THE RELATIONSHIP BETWEEN THE ANTEROLATERAL LIGAMENT AND THE LATERAL MENISCUS

Gillian Corbo
Western University, gcorbo@uwo.ca

Follow this and additional works at: https://ir.lib.uwo.ca/mcap
Part of the Anatomy Commons

Citation of this paper:
https://ir.lib.uwo.ca/mcap/13
THE RELATIONSHIP BETWEEN THE ANTEROLATERAL LIGAMENT AND THE LATERAL MENISCUS

Project format: Integrated Article

by

Gillian Corbo

Graduate Program in Clinical Anatomy

A project submitted in partial fulfillment of the requirements for the degree of Masters of Clinical Anatomy

The School of Graduate and Postdoctoral Studies
The University of Western Ontario
London, Ontario, Canada

© Gillian Corbo 2016
Abstract

The anterolateral ligament (ALL) has recently been of interest due to the belief that it plays a role in controlling anterolateral rotational laxity. However, the relationship of the ALL and its attachment to the lateral meniscus has yet to be addressed. Firstly this investigation determined the effect that sectioning the ALL and lateral meniscus posterior root (LMPR) in an ACL deficient knee has on internal rotation. Secondly this research determined if differences exist in the mechanical properties of the supra- and infra- meniscal fibers of the ALL. The ALL was found to control internal rotation at higher degrees of knee flexion, while the LMPR acted closer to extension and the infra-meniscal fibers were shown to be stronger and stiffer than the supra-meniscal fibers. These findings suggest that the ALL and LMPR work together to limit internal rotation, and there is a biomechanical difference within sections of the ALL.

Keywords

Anterolateral ligament, lateral meniscus posterior root, anterolateral rotational laxity, supra-meniscal fibers of ALL, infra-meniscal fibers of ALL, mechanical properties, biomechanics
Co-Authorship Statement

This thesis was completed by Gillian Corbo under the supervision of Dr. Alan Getgood and Dr. Timothy Burkhart. The written material in this book is the original work of the author unless otherwise stated. Gillian Corbo contributed to the following aspects of this work: data collection, data analysis, authorship of this thesis, and authorship and co-authorship of manuscripts.

Dr. Alan Getgood developed the original research question and provided supervision and guidance throughout the completion of my masters.

Dr. Timothy Burkhart played a key role in data collection, trouble-shooting during testing, and analysis and interpretation of data. He played an invaluable role in editing conference submissions, helping with government funding and providing guidance towards new ideas.

Dr. Tim Lording contributed his expertise as an orthopaedic surgeon to allow for data collection to be possible and co-authorship of one of the manuscripts.

Dr. Luke Spencer contributed some of his data to our study to allow comparisons to be drawn for a more meaningful conclusion.

Madeleine Norris contributed her time and energy in helping preparing specimens and assisted in data collection.
Acknowledgments

Dr. Marjorie Johnson for her continued support throughout the entirety of this project and her willingness to problem-solve and provide invaluable mentorship whenever she could.

Dr. Alan Getgood for the development of this research project and for providing his vital clinical insight.

Dr. Timothy Burkhart for being one of the most supportive supervisors anyone could ask for. Thank you for always being so willing to help me problem-solve, provide some of the most detailed edits to my writing, and show me what it’s like to be a researcher. I could not have completed this master’s degree without you.

Dr. Martin Sandig for his unwavering support and belief in my abilities as a researcher, and his assistance in histological analysis.

Madeleine Norris for being such a dedicated research partner and an incredible support system throughout this degree. Her support in preparing specimens was invaluable, and she brought sunshine into the morgue, and laughter to hours of data analysis and writing.

Haley Linklater and Kevin Walker for their help in ensuring I always had cadaveric specimens, for their patience while I fixed problems with my research, and their countless hours helping me prepare my specimens for testing and cremation.

Tyler Beveridge for his continued support within the department. He was always the first person to offer his time to help or answer questions, despite being one of the busiest people.

The donors and their families for their selfless gift that they have bestowed upon us. This research would not have been possible without them.

My Clinical Anatomy colleagues for providing lasting memories, support, laughter and friendship. You have all become family and I will miss the hours spent in the office.

My loving and supportive family and friends for pushing me to do things I never thought were possible.
# Table of Contents

Abstract ......................................................................................................................... ii  
Co-Authorship Statement ............................................................................................... iii  
Acknowledgments ........................................................................................................ iv  
Table of Contents .......................................................................................................... v  
List of Tables .................................................................................................................. ix  
List of Figures ............................................................................................................... x  
List of Appendices ........................................................................................................ xii  
List of Abbreviations ..................................................................................................... xiii  
Chapter 1 ...................................................................................................................... 1  
  1 General Introduction ................................................................................................. 1  
    1.1 Epidemiology of Knee Injuries ........................................................................... 1  
    1.2 The Knee Joint .................................................................................................... 1  
    1.3 Anatomy of the Anterolateral Ligament .............................................................. 3  
      1.3.1 Origin and Insertion ...................................................................................... 3  
      1.3.2 Attachment to the Lateral Meniscus .............................................................. 4  
      1.3.3 The Anterolateral Ligament’s Fibers and Relationships to Other Structures .................................................................................................................. 5  
      1.3.4 Histology of the Anterolateral Ligament ....................................................... 6  
      1.3.5 Morphological Dimensions of the Anterolateral Ligament ......................... 7  
      1.3.6 Prevalence of the Anterolateral Ligament .................................................... 7  
    1.4 Anatomy of the Lateral Meniscus ......................................................................... 8  
      1.4.1 Attachments of the Lateral Meniscus ............................................................ 8  
      1.4.2 Relationships to Other Structures ................................................................. 9  
    1.5 Function of the Anterolateral Ligament .............................................................. 9
<table>
<thead>
<tr>
<th>Section</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.5.1</td>
<td>Role in Internal Rotation vs. Anterior Translation</td>
<td>9</td>
</tr>
<tr>
<td>1.5.2</td>
<td>Pivot Shift</td>
<td>11</td>
</tr>
<tr>
<td>1.6</td>
<td>Function of the Lateral Meniscus</td>
<td>12</td>
</tr>
<tr>
<td>1.6.1</td>
<td>Normal Function</td>
<td>12</td>
</tr>
<tr>
<td>1.6.2</td>
<td>Function When Damaged</td>
<td>13</td>
</tr>
<tr>
<td>1.6.3</td>
<td>Functional Relationship with the Anterior Cruciate Ligament</td>
<td>13</td>
</tr>
<tr>
<td>1.7</td>
<td>Injury Patterns</td>
<td>14</td>
</tr>
<tr>
<td>1.7.1</td>
<td>Anterolateral Ligament and Anterior Cruciate Ligament</td>
<td>14</td>
</tr>
<tr>
<td>1.7.2</td>
<td>Lateral Meniscus and Anterior Cruciate Ligament</td>
<td>14</td>
</tr>
<tr>
<td>1.7.3</td>
<td>Anterolateral Ligament and Lateral Meniscus</td>
<td>15</td>
</tr>
<tr>
<td>1.7.4</td>
<td>Anterolateral Ligament, Lateral Meniscus and Anterior Cruciate Ligament</td>
<td>15</td>
</tr>
<tr>
<td>1.7.5</td>
<td>Segond Fracture</td>
<td>16</td>
</tr>
<tr>
<td>1.8</td>
<td>Biomechanics of the Anterolateral Ligament</td>
<td>18</td>
</tr>
<tr>
<td>1.8.1</td>
<td>Mechanical Properties</td>
<td>18</td>
</tr>
<tr>
<td>1.8.2</td>
<td>Translation and Rotation</td>
<td>19</td>
</tr>
<tr>
<td>1.8.3</td>
<td>Contribution at Different Angles</td>
<td>20</td>
</tr>
<tr>
<td>1.9</td>
<td>Biomechanics of the Lateral Meniscus</td>
<td>21</td>
</tr>
<tr>
<td>1.9.1</td>
<td>Mechanical Properties</td>
<td>21</td>
</tr>
<tr>
<td>1.10</td>
<td>Clinical Pathology</td>
<td>22</td>
</tr>
<tr>
<td>1.10.1</td>
<td>The Anterolateral Ligament</td>
<td>22</td>
</tr>
<tr>
<td>1.10.2</td>
<td>The Lateral Meniscus</td>
<td>23</td>
</tr>
<tr>
<td>1.11</td>
<td>Study Purpose</td>
<td>23</td>
</tr>
<tr>
<td>1.12</td>
<td>Study Objectives</td>
<td>24</td>
</tr>
<tr>
<td>1.13</td>
<td>Hypothesis</td>
<td>24</td>
</tr>
<tr>
<td>1.14</td>
<td>References</td>
<td>25</td>
</tr>
</tbody>
</table>
Chapter 2 .................................................................................................................................................... 36

2 Histological Analysis of the Supra- and Infra-meniscal Sections of the Anterolateral Ligament .......................................................... 36

2.1 Introduction .............................................................................................................................................. 36

2.2 Materials and Methods ...................................................................................................................... 37

2.3 Results .................................................................................................................................................. 38

2.4 Discussion ............................................................................................................................................ 40

2.5 References .......................................................................................................................................... 41

Chapter 3 .................................................................................................................................................... 42

3 The Contribution of the Anterolateral Ligament and the Lateral Meniscus to the Control of Rotational Knee Laxity ............................................... 42

3.1 Introduction ........................................................................................................................................ 42

3.2 Materials and Methods ..................................................................................................................... 43

3.3 Results ............................................................................................................................................... 47

3.4 Discussion ........................................................................................................................................ 50

3.5 Conclusion ....................................................................................................................................... 54

3.6 References ...................................................................................................................................... 55

Chapter 4 .................................................................................................................................................... 63

4 Tensile Properties of the Supra- and Infra-meniscal Fibers of the Anterolateral Ligament .................................................................................. 63

4.1 Introduction ....................................................................................................................................... 63

4.2 Materials and Methods .................................................................................................................... 64

4.3 Results ................................................................................................................................................ 69

4.4 Discussion ....................................................................................................................................... 71

4.5 Conclusion ...................................................................................................................................... 74

4.6 References ..................................................................................................................................... 75

5 Overall Discussion and Conclusions ........................................................................................................ 79
5.1 Overall Discussion .................................................................................................................. 79
5.2 Future Directions .................................................................................................................. 81
5.3 Conclusions .......................................................................................................................... 82
5.4 References ............................................................................................................................ 83
Appendices ................................................................................................................................ 86
Curriculum Vitae ....................................................................................................................... 94
List of Tables

Table 1 Clinical grading of the pivot shift test. (Jakob, Stäubli, & Deland, 1987). ............ 11

Table 2 Grading scale used for ACL tears when viewed on MRI. (De Maeseneer et al., 2014). ................................................................................................................................................. 18

Table 3 Comparison of the mean (SD) internal rotation (°) between the different tissue conditions, across all knee flexion angles................................................................. 48

Table 4 Comparison of the mean (SD) peak force and stiffness values for the supra- & infra-meniscal construct (*p<0.05). .................................................................................................................. 69

Table 5 Comparison of the mean (SD) width, thickness and cross sectional area of the supra- & infra-meniscal sections of the ALL. .................................................................................................................. 71

Table 6 Comparison of the mean (SD) peak strain of the supra- & infra-meniscal sections experienced by different parts of the ALL-meniscus complex......................................................... 71
List of Figures

**Figure 1** Anatomical drawing of the ALL. ALL, anterolateral ligament; LCL, lateral collateral ligament; GT, Gerdy’s tubercle; LFE, lateral femoral epicondyle; PT, popliteal tendon; PFL, popliteo-fibular ligament. **a)** Knee in extension; **b)** Knee in flexion (*Claes et al., 2013*). .......................................................................................................................... 3

**Figure 2** Intra-articular axial view of the ALL and the popliteal tendon attaching to it ....... 6

**Figure 3** Superior view of an anatomical drawing showing the lateral meniscus (LM), anterior root (long white arrow), posterior root (short white arrow) ACL (asterisk), PCL (P) meniscofemoral ligament (black arrowhead) and transverse genual ligament (white arrowhead). (*Brody et al., 2007*) .......................................................................................................................... 8

**Figure 4** X-ray of a Segond fracture. The white arrow indicates the tibia avulsing. *(Radiopedia.org, Creative Commons Use)* .................................................... 17

**Figure 5** Image of the lateral inferior geniculate artery (LIGA) traveling between the lateral meniscus (LM) and anterolateral ligament (ALL) with the meniscofemoral portion of the ALL (*) displayed (*Claes et al., 2013*). .......................................................................................................................... 38

**Figure 6 a)** LIGA shown in longitudinal section. **b)** LIGA and supra-meniscal fibers shown. .......................................................................................................................... 39

**Figure 7** Fibers of the ALL shown in longitudinal section. **a)** Supra-meniscal fibers of the ALL. **b)** Infra-meniscal fibers of the ALL .......................................................................................................................... 39

**Figure 8** Experimental setup of the knee within the joint simulator including the position of the optical markers. Also shown is the orientation of the anatomical coordinate system. ...... 45

