation in turn decreased to intact levels with ALatL reconstruction [37]. Although Tavlo et al. found that ALatL deficiency led to greater internal rotation in ACL-deficient knees than with ACL deficiency alone, there was no such increase in internal rotation following ACL reconstruction (with the ALatL intact).

A 5 Nm internal rotation torque in combination with a 10 Nm valgus torque increased both anterior translation and internal rotation in ALatL- and ACL-deficient knees, when compared with only ACL-deficiency [35]. On application of a combined loading of 4 Nm internal rotation torque and 8 Nm of valgus torque, sectioning of the ACL increased anterior translation and internal rotation, and sectioning the ALatL increased translation and rotation further [38].

Nitri et al. [37] found that at 30 degrees, anterior translation on combined internal torque (5 Nm) and valgus torque (10 Nm) was not increased after ACL reconstruction, and this was not further changed with ALatL deficiency or reconstruction. In contrast, internal rotation was increased on combination loading in the ACL-reconstructed knee, further increased with ALatL deficiency, and reduced to intact levels following ALatL reconstruction. While loss of the ALatL amplifies the rotational instability of ACL deficiency, where rotational instability has remained after ACL reconstruction, loss/reconstruction of the ALatL increases/alleviates instability.

4.3 Grafts for the anterolateral ligament

Providing a surgeon was to undertake an ALatL reconstruction it would be important to determine the correct method of operation and in particular select the correct replacement graft. Kennedy et al. [5] suggests a graft of the gracilis tendon for an anatomical repair of the ALatL. Mechanical properties of several ligaments as measured by Zens et al. [11] are displayed in **Table 2**. All structures listed are able to provide a sufficient load to failure to replace the ALatL due to the low strength properties of the ligament, however the ultimate tension of the gracilis tendon matches the ALatL the least, with a much higher tension. Based on the data found through the study of Zens et al. [11] other possible graft options, such as the iliotibial band (ITB) or semitendinosis tendon would be a more suitable choice. Kennedy et al. [5] also investigated the best tissue structure to use as a replacement for the ALatL during reconstruction, identifying the semitendinosis tendon and gracilis tendon as appropriate graft selections. This selection was based on their mechanical strength properties and supported choices of both Kennedy et al. [5] and Zens et al. [11].

As well as choosing an appropriate graft for reconstruction of the ALatL, the development of a suitable surgical technique is also important. Kennedy et al. [5] suggested that reconstruction should follow similar techniques as used for other knee reconstructions with grafts used of similar length and properties.

Structure	Ultimate tension [MPa]	Ultimate load to failure [N]	Relation to ALatL (load to failure) [%]
ALatL	**32.78 ± 4.04**	**49.90 ± 14.62**	**100**
ACL	37.80 ± 3.80	1725 ± 269	3457
PCL	35.90 ± 15.20	739–1627	1481
MCL	38.60 ± 4.80	1107 ± 126	2219
Distal sMCL	—	557 ± 55	1116
Proximal sMCL	—	88 ± 36	176
POL	—	256 ± 30	513
Deep MCL	—	101 ± 10	202
LCL	—	309 ± 91	619
PFLC	—	186 N ± 65	373
ITB	19.1 ± 2.9	769 ± 99	1541
Fascia lata	78.7 ± 4.6	628 ± 35	1259
Semitendinosus	88.5 ± 5.0	1216 ± 50	2437
Gracilis	111.5 ± 4.0	838 ± 30	1679

ALatL = anterior lateral ligament; ACL = anterior crutiate ligament; PCL = posterior crutiate ligament; MCL = medial collataeral ligament; sMCL = (superficial) medial collateral ligament; POL = posterior oblique ligament; LCL = lateral collateral ligament; PFLC = popliteofibular ligament complex; ITB = iliotibial band.

Table 2.
Ultimate tension and ultimate load to failure of the ALatL in comparison to other ligaments and possible grafts as researched by Zens et al. [11].