**Figure 9** Flowchart indicating the sectioning protocol of the knee specimens for biomechanical testing. n=16 unless otherwise indicated. .......................................................................................................................... 46

**Figure 10** Degree of internal rotation at each sectioning condition at **a)** 0°; **b)** 15°; **c)** 30°; **d)** 45°; **e)** 60°; **f)** 75°; **g)** 90° of flexion. .......................................................................................................................... 49
Figure 11 Sample showing how the bones for the supra-meniscal group were prepared, leaving only the lateral femoral condyle and the tibial plateau. .................................................. 66

Figure 12 Sample showing how the bones for the infra-meniscal group were prepared, leaving only the tibial plateau with a lateral section removed. .................................................. 66

Figure 13 Experimental set-up displaying how the ALL was pulled with reference to the meniscus. .......................................................................................................................... 67

Figure 14 Comparison of the mean (SD) supra- and infra-meniscal failure forces (left axis) and stiffness’s (right axis) (*p<0.05). .......................................................................................................................... 69

Figure 15 Failure mechanisms of the supra-meniscal ALL construct. a) Mid-ligamentous tear; b) Ligamentous tear at the femoral origin; c) Failure near the meniscal insertion. .... 70

Figure 16 Failure mechanisms of the infra-meniscal ALL construct. a) Mid-ligamentous tear; b) Avulsion of the bony tibial attachment (i.e., Segond fracture). .............................. 70
List of Appendices

Appendix A: Authorization for publication of the work to Scholarship@Western ............ 86

Appendix B: Copyright permission for use of material provided by John Wiley and Sons. 87

Appendix C: Copyright permission for use of material provided by American Roentgen Ray Society .......................................................... 93
List of Abbreviations

ACL – Anterior Cruciate Ligament

ALL – Anterolateral Ligament

ITB – Iliotibial Band

LCL – Lateral Collateral Ligament

LET – Lateral Extra-Articular Tenodesis

LIGA – Lateral Inferior Geniculate Artery

LM – Lateral Meniscus

LMPR – Lateral Meniscus Posterior Root

MCL – Medial Collateral Ligament

MRI – Magnetic Resonance Imaging

PCL – Posterior Cruciate Ligament
Chapter 1

1 General Introduction

1.1 Epidemiology of Knee Injuries

The knee is a very complex structure, and therefore injuries are common and often debilitating. Traumatic injuries of the knee, such as anterior cruciate ligament (ACL) ruptures and meniscal tears, result in approximately 50,000 surgeries annually in Canada (McRae, Chahal, Leiter, Marx, & Macdonald, 2011) at a cost of approximately $300 million (CAD) per annum (Canadian Institute for Health Information, 2014; McRae et al., 2011). These injuries also commonly lead to chronic disabilities, such as post-traumatic osteoarthritis (50% of patients) (Racine & Aaron, 2014) or persistent post-operative problems such as anterolateral laxity (32% of patients) (Kocher et al., 2004; Sonnery-Cottet et al., 2015). Given the burden these injuries place on the economy and health care system, and the effect they have on the quality of life for patients, the research described in this thesis explores areas that will contribute to minimizing this problem.

Despite advances in intra-articular ACL reconstruction, some patients are still left with a knee that is unable to control normal rotatory limits, so recently, surgeons have been performing a combined intra-articular and extra-articular ACL reconstruction when anterolateral laxity is present to help fix this problem (Sonnery-Cottet et al., 2015). The extra-articular part of the procedure involves reconstructing or reinforcing the anterolateral capsule or ligament of the knee to help restore normal rotatory limits (Sonnery-Cottet et al., 2015).

1.2 The Knee Joint

The knee joint is a large synovial hinge joint that forms a weight bearing articulation between the femur and tibia (Drake, Vogl, & Mitchell, 2010). While the fibula does not contribute to the knee joint, it sits in close proximity to it, and functions as an attachment point for important stabilizers of the knee. The patella also sits in close proximity to the femur to form the patellofemoral joint which contributes to the formation of the knee
joint (Drake et al., 2010). The movements of the knee joint are complex, but as a hinge joint it primarily permits flexion and extension, as well as some internal and external rotation and a small amount of varus and valgus movement (Drake et al., 2010). Within the knee joint sit two fibrocartilaginous menisci that move and accommodate the articular surfaces of the femur and tibia while being anchored centrally to the tibial plateau (Drake et al., 2010). The knee is stabilized by a complex interaction of dynamic and static stabilizers. Muscles of the thigh provide dynamic stability, primarily the extensors (quadriceps) and the flexors (hamstrings) (Drake et al., 2010). The knee is passively stabilized by the shape of the articular surfaces of the femur and the tibia and by two extra-articular collateral ligaments on the side of the knee, and two strong intra-articular cruciate ligaments (Drake et al., 2010). The two collateral ligaments are the lateral collateral ligament (LCL) that runs from the lateral femoral epicondyle to the head of the fibula and the medial collateral ligament (MCL) that originates on the medial femoral epicondyle and inserts onto the medial tibial condyle. These structures are responsible for preventing varus and valgus movements of the knee respectively (Drake et al., 2010). The two cruciate ligaments are the anterior cruciate ligament (ACL) that runs posterolaterally from the anterior tibia to the lateral femoral condyle and the posterior cruciate ligament (PCL) that runs anteromedially from the posterior tibia to the medial femoral condyle, and are involved in preventing anterior and posterior displacement of the tibia relative to the femur (Drake et al., 2010). The articular cavity is enclosed by a fibrous membrane known as the joint capsule, and additional reinforcement of the anterolateral capsule is provided in part by the Iliotibial Band (ITB), a lateral thickening of the deep fascia of the leg that inserts on the tibia (Drake et al., 2010).

Recently, a structure known as the anterolateral ligament (ALL) has been described as a ligament capable of supporting the anterolateral joint capsule (Claes et al., 2013; Vincent et al., 2012). This structure was previously described by Segond (1879) who noted a resistant fibrous band of tissue that was implicated in avulsion fractures of the anterolateral tibia (Segond, 1879). Over the last 130 years, many terms have been used to describe the ligament such as: mid-third lateral capsular ligament (Hughston, Andrews, Cross, & Moschi, 1976), lateral capsular ligament (Dietz, Wilcox, & Montgomery, 1986; Johnson, 1979; Patella, Bernardi, Moretti, Pesce, & Simone, 2002), anterior slip of lateral
collateral ligament (Fulkerson & Gossling, 1980), Capsulo-osseous layer of the IT band (Terry, Norwood, Hughston, & Caldwell, 1993), anterior oblique band (Campos et al., 2001; Irvine, Dias, & Finlay, 1987), and most recently, the term anterolateral ligament introduced by Vieira et al., (2007). This specific structure is of particular interest given the important role it is thought to play in controlling knee stability and will be discussed in great depth throughout this literature review.

1.3 Anatomy of the Anterolateral Ligament

1.3.1 Origin and Insertion

The ALL has been defined as a ligament distinct from the joint capsule that originates from the lateral femoral epicondyle and runs anteroinferiorly to insert on the lateral aspect of the tibia, midway between Gerdy’s tubercle and the fibular head approximately 5-11mm below the tibial plateau (Figure 1) (Caterine, Litchfield, Johnson, Chronik, & Getgood, 2014; Claes et al., 2013; Dodds, Halewood, Gupte, Williams, & Amis, 2014; Helito et al., 2013). However, specificity and consistency of the origin on the femur with

![Figure 1 Anatomical drawing of the ALL. ALL, anterolateral ligament; LCL, lateral collateral ligament; GT, Gerdy’s tubercle; LFE, lateral femoral epicondyle; PT, popliteal tendon; PFL, popliteo-fibular ligament. a) Knee in extension; b) Knee in flexion (Claes et al., 2013).](image-url)
respect to the lateral collateral ligament has been described with some variability. For example, some research has indicated that the ALL originates posterior and proximal to the origin of the LCL (Caterine et al., 2014; M. I. Kennedy et al., 2015; Kosy, Soni, Venkatesh, & Mandalia, 2016; Runer et al., 2015), while others have described it as originating anterior and distal to the origin of the LCL (Claes et al., 2013; Pomajzl, Maerz, Shams, Guettler, & Bicos, 2014; Vincent et al., 2012). In addition, others have described the ALL as originating strictly anterior to the origin of the LCL (Tavlo, Eljaja, Jensen, Siersma, & Krogsgaard, 2015), or a split between posterior-proximal and anterior-distal to the origin of the LCL (Bonasia, D’Amelio, Pellegrino, Rosso, & Rossi, 2015; Kosy et al., 2016; Rezansoff et al., 2014). The trend in the literature seems to be that older studies describe the ALL origin as being anterior and distal to the LCL while more recent studies are more likely to describe the ALL origin as originating posterior and proximal to the LCL.

Conversely, the insertion of the ALL onto the tibia is much more consistent with a general agreement that it inserts between Gerdy’s tubercle and the fibular head (Campos et al., 2001; Caterine et al., 2014; Cianca, John, Pandit, & Chiou-Tan, 2014; Claes et al., 2013; Dodds et al., 2014; Helito et al., 2013; Helito, Demange, et al., 2014; Patella et al., 2002; Rezansoff et al., 2014; Van der Watt et al., 2015; Vieira et al., 2007; Vincent et al., 2012), with the insertion point sitting closer to the fibular head than Gerdy’s tubercle (Kosy et al., 2016).

### 1.3.2 Attachment to the Lateral Meniscus

In early descriptions of the ALL, its attachment points were discussed as being only the femur and the tibia. Recent evidence however, suggests that the ALL attaches directly to the lateral meniscus (Caterine et al., 2014; Claes et al., 2013; Helito et al., 2013, 2016; M. I. Kennedy et al., 2015; Kosy et al., 2016), with only two papers in opposition to that idea (Dodds et al., 2014; Runer et al., 2015). Of the two papers that oppose the idea of a meniscal attachment, Runer et al., (2015) suggests the meniscal attachment comes from the joint capsule, however embalmed specimens were used as opposed to fresh-frozen which may have altered the appearance of the tissue, and Dodds et al., (2014) concedes that there are branching attachments from fibers deep to the ALL that run to the lateral
meniscus, likely describing the bifurcation of the ALL. An anatomical attachment seems likely since when the lateral meniscus is pulled in a certain direction, the ALL responds by moving in the same direction (Caterine et al., 2014). By attaching to the meniscus, the ALL has two attachment sites: one on the lateral meniscus and one on the tibia (Helito et al., 2013; Van Dyck et al., 2016). This attachment to the meniscus results in a division of the ALL into two parts: a meniscofemoral / supra-meniscal segment, and a meniscotibial / infra-meniscal segment (Claes et al., 2013). The anatomical specifications of the meniscal attachment have recently been examined with the ALL insertion shown to occur between the anterior horn and body of the meniscus, at approximately 36-42% of the meniscus’ circumference (Helito et al., 2016). Investigation with magnetic resonance imaging (MRI) reveals that the meniscal attachment has been organized into four subgroups: complete (the ALL attaches to the full thickness of the lateral meniscus), central (the superior and inferior edges of the lateral meniscus were bare), bipolar (only the superior and inferior edges had an attachment), and inferior-only (the inferior edge of the lateral meniscus was the only place an attachment occurred) (Kosy, Mandalia, & Anaspure, 2015).

1.3.3 The Anterolateral Ligament’s Fibers and Relationships to Other Structures

With the renewed interest in the ALL, there has been considerable debate regarding the true nature of the ALL. Over the past few years the ALL has been identified as a distinct ligamentous structure in 18 of 19 studies (Van der Watt et al., 2015) while also being likened to a thickening of the joint capsule (Caterine et al., 2014; Kosy et al., 2016) but still being discernible from the anterolateral joint capsule (Claes et al., 2013; Dodds et al., 2014; Zens, Feucht, et al., 2015). The proximal fibers of the ALL origin fan out and integrate with the LCL’s proximal attachment (Caterine et al., 2014; Claes et al., 2013; Dodds et al., 2014; Parsons et al., 2015), and the medial fibers of the ALL are also in close relation to the fibers of the popliteal tendon (Figure 2) (Vincent et al., 2012).
Histological analysis of the ALL has shown that the collagen is organized dense connective tissue, which is characteristic of ligamentous structures (Caterine et al., 2014; Guenther et al., 2015). The ALL is composed primarily of Type I collagen that is responsible for providing strength and resistance to loading (Macchi et al., 2015; Ross & Pawlina, 2006). Furthermore, minor amounts of Type III (that forms reticular, supportive fibers) and Type VI collagen (that contributes to the formation of the cartilage matrix) are also found in the ALL (Macchi et al., 2015; Ross & Pawlina, 2006). The pattern of collagen arrangement in the ALL is consistent with other ligaments, and immunohistochemistry has identified a prominent network of peripheral nerves, indicating a possible role in proprioception (Caterine et al., 2014; Vincent et al., 2012). The ALL is clearly distinguishable from the lateral joint capsule, as the joint capsule resembles loose connective tissue (Caterine et al., 2014). The ALL’s bony insertion is distinct, and the femoral insertion of the ALL displays a transition from ligament, to mineralized cartilage, to bone that is characteristic of ligamentous tissue (Caterine et al.,

**Figure 2** Intra-articular axial view of the ALL and the popliteal tendon attaching to it.
Finally, histological analysis of sections of the ALL and lateral meniscus confirm that the ALL adheres to the lateral meniscus without a cleavage plane, and some fibers do in fact insert into the meniscus (Vincent et al., 2012).

1.3.5 Morphological Dimensions of the Anterolateral Ligament

A recent systematic review of the anatomy of the ALL suggests that the ALL has a relatively consistent length with a range of 34.1-45mm (Kosy et al., 2016; Van der Watt et al., 2015). The width of the ALL superior to the meniscus ranges from 5.1-8.3mm, while the width inferior to the meniscus ranges from 8.9-12.2mm (Macchi et al., 2015; Van der Watt et al., 2015). Although thickness values have been recorded, there has been a lack of identification regarding where those measurements were taken along the ALL’s length. This has resulted in variable values ranging between 0.87-2.7mm (Claes et al., 2013; Dodds et al., 2014; Helito et al., 2013, 2015; Kosy et al., 2016; Vieira et al., 2007; Vincent et al., 2012).

1.3.6 Prevalence of the Anterolateral Ligament

To date, there is no consensus regarding the prevalence of the ALL due to some of the discrepancies in the classification of the ALL’s origin and how the ALL is defined as a structure. In a systematic review of anatomical dissection papers, the prevalence of the ALL was found to be 96% (Van der Watt et al., 2015), with some anatomical dissection papers reaching a prevalence of 100% (Caterine et al., 2014; Helito et al., 2013; M. I. Kennedy et al., 2015). However, some biomechanical papers have stated a prevalence of 78% (the authors acknowledged finding an ALL in 100% of cadavers, however it was determined to have a highly variable appearance and only be well-defined in 78% of knees), while others have reported it as low as 43% (Saiegh et al., 2015; Tavlo et al., 2015). MRI studies have not provided additional consistency, with one study identifying an ALL in 94% of MRIs, all presenting with a meniscal insertion (Kosy et al., 2015), while a different MRI study identified an ALL in only 51% of MRIs and no meniscal insertions were identified (Taneja et al., 2014), despite both studies using 1.5T scanners and 3mm slice thickness. One explanation for the discrepancy in prevalence is that some specimens (or patients in the MRI studies) with a lower prevalence percentage may have
had a previous knee injury that made the ALL difficult to positively identify (Kosy et al., 2015). This was supported by an anatomical follow up study, which identified the ALL in 91% of specimens, and the only specimen it was not found in was a knee that did not have an intact ACL, indicating that the ALL was likely damaged along with the ACL (Kosy et al., 2016)

1.4 Anatomy of the Lateral Meniscus

1.4.1 Attachments of the Lateral Meniscus

The meniscus is composed of five distinct parts: a body, an anterior horn, a posterior horn, an anterior root and a posterior root. The body makes up over half of the fibrocartilaginous disc that tapers into the anterior and posterior horns. From the horns, the respective roots extend out to anchor the meniscus to the tibial plateau (Drake et al., 2010). The anterior root of the lateral meniscus inserts on the anterior intercondylar crest of the tibia, and sits lateral to the ACL, with which its fibers partially blend (Brody, Hulstyn, Fleming, & Tung, 2007). The lateral meniscus posterior root (LMPR) inserts on the posterior or horizontal part of the intercondylar area of the tibia, sitting just posterior to the posterolateral bundle of the ACL (Figure 3) (Brody et al., 2007; Koenig, Ranawat,

![Figure 3](image.png)

**Figure 3** Superior view of an anatomical drawing showing the lateral meniscus (LM), anterior root (long white arrow), posterior root (short white arrow) ACL (asterisk), PCL (P) meniscofemoral ligament (black arrowhead) and transverse genual ligament (white arrowhead). (*Brody et al.*, 2007).
Umans, & Difelice, 2009). However, recent research reveals that the insertion of the LMPR may be somewhat variable, and an MRI investigation revealed that 76% of knees presented with two LMPR attachment sites, with the major attachment inserting posteromedial to the ACL attachment, and the minor attachment inserting on the posterior slope of the lateral tibial tubercle (You, Park, Park, Jin, & Ryu, 2014).

1.4.2 Relationships to Other Structures

The lateral meniscus is in close proximity to the popliteal tendon and the meniscofemoral ligament which attach to the lateral meniscus (Brody et al., 2007) and are difficult to resect from their attachments in some cases. The border of the lateral meniscus attaches to the fibrous joint capsule except at the popliteal tendon hiatus (Brody et al., 2007). This capsular attachment is much less firm compared to the capsular attachment of the medial meniscus, and the attachments from the lateral meniscus to the tibial plateau are also much weaker on the lateral side than the medial side (Musahl et al., 2010). This suggests that the lateral meniscus is not as securely anchored as the medial meniscus, therefore it cannot produce as great of a ‘wedge-effect’ to help resist anterior tibial translation (Musahl et al., 2010)

1.5 Function of the Anterolateral Ligament

1.5.1 Role in Internal Rotation vs. Anterior Translation

The anterolateral structures of the knee play a crucial role in restraining internal rotation and anterior translation, and it comes down to a complex interaction between the ALL, Iliotibial Band (ITB) and anterolateral capsule that all work together (Sonnery-Cottet et al., 2015; Zens, Feucht, et al., 2015). Although the ALL has been recognized as a ligamentous structure (Caterine et al., 2014; Patella et al., 2002; Van der Watt et al., 2015; Vieira et al., 2007), the functional role of the ALL is not fully understood. Early research hypothesized that the ALL played a role in stabilizing the knee (i.e. limiting excessive movement or laxity) in internal rotation (Claes et al., 2013), and anterior tibial translation (Vincent et al., 2012) but with few specifics regarding the extent of its contribution. More recently, it has been hypothesized that the ALL acts as a secondary stabilizer of the ACL to help limit anterolateral rotation of the tibia with respect to the
femur (Rezansoff et al., 2014), thereby reducing rotational laxity (Dodds et al., 2014). However, the resistance to internal rotation provided by the ALL was not found to be consistent throughout knee flexion and was most significant between 30° and 90° of flexion (Claes et al., 2013). While the ALL has been suggested to limit rotation of the knee, it is unlikely to be a primary stabilizer as the tensile strength of the ALL is lower than would be expected with a primary stabilizer (Zens, Feucht, et al., 2015). However, due to the role that it is suggested to play in contributing to rotational stability, irrespective of it being a primary or secondary stabilizer, persistent knee laxity after ACL reconstruction may partially be attributed to unrepaired damage to the ALL (Monaco et al., 2012; Sonnery-Cottet et al., 2016).

There are no definitive guidelines with respect to what movements the ALL acts to restrain, but the general trend of the literature indicates that it plays a larger role in controlling internal rotation than anterior translation. Parsons et al., (2015) carried out a serial sectioning study on the ALL and ACL, but they randomized the order of sectioning, and calculated the percentage force contribution that each ligament provided by dividing the magnitude of the force vector by the magnitude of the intact ligament’s force vector (Parsons et al., 2015). With respect to internal rotation, the ALL has been shown to be an important stabilizer at flexion angles greater than 35°, whereas the ACL plays a greater role in controlling internal rotation at flexion angles less than 35° (Parsons et al., 2015). In other words, there is an inverse relationship between flexion angle and the role the ACL plays in controlling internal rotation. With respect to anterior translation, ALL was approaching 0% contribution at all flexion angles, whereas the ACL was the primary stabilizer for anterior translation at all flexion angles (Parsons et al., 2015). This idea is further supported by the ACL’s decreasing contribution to rotational stability with increasing knee flexion angles, while the ALL has an increasing contribution with increasing knee flexion angles (Tavlo et al., 2015). This suggests that an ALL injury may result in rotational instability, but only at higher knee flexion angles (Parsons et al., 2015).
1.5.2 Pivot Shift

The pivot shift phenomenon occurs as a result of ACL injury and results in increased anterolateral rotatory laxity, with the central pivot of the knee being moved medially as a result of ACL deficiency (Dodds et al., 2014; Galway & Macintosh, 1980). It is classified as anterior subluxation of the lateral tibial plateau underneath the lateral condyle of the femur as the knee approaches extension (Galway & Macintosh, 1980; Monaco et al., 2012). The clinical pivot shift test is better able to correlate with functional instability and the outcomes of a patient compared to other clinical tests, such as the Lachman test, because the pivot shift replicates a combination of rotational and translational laxity (Monaco et al., 2012). Traditionally it was assumed that this unique form of laxity occurred as a result of isolated ACL damage (Galway & Macintosh, 1980); however, recent evidence would suggest that the anterolateral capsule/ALL may have a role in contributing to this phenomenon. It is well established that following an ACL reconstruction, a high percentage of patients (up to 25%) can present with a positive pivot shift sign (Sonnery-Cottet et al., 2015; Spencer et al., 2015). Since it was hypothesized that the ALL played a role in maintaining anterolateral stability, a number of researchers hypothesized that it may also be responsible for preventing the pivot shift (Monaco et al., 2012; Sonnery-Cottet et al., 2015; Spencer et al., 2015). To test this hypothesis, Sonnery-Cottet et al., (2015) performed a combined ACL & ALL reconstruction on patients with damage to their ACL and anterolateral capsule and presenting with a positive pivot shift sign (grades I-III) (Table 1).

Table 1 Clinical grading of the pivot shift test. (Jakob, Stäubli, & Deland, 1987).

<table>
<thead>
<tr>
<th>Injury Grade</th>
<th>Anterior displacement of the lateral tibial plateau</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>12 mm</td>
</tr>
<tr>
<td>II</td>
<td>18 mm</td>
</tr>
<tr>
<td>III</td>
<td>22 mm</td>
</tr>
</tbody>
</table>

They reported a dramatic reduction in residual pivot shift compared to other investigations that only performed an ACL reconstruction, with less than 8% of patients presenting with a residual, post-operative pivot shift (grade I). However, pre-operatively, 41 of 92 patients presented with only a grade I injury, indicating that the ALL may not have been damaged. Another study that aimed to determine the relationship between the
pivot shift and injuries to the ACL and anterolateral structures found that the pivot shift was only present following sectioning of the anterolateral structures (i.e. a secondary lesion to the ALL was necessary along with an ACL injury to produce the pivot shift) (Monaco et al., 2012). Spencer et al., (2015) found a similar result when simulating an early phase pivot shift such that, compared to an ACL deficient knee, the magnitude of internal rotation increased significantly following sectioning of the ALL. While the pivot shift is a highly subjective clinical test, the ALL could help explain why there is variation found with respect to the pivot shift sign during a physical exam after an ACL injury occurs; if the ALL is not damaged, it is likely still able to control internal tibial rotation and anterior tibial translation, therefore a pivot shift sign may not be present (Taneja et al., 2014). The pivot shift sign is an important clinical indicator, as a positive pivot shift has been shown to be correlated with negative functional outcomes after ACL reconstruction and can affect longer term outcomes (Ayeni, Chahal, Tran, & Sprague, 2012).

1.6 Function of the Lateral Meniscus

1.6.1 Normal Function

The primary function of the menisci is to allow for load transmission, joint congruency, and shock absorption in the knee (Brody et al., 2007; Koenig et al., 2009). The menisci also play a role in stability of the knee joint, but for the menisci to function in stability, load transmission and shock absorption, they themselves must be stable (Koenig et al., 2009). Therefore intact meniscal roots are essential for normal meniscal function (Guess, Razu, Jahandar, & Stylianou, 2015). The menisci help distribute load across the tibia by making the knee joint more congruent which reduces stress on the articular cartilage (Andrews, Shrive, & Ronsky, 2011). Without functional menisci, the pressures placed on the knee joint can become pathological. For example, during standing, approximately 55% of the downward pressure experienced by the tibia is dissipated by the stretching of the collagen fibers within the meniscus (Brody et al., 2007). This channels the axial force and converts it into ‘hoop stress’, or radially directed force, allowing the wear on the articular cartilage of the knee joint to be reduced (Brody et al., 2007). For ‘hoop stress’ to
be resisted, it is crucial that the meniscal roots are intact and tightly fixated to the tibial plateau to prevent meniscal extrusion during axial loading (Brody et al., 2007)

1.6.2 Function When Damaged

Tears located within the body of the meniscus are well understood and are easily treatable; however a tear to a meniscal root results in a unique pattern of injury that is not as well understood (Koenig et al., 2009). In a study conducted on the medial meniscus, a posterior root tear resulted in meniscal extrusion, causing the articular cartilage of the knee to be exposed to unique pathological loads (Allaire, Muriuki, Gilbertson, & Harner, 2008) that have been identified as a precursor to the development of osteoarthritis (Jones, Houang, Low, & Wood, 2006). Meniscal extrusion is a serious complication of increased laxity of the meniscal roots that results in a loss of function of the menisci (Guess et al., 2015). A complete posterior root tear to the medial meniscus also causes a dramatic increase in contact pressure to the articular cartilage that is functionally equivalent to a complete meniscectomy (Allaire et al., 2008). While the study by Allaire et al., (2008) focuses on the posterior root of the medial meniscus and not the posterior root of the lateral meniscus, it is likely that similar, results would be seen with tears to the LMPR (Shybut et al., 2015). However, more research is needed to determine the biomechanical consequences of lateral meniscus root tears (Koenig et al., 2009).