4.4 Discussion

To summarise the results of the ALatL studies described, ALatL-deficiency makes the pattern of ACL deficiency (specifically anterior tibial translation and internal rotation) worse, as there is a greater range of movement with application of a load. In the ACL-reconstructed knee, ALatL-deficiency does not increase anterior tibial translation, but does increase internal rotation, an increase that is reversed after reconstructing the ALatL.

However, the results of ALatL studies cannot simply be superimposed on a background of solid agreement of ACL-related kinematics. While anterior tibial translation on the Lachman test is taken as read, not all researchers have found rotational laxity when applying internal rotation torque, and the pivot shift test may show rotational instability through an increase in translation.

The position and alignment of the ALatL make it logical that it would restrain internal rotation to a degree at least, but that degree aside, it is not always clear what a restraint of internal rotation might be useful for.

Of greater importance in this context is whether instability following ACL reconstruction can be attributed to ALatL injury. Nitri et al. [37] claimed that residual instability on the pivot shift test following ACL reconstruction "reaffirms the theory that tears of secondary restraints… should be properly recognized and treated". Although tears to secondary structures may be responsible for residual instability, it is clear from their results that residual instability results from ACL reconstruction in the presence of an intact ALatL, so at least part of the residual instability is likely to result from the simple fact that ACL reconstruction does not completely restore rotational stability to ACL intact levels. This supports the hypothesis that ACL reconstruction does not completely reproduce ACL-intact

kinematics, but given an ALatL lesion its reconstruction could be expected to reduce post-ACL reconstruction instability.

Such a conclusion though is theoretically more difficult to support under in vivo conditions. Less than complete restoration of normal stability appears to be typical following ACL reconstruction, and even with a reliable method of diagnosing ALatL injury, the confounding factor of imperfect ACL reconstruction would still cast uncertainty of the role of the ALatL and the clinical wisdom of reconstructing it.

Within these studies, ALatL reconstruction reverses the rotational deficits seen after ACL reconstruction when the ALatL is deficient. However, in the wider world of ACL research, where the pattern of rotational instability is controversial, and where translation may be a bigger problem, the benefit of recognising and repairing anterolateral ligament injuries is less clear.

5. Summary

The anterolateral ligament (ALatL) of the knee is an under investigated soft tissue structure with its existence within the body mostly unknown to the layperson. It was determined the ALatL has a femoral origin which is either anterior and distal, or posterior and proximal to the origin of the lateral collateral ligament, varying depending on the specimen being investigated. It is not clear exactly why there is such variation. The tibial attachment site was commonly found as midway between Gerdy's tubercle and the fibular head. These origin and attachment sites result in the ALatL running from the lateral femur to the anterior tibia with average physical properties of the ALatL; length of 40–42 mm, width 4–7 mm and thickness 0.8–1.3 mm. The ALatL was found to have mechanical properties on average of; maximum strength of 50–200 N, stiffness of 20–42 N/mm, deformation of 30% of its length and tension of 33 N/mm^2. Studies conducted found that the ALatL is in tension during internal rotations of the knee and therefore acts a stabiliser during internal tibial rotation. All studies presented the issue of lateral instability remaining after patients have undergone isolated ACL reconstruction and had the common belief that this is due to damage caused to lateral soft tissue structures on the knee.

Findings of many studies have not been conclusive enough to confidently suggest ALatL reconstruction as a viable option due to the lower maximum mechanical properties compared to other structures of the knee.

While there have been several studies recently conducted investigating the function of the ALatL of the knee through analysing a number of different factors including origin and insertion along with both physical and mechanical properties, there is still much mystery surrounding this ligament. Hence, further research should be conducted to accurately quantify the importance of the anterolateral ligament to internal tibial rotation stability, and the effect that a damaged ALatL can have on the stresses experienced by surrounding soft tissue structures of the knee.

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