1.6.3 Functional Relationship with the Anterior Cruciate Ligament

Similar to the ALL, the menisci act as secondary stabilizers to the ACL, and are particularly important when the ACL is injured (Andrews et al., 2011; Shybut et al., 2015). The LMPR and ACL function together to reduce anterior translation and internal rotation. When performing the pivot shift, a knee that was ACL and LMPR deficient had significantly greater anterior translation than a knee that was only ACL deficient (Shybut et al., 2015). When resection of the lateral meniscus occurs in an ACL deficient knee the anterior translation of the lateral compartment increases significantly by 6mm when a pivot shift exam is performed (Musahl et al., 2010). A tear to the LMPR causes a reduction in knee joint stability by increasing rotational laxity in an ACL deficient knee (Bhatia, Laprade, Ellman, & Laprade, 2014) and if a lateral meniscectomy is performed,
that same knee becomes more unstable during a pivot shift maneuver, further suggesting that the lateral meniscus assists the ACL in resisting rotational moments (Musahl et al., 2010).

1.7 Injury Patterns

1.7.1 Anterolateral Ligament and Anterior Cruciate Ligament

Over the last 20 years it has become evident that there is a relationship between the rupturing of the anterolateral structures of the knee and the ACL (Rezansoff et al., 2014; Terry et al., 1993; Van Dyck et al., 2016). The mid-third lateral capsular ligament, which corresponds to the ALL, is frequently injured during a rupturing event to the ACL (Hughston et al., 1976; Sonnery-Cottet et al., 2015) potentially due to their shared injury mechanism of internal rotation; a movement that they both work to resist. However the study by Hughston et al., (1976) is a descriptive surgical report, and therefore despite finding the mid-third lateral capsular ligament (ALL) to be damaged in 100% of the knees, this may be due to a bias in only reporting the cases that presented this way. In a surgical study that examined 82 patients with acute knee injuries and anterolateral rotatory laxity, 98% of knees presented with an ACL tear, and of those, 93% also presented with injuries to the capsule-osseous layer of the ITB which corresponds with the ALL (Terry et al., 1993). A systematic review article examined the literature that exists regarding ACL and ALL injury and found that in most of the literature there is a high incidence of ALL lesions (up to 46%) (Van Dyck et al., 2016) in any ACL deficient knee, irrespective of whether anterolateral rotatory instability was present or not, although severity of lesions was not assessed (Bonasia et al., 2015).

1.7.2 Lateral Meniscus and Anterior Cruciate Ligament

While injuries to the lateral meniscus in conjunction with an injury to the ACL are not as well documented compared to the injury pattern between the medial meniscus and ACL, a clear relationship between lateral meniscus and ACL injuries has been demonstrated (Brody et al., 2007; Shelbourne & Gray, 2000). In a retrospective study examining 156 patients, it was reported that lateral meniscus injuries likely occur simultaneously with an ACL injury as opposed to being a chronic injury that transpires over time after the ACL
injury has occurred (Gadeyne, Besse, Galand-Desme, Lerat, & Moyen, 2006). Concomitant ACL and meniscal injuries usually involve the LMPR (Brody et al., 2007) in the form of a radial tear located next to the meniscal root (Bellabarba, Bush-Joseph, & Bach, 1997; Binfield, Maffulli, & King, 1993; Bray & Dandy, 1989). Given the function of the meniscal roots to anchor the menisci to the tibial plateau, it is no surprise that with these sorts of tears, meniscal extrusion is often associated with these concomitant injuries because the meniscus is no longer anchored to the tibial plateau (Brody et al., 2007; Koenig et al., 2009).

1.7.3 Anterolateral Ligament and Lateral Meniscus

While there has not been a definitive study that links injury to both the ALL and the lateral meniscus, it has been postulated that this relationship exists. Helito et al., (2013) hypothesized that due to the strong connection between the ALL fibers and the lateral meniscus, the ALL contributes to the formation of lateral meniscus tears, specifically rim detachment tears. Helito et al., (2013) are not alone in their belief and Vincent et al., (2012) even proposed an injury mechanism where they suggested that in the case of an avulsion of the ALL from its insertion on the tibia, any form of additional tibial translation could place tension on the meniscus due to the ALL’s meniscal attachment and remaining femoral attachment, and this force may contribute to the rupture of the lateral meniscus which is still anchored at its roots (Vincent et al., 2012). Alternatively, Shybut et al., (2015) suggests that lateral meniscal injuries may occur first, further destabilizing the ACL deficient knee, which may result in damage to the ALL.

1.7.4 Anterolateral Ligament, Lateral Meniscus and Anterior Cruciate Ligament

To date, there is limited data that describes the concomitant injury pattern between the ALL, lateral meniscus and ACL, however two studies exist that have looked at associated findings when the anterolateral compartment is injured. Prior to anterolateral ligament becoming a standard term, Hughston et al., (1976) identified the capsular ligament as the middle third of the lateral ligament structures. In one of the earliest studies, tears to the middle third of the lateral ligaments were found in five knees during surgical operations.
for acute lateral compartment instability, and of those five knees, only one presented with an isolated tear to what is now referred to as the ALL (Hughston et al., 1976). Furthermore, three of those five injured knees also had a tear in the lateral meniscus, and one had an associated tear in the ACL (Hughston et al., 1976). In a follow up study, 20 knees with chronic lateral compartment instability were examined in surgery and all presented with a lesion (scar tissue or excessive laxity) to the middle third of the lateral ligaments with tears of the meniscus and/or the ACL identified (Hughston et al., 1976). In a more recent study, MRIs of 90 patients with an acute ACL-rupture were examined. Of those knees, 46% presented with an ALL injury and when an abnormal ALL was present, 61% also had a torn lateral meniscus with 85% occurring at the posterior horn (Van Dyck et al., 2016). Of the knees that presented with no injury to the ALL, only 31% had a torn lateral meniscus. This study shows that ALL injuries are common in patients with acute ACL ruptures, and ALL injuries are highly associated (p= 0.008) with lateral meniscus injuries (Van Dyck et al., 2016).

1.7.5 Segond Fracture

The Segond fracture was first described over 130 years ago and is an avulsion of the tibia posterior to Gerdy’s tubercle (Irvine et al., 1987) (Figure 4). Dr. Paul Segond also described a band of the fibrous capsule that placed tension in the location of the Segond fracture when the knee was placed into forced internal rotation (De Maeseneer et al., 2014). Since the insertion point of the ALL is consistent with the footprint of the Segond fracture, much research has focused on the role of the ALL in Segond fractures, with the majority of research hypothesizing that the ALL is responsible for these fractures (Claes et al., 2013; De Maeseneer et al., 2014; Helito et al., 2013; M. I. Kennedy et al., 2015; Parsons et al., 2015; Porrino et al., 2015; Van der Watt et al., 2015). Some studies have looked at the ALL and Segond fracture in isolation (M. I. Kennedy et al., 2015; Porrino et al., 2015), while others have looked at the ALL and Segond fracture in combination with the ACL (Campos et al., 2001; De Maeseneer et al., 2014; Irvine et al., 1987; Parsons et al., 2015). Of the first group of studies, biomechanical data supports the notion that the ALL is responsible for the Segond fracture given that the insertion location and tensile strength of the ALL were able to produce a Segond fracture in six out of 15 specimens.
(M. I. Kennedy et al., 2015). When examining the rupture pattern of the ALL in pull-to-failure testing, a Segond fracture from the tibial attachment was the most common mechanism of failure for the ALL (M. I. Kennedy et al., 2015). In an MRI study of 20 knees with Segond fractures, 19 showed the ALL attaching to the tibial fracture fragment (Porrino et al., 2015). In a similar study that broadened the scope of investigation for the Segond fracture, De Maeseneer et al., (2014) examined whether the Iliotibial Band was also involved. Their results showed that 85% of the fracture fragments had the ITB attached to it, and 77% also had the ALL attached to it, with the ALL never attaching in isolation without the ITB, indicating that the ITB may also play an important role in the genesis of the Segond fracture.

![X-ray of a Segond fracture.](Radiopedia.org, Creative Commons Use)

**Figure 4** X-ray of a Segond fracture. The white arrow indicates the tibia avulsing. *(Radiopedia.org, Creative Commons Use).*
For the studies that have examined the Segond fracture in relation to both the ALL and ACL, it appears that many papers would consider the Segond fracture to be pathognomonic for an ACL rupture, meaning that if a Segond fracture is present, an ACL rupture must therefore also be present too (Campos et al., 2001; Irvine et al., 1987; Parsons et al., 2015). This logic follows given that the Segond fracture results from a combination of internal rotation and a varus moment which is one of the potential injury mechanisms for ACL (De Maeseneer et al., 2014; Parsons et al., 2015; Yu & Garrett, 2007). This was also confirmed in an MRI study in which any knee with a Segond fracture also presented with an injury to the ACL (ranging from grade I-III) (Table 2) (De Maeseneer et al., 2014). Given that both the ALL and ACL contribute to the anterolateral rotational stability of the knee, a Segond fracture is associated with increased instability of the knee joint due to the rupturing of the ALL from its insertion point, in an already unstable, ACL deficient knee (Campos et al., 2001).

Table 2 Grading scale used for ACL tears when viewed on MRI. (De Maeseneer et al., 2014).

<table>
<thead>
<tr>
<th>Injury Grade</th>
<th>Lesion Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Continuity of ligament, but edematous changes or fluid surrounding fibers</td>
</tr>
<tr>
<td>II</td>
<td>Tear of one of the bundles of the ACL or a wavy contour but with global preservation of continuity</td>
</tr>
<tr>
<td>III</td>
<td>Complete interruption of the ACL</td>
</tr>
</tbody>
</table>

1.8 Biomechanics of the Anterolateral Ligament

1.8.1 Mechanical Properties

When defining the behavior of a material it is important to quantify its mechanical properties. However, little work has been performed specifically on the ALL. The mechanical properties that are important to define are: i) ultimate load to failure; the maximum force a material can withstand before it fails; ii) ultimate strain; the amount of deformation a material undergoes at failure; and iii) stiffness; the resistance a structure offers to deformation (Pal, 2014). What is known about the ALL is that its tensile
properties are considerably weaker than the primary stabilizers of the knee joint (Zens, Feucht, et al., 2015). In a standard load-to-failure protocol, the ultimate load of the ALL was 49N, in comparison to the ACL, MCL and PCL which can all withstand over 1,000N of force before failing (Noyes, Butler, Grood, Zernicke, & Hefzy, 1984; Zens, Feucht, et al., 2015). In a different load-to-failure study, the ALL was determined to have an ultimate load of 175N (M. I. Kennedy et al., 2015), which is greater than a three-fold increase compared to the study by Zens et al., (2015). It has been noted that the ALL has an ultimate strain of 0.36, and the failure point occurred at approximately 1/3 of the length of the ALL, from the femoral insertion (Zens, Feucht, et al., 2015). The average ALL stiffness has been calculated at 20 N/mm (M. I. Kennedy et al., 2015). Given the paucity of data and the variability of values between studies, this highlights the lack of understanding regarding the mechanical properties of the ALL.

1.8.2 Translation and Rotation

As previously stated, the ALL has been hypothesized to control anterolateral stability of the knee (Rezansoff et al., 2014), and recent investigations have examined the ALL’s contribution to internal rotation and anterior translation. Parsons et al., (2015) determined that the ALL is minimally loaded in anterior translation and does not play a significant role in preventing anterior translation at any flexion angle. This idea was further supported by Spencer et al., (2015) and Saiegh et al., (2015), who also discovered no difference in anterior translation when the ALL was sectioned in an ACL deficient knee. In contrast, a serial-sectioning study by Tavlo et al., (2015) found that anterior-posterior stability decreased, as measured by a maximum, manual pull anteriorly, when the ALL was cut in an ACL deficient knee. In another study, it was determined that cutting the anterolateral structures of the knee increased anterior translation in an ACL deficient knee by 2-4mm, at 30°, 60° and 90° of knee flexion (Monaco et al., 2012). Further support for its role in resisting anterior translation is provided by another serial-sectioning study that found a small but significant increase in anterior translation of 0.2-1.6mm across all knee flexion angles (0-120°) when the ALL was sectioned in an ACL deficient knee (Rasmussen et al., 2015). As this is a new area of research for the ALL, it is clear
that more studies need to be conducted to be able to conclusively determine whether the ALL plays a role in controlling anterior translation.

Conversely, the literature provides slightly more insight into the ALL’s contribution to resisting internal rotation. In one serial-sectioning study, there were no further increases in internal rotation when the ALL was cut in an ACL deficient knee (Saiegh et al., 2015). However, Saiegh et al., (2015) used whole lower limbs and therefore the Iliotibial band remained intact. Since the ITB is an important stabilizer for anterolateral rotation, a disrupted ITB could affect rotational stability (Saiegh et al., 2015). However, the majority of the literature does not support the results presented by Saiegh et al., (2015). Monaco et al., (2012) who utilized intact lower limbs with the ITB undisrupted, concluded that no significant rotational laxity was present in an ACL deficient knee; the anterolateral structures had to be sectioned to cause a large increase in internal rotation of 5.5° (Monaco et al., 2012). Additional biomechanical studies have found positive results and concluded that sectioning of the ALL resulted in a significant increase in internal rotation of about 2° (Rasmussen et al., 2015; Spencer et al., 2015) in an ACL deficient knee (Rasmussen et al., 2015; Spencer et al., 2015; Tavlo et al., 2015).

### 1.8.3 Contribution at Different Angles

In one study that examined the ALL’s role in resisting internal rotation, we see the ALL playing a key role at angles >35° with it achieving a maximum estimated contribution (calculated by the division of the force vector magnitude by the intact ligament force vector magnitude) of 45% at 75° (Parsons et al., 2015). This is supported by research that has investigated the change of length in the ALL during flexion and during internal rotation at different degrees of knee flexion. Dodds et al., (2014) examined the length of the ALL from 0-90° of knee flexion and found that while the ALL slackened at flexion angles >60°, it could be tightened and lengthened by applying an internal rotation torque. A more recent study corroborated these findings, noting that the ALL significantly increased in length with applied internal rotation, and it’s greatest change in length (12%) was significantly greater at 90° of knee flexion compared to any other knee flexion angle (Zens, Niemeyer, et al., 2015). This work would classify the ALL as a non-isometric structure that comes under tension with applied internal rotation and knee flexion (Zens,
Niemeyer, et al., 2015). However some discrepancy in the literature does exist such as a serial sectioning study that concluded that when the ALL was sectioned in an ACL deficient knee, internal rotation only increased significantly at low degrees of knee flexion (30°) (Monaco et al., 2012). While in another serial sectioning study, sectioning the ALL and ACL resulted in a significant increase of 2.7° of internal rotation compared to just sectioning the ACL for all knee flexion angles between 0°-120° (in 15° increments) (Rasmussen et al., 2015). However, for that same study, the ACL deficient state had a significant increase in internal rotation compared to an intact state at flexion angles <45°. This may help corroborate the findings by Parsons et al., (2015) that the ACL plays a more dominant role than the ALL in limiting internal rotation at smaller degrees of flexion (Rasmussen et al., 2015). The ALL is involved in resisting internal rotation when the knee moves into flexion, but the literature is not definitive on how much flexion.

1.9 Biomechanics of the Lateral Meniscus

1.9.1 Mechanical Properties

The roots of the menisci are a unique part of the meniscus, and therefore have their own unique mechanical properties from the body of the meniscus. When comparing the anterior roots to the posterior roots for the lateral meniscus, the anterior root is stronger than the posterior root (Hauch, Villegas, & Haut Donahue, 2010). During a pull-to-failure test, the anterior root was determined to withstand forces of 652N compared to the posterior root which could only withstand forces of 330N (Hauch et al., 2010). The anterior root was calculated to have a stiffness of 216N/mm compared to the posterior root which was calculated at 130 N/mm (Hauch et al., 2010). When focusing specifically on the LMPR, it was noted to exhibit the greatest failure strain, at 0.94, compared to the other meniscal roots that ranged from 0.49-0.82 (Abraham, Moyer, Villegas, Odegard, & Haut Donahue, 2011). It has also been found that posterior root tears of the menisci are more common than anterior root tears (Abraham et al., 2011; Ahn et al., 2009), and this may be due to the smaller peak loads that the posterior root can withstand (Hauch et al., 2010). Despite being composed of identical materials, the difference in mechanical properties of the meniscal roots can potentially be explained by the variation in how the
collagen and its interwoven fibrils are organized in each of the different meniscal roots (Abraham et al., 2011).

1.10 Clinical Pathology

1.10.1 The Anterolateral Ligament

While the biomechanical and functional roles of the ALL have been discussed, the clinical effects of damage to the ALL are important considerations for health practitioners such as surgeons and physiotherapists. Although this area has received little attention to date, one finding was that lateral compartment instability is unique and perhaps more debilitating when compared to medial compartment instability of the knee (Hughston et al., 1976). This is because during terminal extension in gait for example, the ligaments of the lateral compartment are under a considerably greater amount of tension than the ligaments of the medial compartment (Hughston et al., 1976). Thus, this early work suggests that damage to the lateral compartment should be addressed through surgical means. More recently, the idea of looking at ALL damage with ACL damage has become a popular area of research. A study examining ACL reconstructions concluded that a surgery completed without any technical flaws may not be capable of restoring normal rotatory control and a positive pivot-shift may remain in up to 7% of those patients (Scheffel, Henninger, & Burks, 2013). This suggests that reconstruction of the intra-capsular structures such as the ACL, is not always capable of restoring normal rotatory control, and the presence of extra-capsular structures such as the ALL may be responsible for assisting in the control of rotational laxity (Helito et al., 2013). A systematic review was recently conducted that examined whether including a lateral extra-articular tenodesis (LET) to ACL reconstruction would improve control of rotational laxity (Hewison et al., 2015). LET is a procedure that reduces anterolateral laxity by reinforcing the anterolateral joint capsule using part of the ITB (Spencer et al., 2015). This systematic review found that there was a significant reduction in pivot shift when the combination of ACL reconstruction and LET were performed (Hewison et al., 2015), suggesting that intra-articular reconstruction is not capable of restoring normal function on its own, and that additionally focusing on the anterolateral capsule is most likely to produce positive patient outcomes.
1.10.2 The Lateral Meniscus

Similar to the ALL, the lateral meniscus is a clinically important structure regarding injuries to the knee. One study found that the lateral meniscus was responsible for providing restraint to anterior tibial translation in an ACL deficient knee experiencing rotational forces during a pivoting maneuver often experienced in sports (Musahl et al., 2010). Given that it plays a role as a secondary stabilizer of the knee, it was suggested that it should be repaired and preserved whenever possible (Musahl et al., 2010). In another study that looked at the medial meniscus, it was found that resecting part of the meniscus (predominantly the posteromedial horn), caused the tibia to rotate externally to a significantly greater degree during the stance phase of gait compared to an intact meniscus condition (Netravali, Koo, Giori, & Andriacchi, 2011). The suggested reason for this is that the loss of meniscal tissue results in less resistance to external rotation (Netravali et al., 2011) and it is possible that the same logic could be applied to the lateral meniscus with respect to internal rotation increasing after resection of the posterior horn. Lastly, James et al. (2014) performed a study that assessed accelerated cartilage degeneration and found a connection between root tears and degeneration of the cartilage. When repairing the root via suturing, it was found that if it was misaligned by a minimum of 3mm, it had the potential to impede normal function for the patient (James et al., 2014). The evidence would suggest that having healthy, intact menisci and meniscal roots is important for overall function of the knee, but it is also crucial to restore the anatomy accurately when surgically repairing the knee to prevent disability.

1.11 Study Purpose

It has been shown that the ALL inserts onto the lateral meniscus (Helito et al. 2013), and that it plays a role in limiting internal rotation (Spencer et al., 2015), but this functional relationship between the ALL and lateral meniscus requires further exploration, as well as the implications of this connection investigated. The ALL and lateral meniscus were evaluated biomechanically to determine the effect the ALL and LMPR have on controlling internal rotation. In addition, the different parts of the ALL as they relate to the meniscus were studied.
1.12 Study Objectives

1) To determine the role of the ALL and LMPR in controlling internal rotation of the knee through biomechanical testing and serial sectioning of the ACL, ALL and LMPR.

2) To explore the supra-meniscal and infra-meniscal parts of the ALL and their connection to the meniscus to determine their gross anatomy, histology and biomechanical properties.

1.13 Hypothesis

1) It is hypothesized that the ALL and lateral meniscus will work synergistically, and that a tear to the LMPR will have the same effect on anterolateral laxity as an ALL tear in the ACL deficient knee.

2) It is hypothesized that the supra-meniscal part of the ALL will have more tensile strength and the connection between the femur and meniscus will be able to withstand more load than the connection between the tibia and the meniscus.
1.14 References


Segond, P. (1879). *Recherches cliniques et expérimentales sur les épanchements*
sanguins du genou par entorse. Aux Bureaux du Progrès médical.


Chapter 2

2 Histological Analysis of the Supra- and Infra-meniscal Sections of the Anterolateral Ligament

2.1 Introduction

This chapter explores the histological composition of the anterolateral ligament, specifically comparing the supra-meniscal and infra-meniscal fibers to one another. Previous histological analysis has shown that the ALL is composed of dense regular connective tissue which is characteristic of ligamentous structures (Caterine et al., 2014; Guenther et al., 2015) and primarily consists of Type I collagen (Macchi et al., 2015). The ALL is clearly distinguishable from the lateral joint capsule, as the joint capsule resembles loose connective tissue (Caterine et al., 2014), and the ALL’s bony insertion is distinct, as the femoral insertion of the ALL displays a transition from ligament, to mineralized cartilage, to bone that is characteristic of ligamentous tissue (Caterine et al., 2014). A study that performed immunohistochemistry on the ALL identified a prominent network of peripheral nerves, indicating a possible role in proprioception (Caterine et al., 2014). A histological study that examined the ALL and the lateral meniscus confirmed that the ALL adheres to the lateral meniscus without a cleavage plane, and some fibers do in fact insert into the meniscus (Vincent et al., 2012). To date however, no study has investigated differences of the collagenous arrangement between the supra- and infra-meniscal fibers of the ALL. Therefore, the purpose of the work described in this chapter was to determine if the supra- and infra- meniscal fibers of the ALL differed in their microscopic structure and collagen organization. It was hypothesized that the histological analysis would reveal no differences in the collagen organization between the supra- and infra-meniscal sections of the ALL. While the ALL has been examined as a whole in the past, the two sections have never been compared to each other, and this part of the study accomplished that.
2.2 Materials and Methods

One fresh-frozen specimen (81 year-old male) was used for histological analysis. Once thawed, all skin was removed from the knee and the IT band was identified and reflected inferiorly to its point of insertion. With the knee placed in 30°-60° of flexion, a varus and internal rotational force was applied to place the ALL fibers under tension. The ALL was determined to be any tissue running from the lateral femoral epicondyle to the lateral aspect of the tibia, just below the tibial plateau and midway between Gerdy’s tubercle and the fibular head. The ALL’s borders were defined and the lateral collateral ligament (LCL) was isolated by blunt dissection due to its shared origin at the lateral femoral epicondyle. All soft tissue except the ALL and lateral meniscus was removed from the knee using both an extra-articular and intra-articular dissection approach to fully isolate the ALL from the tissue and joint capsule on the lateral aspect. The popliteal tendon was dissected off the inner aspect of the ALL and resected along with the meniscofemoral ligament. Lastly, the LCL was sectioned and removed along with any remaining tissue posterior to it. The ALL was then resected from its bony attachments using a scalpel, along with a section of lateral meniscus that was attached to the ALL. The specimen was trimmed down so a small piece of lateral meniscus remained. The specimen was fixed in 10% formaldehyde and embedded longitudinally in paraffin wax. Using a microtome (microm HM-325; GMI Inc., Ramsey, MN) the specimen was sectioned at a thickness of 5 µm, placed in a warm water bath, transferred onto glass slides and placed in a 40° F oven to fix the slides. The slides were then stained with Masson’s Trichrome using standard protocol to allow for better visualization of collagen arrangement. Qualitative analysis of the tissue was carried out using a Zeiss AxioScope.A1 microscope (Zeiss, Toronto, ON) and the images were captured using AxioVision Microscopy Software (LE Rel 4.8; Zeiss, Toronto, ON). The meniscus had to be twisted from its perpendicular orientation to the ALL during the embedding process, so to landmark where the supra- and infra-meniscal fibers were located, the lateral inferior geniculate artery (LIGA) was used as a reference point since it travels between the lateral meniscus and the ALL (Figure 5).
2.3 Results

The LIGA was identified in the histological section and appears bright pink when stained with Masson’s Trichrome (Figure 6a). Throughout the ALL, the collagen was arranged in a dense regular organization that is consistent with the arrangement of ligamentous tissue. In the longitudinal section, the fibers of the ALL revealed collagen running parallel to each other and they exhibited a crimped pattern that is characteristic of dense regular connective tissue (Figure 7a&amp;b). Examination of the supra-meniscal fibers (Figure 7a) and the infra-meniscal fibers (Figure 7b) show a similar collagenous arrangement, with this image of the infra-meniscal fibers being slightly more organized. However, the majority of the infra-meniscal fibers examined, exhibited less organization than this image.
Figure 6 a) LIGA shown in longitudinal section. b) LIGA and supra-meniscal fibers shown.

Figure 7 Fibers of the ALL shown in longitudinal section. a) Supra-meniscal fibers of the ALL. b) Infra-meniscal fibers of the ALL.
2.4 Discussion

The histological analysis confirmed that the anterolateral ligament is ligamentous in nature, and that the supra- and infra-meniscal fibers of the ALL have a similar collagenous organization. Although the infra-meniscal fibers appear slightly more organized than the supra-meniscal fibers in this image, this may have occurred because they were closer to the bony insertion than the supra-meniscal fibers. It should be noted that the histological sections did contain some separation artifact as seen in (Figure 7b). This was difficult to avoid, as the meniscus was included in the section and its rigidity in comparison to the ALL made sectioning difficult.

In both the supra- and infra-meniscal sections of the ALL, the collagen organization was that of dense connective tissue and this supports past histological studies of the ALL (Bonasia et al., 2015; Caterine et al., 2014; Helito et al., 2013; Vincent et al., 2012). Helito et al., (2013) noted the high degree of organization in the tissue that reflects the results we have seen. One study noted that the dense fibrous tissue that composed the ALL appeared more abundant in sections obtained superior to the lateral meniscus (Vincent et al., 2012) which may additionally help explain some of the separation artifact we observed in our infra-meniscal section. Future directions for histological analysis of the ALL should focus on cross-sections of the supra- and infra-meniscal fibers as they insert into the lateral meniscus. This would allow for a comparison of the supra- and infra-meniscal attachment to the meniscus, to determine if they have equivalent insertions. Also, it should be attempted to quantify the amount of collagen within the supra- and infra-meniscal fibers to give a quantitative value to the organization between the two parts of the ligament.
2.5 References


Chapter 3

3 The Contribution of the Anterolateral Ligament and the Lateral Meniscus to the Control of Rotational Knee Laxity

This manuscript was co-first authored by Dr. Tim Lording and Gillian Corbo

Submitted for publication to the American Journal of Sports Medicine

3.1 Introduction

Anterior cruciate ligament (ACL) injury results in both translational and rotational laxity. It is well understood that ACL reconstruction may fail to fully restore rotational stability to the knee (Georgoulis, Ristanis, Chouliaras, Moraiti, & Stergiou, 2007; Ristanis et al., 2005; Tashman, Collon, Anderson, Kolowich, & Anderst, 2004), and that residual rotational laxity is associated with poor patient outcomes (Kocher, 2004; Kocher et al., 2002).

Recent interest in the anterolateral ligament (ALL) has refocused attention on the secondary restraints to internal rotation and the potential contribution that injury to these structures may make to residual instability. In addition to the ACL, the anterolateral ligament (Parsons, Gee, Spiekerman, & Cavanagh, 2015), iliotibial band (Gadikota et al., 2013; Jakob, Hassler, & Staebli, 1981), lateral meniscus (Musahl et al., 2010), and medial meniscotibial ligament (Peltier et al., 2015) all act as restraints to internal rotation at the knee. Anatomic and imaging studies of the anterolateral capsule have shown the ALL to be a distinct ligamentous structure (Caterine, Litchfield, Johnson, Chronik, & Getgood, 2015; Claes et al., 2013; Dodds, Halewood, Gupte, Williams, & Amis, 2014; Kennedy et al., 2015; Vincent et al., 2011), with cadaveric biomechanical studies demonstrating an increase in anterolateral rotation following sectioning of the ALL in the ACL deficient knee (Spencer et al., 2015). However, the clinical relevance of this structure has yet to be fully determined.
The lateral meniscus posterior root (LMPR) has also been shown to contribute to rotational laxity following an injury to the ACL (Shybut et al., 2015). LMPR tears have been shown to be found in 7-12% of patients with ACL injury (Feucht et al., 2014), whilst in a cadaveric study, Shybut et al., (2015) demonstrated a significant increase in anterior translation during a simulated pivot shift when the LMPR was sectioned in the ACL deficient knee. While the role of the LMPR in aiding the control of anterolateral laxity seems evident following an ACL injury, less is known regarding the functional relationship between the LMPR and the ALL. Anatomical studies of the ALL have demonstrated a firm attachment of the ALL to the lateral meniscus (Caterine et al., 2015; Claes et al., 2013; Helito et al., 2013; Kennedy et al., 2015; Macchi et al., 2015; Vincent et al., 2011), the importance of which remains unknown, but which may indicate a functional interdependence of these two structures in the restraint of internal rotation.

The purpose of this study was to determine the functional relationship between the ALL and LMPR in the control of internal rotation throughout the range of flexion. Our hypothesis was that division of these structures would have a similar effect on increasing internal rotation in the ACL deficient knee.

3.2 Materials and Methods

*Experimental Setup:* Sixteen fresh-frozen cadaveric specimens were tested (8 males) with a mean (SD) age of 79.3 (11.0) years. The approval for the use of this cadaveric material was granted by the Committee for Cadaveric Use in Research, Division of Clinical Anatomy, Western University in accordance with the Anatomy Act of Ontario, Canada (Approval No. 03012013 & 10032014). The cadaveric specimens were thawed at room temperature for 24 hours prior to testing. The specimens were sectioned at the mid-femur and mid-tibia with 5 cm of soft tissue removed from the proximal femur and distal tibia to allow for potting of the specimens into sections of PVC via dental cement (Denstone dental cement; Hereaus Holdings Group GmbH, Hanau, HE, Germany). The femoral end of the specimen was inserted into a custom designed hip joint simulator rigidly mounted to a surgical table (Spencer et al., 2015). The tibial end was rigidly attached to a six degree-of-freedom load cell (MM3A-500; Advanced Mechanical
Technology Inc., Watertown, MA) that provided a measurement accuracy of 5N and 0.14Nm (Figure 8).

Two rigid optical tracking smart marker clusters (Optotrak Certus; Northern Digital Inc., Waterloo ON) were inserted into the proximal tibia and distal femur using orthopaedic bone pins (Figure 8). These markers allowed the tracking of tibial motion with respect to the femur during testing, with an accuracy of 0.1 mm. A series of anatomical landmarks on the femur and tibia were digitized following testing allowing the creation of bone specific coordinate systems. Three dimensional knee joint kinematics were calculated using the joint coordinate system method as described by Grood and Suntay (Grood & Suntay, 1983; Spencer et al., 2015) (Figure 8).
Figure 8 Experimental setup of the knee within the joint simulator including the position of the optical markers. Also shown is the orientation of the anatomical coordinate system.
Sectioning Protocol: Once the specimens were attached to the simulator they were tested under the following conditions (Figure 9): i) intact knee (Intact) – all soft tissues surrounding the knee joint remained intact; and ii) complete sectioning of the ACL (ACL-) through an anteromedial arthrotomy, followed by sectioning of the ACL, and then sectioning of either the infra-meniscal portion of the ALL (M+/ALL-) or the LMPR (M-/ALL+) which was randomly determined. The ALL was sectioned through an oblique anterolateral incision, with anterior retraction of the iliotibial band and preservation of its capsule-osseous layer, while the LMPR was sectioned through the previously created arthrotomy. Finally, the remaining tissue (either the ALL or LMPR) was sectioned (M-/ALL-). For each condition, a 5Nm internal rotation moment was applied with each motion occurring three times. Testing was performed between 0° and 90° of knee flexion in 15° increments.

![Figure 9](image.png)

Figure 9 Flowchart indicating the sectioning protocol of the knee specimens for biomechanical testing. n=16 unless otherwise indicated.

Data Analysis and Statistics: The magnitude of each motion was determined at each of the force targets on the load cell, and the mean value across each of the three trials was used for all combinations of condition and knee angles. To determine the effect of the intact, ACL-, and M-/ALL- conditions, a one-way ANOVA (three sectionings) was
conducted. To determine the effect of M-/ALL+ and M+/ALL-, separate one-way ANOVAs were conducted within each data set. Finally, an independent t-test was used to determine whether the kinematics were significantly different between M-/ALL+ and M+/ALL-. Post-hoc testing was performed with a Bonferonni adjustment and effect sizes were calculated as partial eta squared ($\eta^2$) and interpreted as small (0.01), medium (0.06), or large (0.14) (Maher et al., 2013). All statistical analyses were performed separately at each knee angle and were performed with SPSS statistical software (v21; IBM Corp, Armonk, NY) with alpha set at 0.05.

3.3 Results

At 0° of flexion (i.e., full extension), there was a significant effect of tissue sectioning ($p=0.02; \eta^2=0.15; \text{power}=0.75$) such that M-/ALL- was significantly greater than ACL- ($p=0.03$) and the Intact ($p=0.01$) conditions by 1.2° and 2.5° respectively (Table 3, Figure 10a). In addition, M+/ALL- ($p=0.05$) and M-/ALL+ ($p=0.04$) were significantly greater than the Intact knee (Table 3, Figure 10a). A trend towards greater internal rotation with LMPR sectioning was observed but this was not statistically significant.

At 15° and 30° of knee flexion there was no significant effect of sectioning the tissues ($p=0.15$ and $p=0.08$, respectively) (Table 3, Figure 10b&c).

As the knee was flexed to 45° there was a significant tissue sectioning effect on internal rotation ($p<0.01; \eta^2=0.29$) (Table 3, Figure 10d). Here, ACL- ($p=0.02$), M-/ALL- ($p=0.01$), and M+/ALL- ($p<0.01$) were significantly greater than the Intact condition by an average of 7°. A trend of increasing internal rotation with ALL sectioning and an intact LMPR was observed but this was not statistically significant.

A similar (non-significant; $p=0.45$) trend was found at 60° such that M+/ALL- resulted in 6.4° and 7.4° of additional rotation compared to the Intact and ACL- conditions (Table 3, Figure 10e). Although not statistically significant, the large effect size ($\eta^2=0.15$) suggests that these are meaningful differences.

At 75° there was a statistically significant difference ($\eta^2=0.24$) between the Intact and M+/ALL- ($p<0.01$) and M-/ALL- ($p=0.01$) conditions. Furthermore, significant
differences were observed between ACL- and M+/ALL- (p<0.01), as well as between M+/ALL- and M-/ALL- (p=0.01) (Table 3, Figure 10f).

At 90° of knee flexion there was no significant main effect of sectioning the tissues (p>0.05) (Table 3, Figure 10g).

Table 3 Comparison of the mean (SD) internal rotation (°) between the different tissue conditions, across all knee flexion angles.

<table>
<thead>
<tr>
<th>Knee Angle</th>
<th>Intact</th>
<th>ACL-</th>
<th>M-/ALL+</th>
<th>M+/ALL-</th>
<th>M-/ALL-</th>
</tr>
</thead>
<tbody>
<tr>
<td>0°</td>
<td>9.12</td>
<td>9.90</td>
<td>12.05</td>
<td>9.99</td>
<td>11.42</td>
</tr>
<tr>
<td></td>
<td>(4.32)</td>
<td>(4.67)</td>
<td>(5.34)</td>
<td>(5.39)</td>
<td>(4.77)</td>
</tr>
<tr>
<td>15°</td>
<td>13.63</td>
<td>15.00</td>
<td>16.04</td>
<td>15.86</td>
<td>15.20</td>
</tr>
<tr>
<td></td>
<td>(4.95)</td>
<td>(6.38)</td>
<td>(7.02)</td>
<td>(11.07)</td>
<td>(6.01)</td>
</tr>
<tr>
<td>30°</td>
<td>17.66</td>
<td>19.98</td>
<td>18.98</td>
<td>21.70</td>
<td>17.94</td>
</tr>
<tr>
<td></td>
<td>(8.32)</td>
<td>(8.03)</td>
<td>(9.50)</td>
<td>(8.61)</td>
<td>(6.68)</td>
</tr>
<tr>
<td>45°</td>
<td>12.78</td>
<td>19.15</td>
<td>17.35</td>
<td>23.70</td>
<td>18.80</td>
</tr>
<tr>
<td></td>
<td>(9.23)</td>
<td>(9.49)</td>
<td>(7.22)</td>
<td>(7.00)</td>
<td>(8.27)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>60°</td>
<td>15.59</td>
<td>16.57</td>
<td>16.03</td>
<td>22.96</td>
<td>17.34</td>
</tr>
<tr>
<td></td>
<td>(6.47)</td>
<td>(8.84)</td>
<td>(6.80)</td>
<td>(6.90)</td>
<td>(5.00)</td>
</tr>
<tr>
<td>75°</td>
<td>13.96</td>
<td>16.28</td>
<td>17.80</td>
<td>23.22</td>
<td>18.05</td>
</tr>
<tr>
<td></td>
<td>(5.34)</td>
<td>(6.44)</td>
<td>(8.16)</td>
<td>(4.46)</td>
<td>(7.31)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>90°</td>
<td>14.02</td>
<td>15.38</td>
<td>16.52</td>
<td>16.10</td>
<td>17.22</td>
</tr>
<tr>
<td></td>
<td>(5.97)</td>
<td>(6.27)</td>
<td>(8.81)</td>
<td>(5.51)</td>
<td>(8.54)</td>
</tr>
</tbody>
</table>

aSignificantly different from intact (p<0.05)
bSignificantly different than ACL- (p<0.05)
cSignificantly different than M-/ALL- (p<0.05)
Figure 10 Degree of internal rotation at each sectioning condition at a) 0°; b) 15°; c) 30°; d) 45°; e) 60°; f) 75°; g) 90° of flexion.
3.4 Discussion

The most important finding of this study was that sectioning either the ALL or the LMPR resulted in a significant increase in internal rotation in the ACL deficient knee, with results suggesting that the contribution of the meniscus and the anterolateral ligament to internal rotation of the knee is dependent on flexion angle. The LMPR appeared to have a greater role in controlling internal rotation in extension whilst the ALL was observed to exert greater control in higher degrees of flexion. Subsequent sectioning of the other structure was not observed to have an additive effect. Sectioning studies generally show an additive effect of dividing various structures that act as restraints to the same movement (Amann et al., 2013; Haimes, Wroble, Grood, & Noyes, 1994; Wroble, Grood, Cummings, Henderson, & Noyes, 1993). In this study, sectioning the ALL after the LMPR, or the LMPR after the ALL did not further increase internal rotation. This suggests a functional link between the two structures, possibly related to their anatomical attachment and degree of knee flexion.

Biomechanical studies examining the role of the ALL in knee kinematics have shown conflicting results. Monaco examined the effect of cutting the ACL and lateral capsular ligament using a navigation system and manually applied forces (Monaco et al., 2011). His description of division of the lateral capsular ligament would have involved division of the ALL. He demonstrated increased internal rotation at all knee flexion angles in the ACL deficient knee following division of the lateral capsular ligament, which was significant at 30° with a 5.5° increase in internal rotation. Spencer investigated both sectioning and reconstruction of the ALL using navigation and manually applied forces (Spencer et al., 2015). Internal rotation in extension increased only 2° after division of the ALL in the ACL deficient knee while performing a simulated pivot shift. Lording, in a cadaveric experiment using a robotic knee examination device, found division of the ALL in the ACL intact knee increased internal rotation at 30° of knee flexion by 2.4° (Lording et al., n.d.). Division of the Iliotibial band increased internal rotation by 2.6°, and sectioning of both the ALL and Iliotibial band by 3.4°.

Parsons, using a six-degree-of-freedom robot and a force subtraction model, found the ALL to be the primary restraint to internal rotation at knee flexion angles greater than
35°, with the ACL providing the greatest restraint closer to extension (Parsons et al., 2015). The iliotibial band was removed in this experiment. Zens et al., (2015) also showed that the ALL increased in length with greater degrees of flexion, suggesting that an ALL reconstruction should be tensioned at 90° to avoid over constraint. These studies corroborate the findings from this present experiment, showing that the ALL has a greater role in controlling internal rotation at higher degrees of flexion.

In contrast to these studies, Kittl found the ALL played no significant role in internal rotational control (Kittl et al., 2016). Using a similar robotic system to Parsons, he determined the superficial and deep components of the ITB to be the primary restraints to internal rotation from 30-90°, with the ACL having a significant contribution at 0° only. Similarly, Saiegh found no significant increase in internal rotation after division of the ALL in the ACL deficient knee (Saiegh et al., 2015); however, the capsulosseous layer of the ITB was removed in this study.

Injuries to the meniscal roots have biomechanical consequences similar to total meniscectomy (Allaire, Muriuki, Gilbertson, & Harner, 2008; Marzo & Gurske-DePerio, 2008), and may lead to meniscal extrusion and progressive degenerative change (Brody, Lin, Hulstyn, & Tung, 2006; Hein, Deperio, Ehrensberger, & Marzo, 2011; Lerer, Umans, Hu, & Jones, 2004). These effects may be somewhat mitigated in the case of the lateral meniscus by the presence of intact meniscofemoral ligaments (Bao, Zhu, Gong, & Gu, 2012; Forkel et al., 2013; Forkel, Reuter, et al., 2014b; Perez-Blanca et al., 2015). Tears involving the posterior root of the lateral meniscus have been reported in up to 12% of ACL injured knees (West, Kim, Armfield, & Harner, 2004). Despite a number of studies of contact pressures after root injury (Bao et al., 2012; Ode et al., 2012; Schillhammer, Werner, Scuderi, & Cannizzaro, 2012), numerous reports of repair techniques (Ahn et al., 2009; Petersen & Zantop, 2006) and three described classification systems (Ahn et al., 2010; Forkel, Reuter, et al., 2014b; West et al., 2004), there is relatively little data regarding the effect of lateral root injuries on knee stability. Musahl reported a significant increase in the pivot shift, but not anterior translation, after lateral meniscectomy in the ACL deficient knee, suggesting a role for the lateral meniscus in internal rotational control (Musahl et al., 2010). Petrigliano reported increased rotational
instability after uni- and bi-compartmental meniscectomy, although they did not differentiate between which compartment was meniscectomized first (Petrigliano, Musahl, Suero, Citak, & Pearle, 2011). These findings are further supported by a recent biomechanical study by Shybut et al., (2015). In this cadaveric study using navigation and a computer controlled pivot shift simulator, release of the lateral meniscal posterior root and the meniscofemoral ligaments significantly increased tibial displacement under a rotational load when compared to the ACL deficient knee. Anterior translation was not significantly increased. Our findings of increased internal rotation after release of the lateral meniscal posterior root are consistent with these studies.

There is one clinical outcome study of lateral meniscal root tears left untreated at the time of ACL reconstruction. Shelbourne reported long term clinical results at a mean follow-up of 10 years (Shelbourne, Roberson, & Gray, 2011). There was no difference in IKDC subjective or objective outcome measures between those with a root tear compared with a cohort with no meniscal tear; however there was 1 mm loss of lateral joint space in the tear group. Pivot shift results were not reported.

Biomechanical studies of transosseous repair techniques in lateral root tears generally show a reduction in contact pressures to near normal levels compared to the injured state (Forkel, Herbort, et al., 2014a; LaPrade et al., 2014; Perez-Blanca et al., 2015), with clinical studies showing encouraging results. Anderson reported the mid-term results of eight radial root tear suture repairs and 16 posterior horn reattachments through a tibial ACL tunnel (Anderson et al., 2010). Twenty-two of 24 repairs functioned successfully, with better subjective results in the transosseous repair group. Ahn reported second look arthroscopic results for eight patients treated with either side-to-side or transosseous techniques, with almost complete healing noted in all cases at a mean of 18 months (Ahn et al., 2009). Kenny reported the 20 year outcome of a case of transosseous repair for isolated posterior root avulsion (Kenny, 2009). Subjective function was good, articular cartilage thickness was maintained and the meniscal appearance was nearly normal on magnetic resonance imaging.
Clinical studies investigating the relationship between the ALL and the lateral meniscus are scarce. In a recent case series of 90 knees with ACL injury shown on MRI, 46% were noted to have an abnormality of the ALL (Van Dyck et al., 2016). Of those knees with an intact ALL, 31% were observed to have a lateral meniscus tear. In contrast, 61% of knees with an ALL injury had a lateral meniscus tear.

The combined results of these studies question the logic of ALL reconstructions in combination with ACL reconstruction to control the pivot shift, a clinical examination performed between 0-30° of flexion. The majority of biomechanical studies show that the ALL has its greatest role in controlling internal rotation in higher degrees of flexion. Based on this information, in combination with the results of our study, we suggest that the lateral meniscus should be thought of as part of an anterolateral capsulomeniscal complex – the anterolateral corner - involving the anterolateral capsule (ALL), lateral meniscus and iliotibial band, that acts as a stabilizer to anterolateral rotational laxity throughout the range of flexion in conjunction with the ACL. During ACL reconstruction, the posterior roots of both menisci should be inspected carefully to rule out concomitant injury. Surgical repair of these lesions may therefore be both chondroprotective and improve stability. The addition of ALL reconstruction or lateral extra-articular tenodesis requires further investigation in a well-controlled randomized clinical trial.

The limitations of the study include the use of elderly cadavers that are not representative of the normal ACL injured population. A hip simulator with manually applied forces with optical tracking to assess kinematics was used in this study, which has been shown to produce measurement accuracy with similar standard deviations to that of a six degree of freedom robot and has been validated for use in a previous study. However, it is clear from the results that whilst there are trends in the data with large effect size, many comparisons did not reach statistical significance. This most likely is an issue of random sampling error, with a greater sample size likely to reflect more consistent results and subsequently recommendations could be made with greater certainty and translated through to clinical practice.
3.5 Conclusion

Increased internal rotational laxity in the ACL deficient knee may be attributed to injury of either the anterolateral ligament or the posterior root of the lateral meniscus, two structures of the ‘anterolateral corner’ of the knee. Both structures seemingly work synergistically throughout varying degrees of flexion, with the ALL exerting a greater control of internal rotation in higher degrees of flexion. The meniscal roots should be carefully evaluated at the time of ACL reconstruction, where repair of the posterior root of the lateral meniscus may be beneficial both for stability and long-term chondral protection.
3.6 References


http://doi.org/10.1007/s00167-011-1580-3

http://doi.org/10.1016/j.arthro.2004.02.061


Chapter 4

4  Tensile Properties of the Supra- and Infra-meniscal Fibers of the Anterolateral Ligament

*Submitted for publication to Knee Surgery, Sports Traumatology, Arthroscopy (KSSTA)*

4.1  Introduction

The anterolateral ligament (ALL) is an extra-articular structure of the knee (Caterine et al., 2014; Claes et al., 2013; Helito et al., 2016; M. I. Kennedy et al., 2015; Kosy et al., 2016) and injury to the ALL has been highly correlated with injury to the anterior cruciate ligament (ACL) and the lateral meniscus (Hughston et al., 1976; Van Dyck et al., 2016). Recent evidence suggests that the ALL’s anatomic connection to the lateral meniscus may be related to the creation of lateral meniscus tears during an ACL injury (Helito et al., 2013; Shybut et al., 2015; Vincent et al., 2012). It has been hypothesized that the strong connection between the ALL and the lateral meniscus, contributes to the formation of tears (Helito et al., 2013), specifically within the horn of the meniscus near its root (Van Dyck et al., 2016). Injuries to the lateral meniscus may occur when the ALL is avulsed from the tibia (i.e., a Segond fracture) (Segond, 1879). It has been proposed that if the ALL avulses, any additional tibial translation could place tension on the meniscus as transferred through the ALL’s intact femoral origin, and the force may contribute to the rupture of the lateral meniscus still anchored at its roots (Vincent et al., 2012).

It has recently been suggested that the ALL can be divided into two sections; supra-meniscal fibers that run from the femoral origin to the meniscus, and infra-meniscal fibers that extend from the meniscus to the tibial insertion (Helito et al., 2013). Given the ALL’s role in controlling rotational laxity (i.e., internal rotation) of the knee (Monaco et al., 2012; Parsons et al., 2015; Rasmussen et al., 2015; Sonnery-Cottet et al., 2016; Spencer et al., 2015; Tavlo et al., 2015), it is possible that the two ALL sections have different biomechanical functions in controlling rotation (Helito et al., 2013). Therefore, the ALL-meniscus interaction may have confounded results in past studies that have
sectioned only one of the ALL attachments (most commonly the infra-meniscal fibers) (Helito et al., 2013). Although past research has quantified the mechanical properties of the ALL as a whole (M. I. Kennedy et al., 2015; Zens, Feucht, et al., 2015), no investigation has determined whether the different ALL sections, with respect to their attachment to the lateral meniscus, have different biomechanical functions that would ultimately affect the contribution the ALL makes to anterolateral stability of the knee. Therefore the purpose of this study was to determine if biomechanical differences exist between the supra-and infra-meniscal sections of the ALL.

4.2 Materials and Methods

Anatomic Dissection: Ten fresh-frozen cadaveric knee specimens (7 males; mean (SD) age of 79 (14) years) were used for testing. The approval for the use of cadaveric material was granted by the Committee for Cadaveric Use in Research, Division of Clinical Anatomy, Western University in accordance with the Anatomy Act of Ontario, Canada (Approval No. 10032014). The specimens were thawed at room temperature for 24 hours prior to dissection and testing. Once thawed, the skin was removed from the knee and the IT band was identified and reflected inferiorly to its insertion. With the knee placed in 45° of flexion, a varus and internal rotational moment was applied. This placed the ALL fibers under tension and allowed better visualization and identification of the ALL. The ALL was defined as any tissue running from the lateral femoral epicondyle to the lateral aspect of the tibia, just below the tibial plateau and midway between Gerdy’s tubercle and the fibular head (Van der Watt et al., 2015). The borders of the ALL were defined and the lateral collateral ligament (LCL) was isolated by blunt dissection due to its shared origin at the lateral femoral epicondyle. All of the soft tissues, except the ALL and lateral meniscus were removed from the knee using both an extra-articular and intra-articular dissection approach to fully isolate the ALL from the tissue and joint capsule on the lateral aspect. The popliteal tendon was dissected off the inner aspect of the ALL and resected along with the meniscofemoral ligament. Lastly, the LCL was sectioned and removed along with any remaining tissue posterior to it. Using a digital caliper (EZcal iGaging; San Clemente CA), mid-substance width and thickness measurements of the
ALL were taken superior and inferior to the meniscus in order to calculate cross sectional area.

**Biomechanical Testing:** The specimens were randomly assigned to one of two groups: i) supra-meniscal; and ii) infra-meniscal. The supra-meniscal group (n=5) was prepared by sectioning the bone, leaving only the tibial plateau and the lateral femoral condyle (Figure 11). Any fibers running from the meniscus to the tibia were sectioned, leaving only the supra-meniscal fibers intact and attached to the lateral meniscus. The infra-meniscal specimens (n=5) were prepared by sectioning the bone leaving only the tibial plateau intact. A lateral section of bone (i.e., where the ALL inserts) was removed using a surgical saw (ConMed Linvatec; Utica NY) and care was taken not to damage any part of the meniscus (Figure 12). Therefore the insertion of the ALL was free from the remaining tibia where the meniscus was anchored. Any fibers running from the meniscus
Figure 11 Sample showing how the bones for the supra-meniscal group were prepared, leaving only the lateral femoral condyle and the tibial plateau.

Figure 12 Sample showing how the bones for the infra-meniscal group were prepared, leaving only the tibial plateau with a lateral section removed.
to the femur were sectioned, leaving only the infra-meniscal fibers intact and attached to the lateral meniscus. The bones (tibia and tibial plateau for the infra-meniscal specimens, and femur and tibial plateau for the supra-meniscal specimens) were potted into sections of PVC via dental cement (Denstone dental cement; Hereaus Holdings Group GmbH, Hanau, HE, Germany) such that the remaining portion of ALL was pulling laterally on the meniscus (Figure 13). Sections of wire were threaded through the bone and held in place with bone cement prior to potting to increase adherence within the PVC sections.

**Figure 13** Experimental set-up displaying how the ALL was pulled with reference to the meniscus.
The specimens were then rigidly secured within an Instron® materials testing machine (Instron® 8874; Norwood MA) (Figure 13) where they were statically preloaded at 20N for two minutes. Cyclic pre-loading was performed between 10N and 30N for 10 cycles at 1Hz, immediately followed by a pull to failure test at 1mm/s. Prior to testing, a series of markers were placed on the ALL and meniscus and were tracked with a custom optical tracking system (Basler Pilot GigE Camera; Ahrensborg, Germany and LabVIEW Vision Acquisition System; Austin TX, USA).

The peak force (i.e., the force just prior to ultimate failure) was extracted from the force-time curve. In addition, the stiffness of the ALL-meniscus construct was calculated as the slope of the linear portion of the force-deformation curve between 30% and 70% of the peak force (Burkhart & Andrews, 2010; Duquette & Andrews, 2010). Stress was also calculated using peak force values and cross sectional area. The marker displacement data was analyzed using ProAnalyst motion analysis software (Xctiex Inc.; Woburn MA) resulting in the calculation of strain values for each specimen: i) within the ALL; ii) at the meniscal interface (where the ALL meets the rim of the meniscus); and iii) within the body of the meniscus. A dot was also placed as close to the roots of the meniscus as possible to determine if any displacement was occurring. Finally, the video data allowed an analysis of the failure mechanism and the location of the failure.

Data Analysis and Statistics: The mechanical properties of each ALL-meniscus construct were categorized with the load to failure, stiffness and maximum strain. An independent samples (one-tailed) t-test was used to determine if there were statistically significant differences in peak load and stiffness between the supra- and infra- meniscal constructs. Technical difficulties prevented analysis of all strains in all specimens and therefore, only a descriptive analysis of this data will be included. One of the specimens in the infra-meniscal group had porous bone that prevented secure potting for testing and therefore was removed, resulting in an n=4 for the infra-meniscal group. Effect sizes ($\eta^2$) were calculated with G*power statistical software (v3.1.9.2; Heinrich-Heine-Universitat, Dusseldorf, Germany) (Faul, Erdfelder, Lang, & Buchner, 2007), and were interpreted as small (0.2), medium (0.5), or large (0.8), as described by Cohen’s d criteria. All statistical
analyses were performed with SPSS statistical software (v21; IBM Corp, Armonk, NY) and alpha was set at 0.05.

4.3 Results

There was a significant difference in the peak force between the supra- and infra-meniscal attachments such that the infra-meniscal constructs resulted in greater forces compared to the supra-meniscal constructs (p=0.028; $\eta^2 = 1.58$) (Table 4, Figure 14). The infra-meniscal construct was also significantly stiffer than the supra-meniscal construct (p=0.023; $\eta^2 = 1.80$) (Table 4, Figure 14).

Table 4 Comparison of the mean (SD) peak force and stiffness values for the supra- & infra-meniscal construct (*p<0.05).

<table>
<thead>
<tr>
<th></th>
<th>Supra-meniscal</th>
<th>Infra-meniscal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak Force (N)</td>
<td>132.11 (45.97)*</td>
<td>197.49 (36.12)</td>
</tr>
<tr>
<td>Stiffness (N/mm)</td>
<td>12.30 (7.12)*</td>
<td>24.84 (6.76)</td>
</tr>
</tbody>
</table>

Figure 14 Comparison of the mean (SD) supra- and infra-meniscal failure forces (left axis) and stiffness’s (right axis) (*p<0.05).
For the five supra-meniscal specimens, failure of the ALL-meniscal construct occurred by three distinct mechanisms: mid-ligamentous tear (n=3), ligamentous tear at the femoral origin (n=1) and failure near the meniscal insertion (n=1) (Figure 15). For the four infra-meniscal specimens, failure of the ALL-meniscal construct occurred by two mechanisms: mid-ligamentous tear (n=3) and avulsion of the bony tibial attachment (i.e., Segond fracture) (n=1) (Figure 16).

**Figure 15** Failure mechanisms of the supra-meniscal ALL construct. **a)** Mid-ligamentous tear; **b)** Ligamentous tear at the femoral origin; **c)** Failure near the meniscal insertion.

**Figure 16** Failure mechanisms of the infra-meniscal ALL construct. **a)** Mid-ligamentous tear; **b)** Avulsion of the bony tibial attachment (i.e., Segond fracture).
The cross sectional area of both the supra- and infra-meniscal fibers was determined to be the same at 25.47 mm$^2$ and 25.76 mm$^2$ respectively (Table 5), and the stress for the supra- and infra-meniscal constructs was calculated to be 5.2 N/mm$^2$ and 7.7 N/mm$^2$ respectively.

**Table 5** Comparison of the mean (SD) width, thickness and cross sectional area of the supra- & infra-meniscal sections of the ALL.

<table>
<thead>
<tr>
<th></th>
<th>Supra-meniscal</th>
<th>Infra-meniscal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Width (mm)</td>
<td>9.28 (1.80)</td>
<td>16.47 (2.46)</td>
</tr>
<tr>
<td>Mean Thickness (mm)</td>
<td>2.75 (0.54)</td>
<td>1.56 (0.65)</td>
</tr>
<tr>
<td>Cross Sectional Area (mm$^2$)</td>
<td>25.47</td>
<td>25.76</td>
</tr>
</tbody>
</table>

The largest peak strains occurred within the ALL, with the supra-meniscal fibers straining three times more than the infra-meniscal fibers (1.3 vs. 4, respectively) (Table 6). The peak strain between the meniscal interface and the body of the meniscus were similar between the supra- and infra- meniscal sections and were less than those quantified in the ALL (Table 6). The strain within the body of the meniscus was greater when loaded through the infra-meniscal fibers (Table 6). The mean (SD) displacement of the roots of the lateral meniscus was minimal at 0.08 (0.09) along the y-axis and 0.21 (0.39) along the x-axis.

**Table 6** Comparison of the mean (SD) peak strain of the supra- & infra-meniscal sections experienced by different parts of the ALL-meniscus complex.

<table>
<thead>
<tr>
<th></th>
<th>Supra-meniscal</th>
<th>Infra-meniscal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strain within ALL</td>
<td>1.26 (0.93); n=5</td>
<td>4.09; n=1</td>
</tr>
<tr>
<td>Strain at meniscal interface</td>
<td>0.16 (0.13); n=3</td>
<td>0.18; n=1</td>
</tr>
<tr>
<td>Strain of body of meniscus</td>
<td>0.04 (0.02); n=2</td>
<td>0.37 (0.001); n=2</td>
</tr>
</tbody>
</table>

**4.4 Discussion**

The most important finding of this study was that the ALL-meniscal construct that included the infra-meniscal fibers was significantly stronger and stiffer when compared to the supra-meniscal fiber construct and presented with the same cross sectional area.
The roots of the menisci were found to experience little displacement with the majority of the strain occurring within the ALL.

There is little information available regarding the mechanical properties of the ALL with no clear consensus recorded. In a standard load-to-failure protocol, the ultimate load that the ALL could withstand was 49N (Zens, Feucht, et al., 2015) with a peak strain of 0.36 and a failure point at approximately 1/3 the length of the ALL, closer to the femoral insertion. Conversely, Kennedy et al., (2015) reported a peak load of 175N and an average stiffness of 20 N/mm (range of 9-39 N/mm). Although Kennedy et al., (2015) did not analyze the separate sections of the ALL, the results in the current investigation support their findings as the overall mean peak failure force and stiffness were 132N & 197N and 12 N/mm & 25 N/mm for the supra- and infra-meniscal constructs respectively. The large differences that were seen when compared to Zens et al., (2015) most likely occurred because Zens did not pull to complete failure, and therefore may have stopped prematurely, whereas Kennedy pulled the specimen until it split in half, which was in line with our protocol.

The significantly stronger infra-meniscal fibers that were found here, may in part explain the role of the ligament in Segond fractures (Claes et al., 2013; De Maeseneer et al., 2014; Helito et al., 2013; Irvine et al., 1987; M. I. Kennedy et al., 2015; Parsons et al., 2015; Porrino et al., 2015; Van der Watt et al., 2015). The fact that only a single Segond fracture occurred in the current study may be a result of the potting procedure that was used. To ensure that the specimen was rigidly secured within the pot it was necessary to include cement as close to the ALL-tibia footprint as possible, which may have interfered with these fibers, preventing more Segond fractures. The strength of the ALL-meniscus interface is further evidenced by the fact that only a single specimen failed at the meniscal insertion, suggesting that any damage to the lateral meniscus caused by the ALL would most likely occur within the horns rather than the body of the meniscus (Van Dyck et al., 2016). Additionally, radial tears of the meniscus, frequently located in the middle third of the lateral meniscus, are not uncommon (14% incidence) during arthroscopy (Magee, Shapiro, & Williams, 2002). The ALL may be responsible for some of these tears, as the strong attachment of the ALL at the meniscal body may apply significant
shear through the meniscus during the anterior subluxation of the knee, with the posterior root being held posteriorly by the femoral condyle. In addition, the finding presented here showed minimal root displacement in a pull-to-failure protocol; given that very little displacement occurred at the roots, it is likely that the roots of the menisci exhibit very little strain in comparison to the ALL-meniscus construct, and are therefore more likely to be the source of a meniscal tear. A biomechanical study examining the hyperelastic properties of the meniscal roots reported peak strains for all four roots ranging from 0.49 - 0.94 (Abraham et al., 2011). While some of these strain values are starting to approach the strain values observed in the current study for the body of the meniscus, Abraham’s study harvested the meniscal roots and did a pull-to-failure protocol just on sections of the meniscal roots. It is possible that when the meniscal roots remain in an anatomic setting, the majority of the strain is experienced in the body of the meniscus, which would explain the limited displacement the roots of this study exhibited. The authors acknowledge that only inferences can be made regarding the strain that occurred at the meniscal roots since only displacement, and not strain could be calculated.

Van Dyck et al., (2016), was one of the first to indicate where damage to the ALL was occurring in imaging studies. He noted that 71% of lesions were located in the distal component of the ALL (the component of the ALL inferior to the meniscus) (Van Dyck et al., 2016). Although this appears to contradict the findings of a stronger infra-meniscal ALL, it may be that the supra-meniscal fibers do not experience as much load, therefore, the fibers would not tear, whereas the infra-meniscal fibers may experience more force, and are thus more likely to tear. This may be due to the fact that during a rupturing event of the ACL, the knee collapses into valgus load and flexion, causing the lateral meniscus to move posteriorly off the tibial plateau as it subluxes anterolaterally, causing the infra-meniscal fibers to be placed under tension. Therefore, when the knee moves into valgus and flexion during a non-rupturing event, the infra-meniscal fibers are still loaded more than the supra-meniscal fibers and therefore may adapt to become stronger.

The tensile properties of the ALL and the strong connection at the meniscal interface found in this study, combined with the available research, contributes to the suggestion that the lateral meniscus and ALL, along with the ITB, can be thought of in terms of an
anterolateral capsulomeniscal complex – the anterolateral corner. As such, reconstruction of one or a mixture of these structures in combination with the ACL reconstruction may help restore anterolateral rotational laxity control and warrants further investigation.

As is common with biomechanical studies of this nature, the cadaveric specimens used in the current investigation would be considered elderly and thus the limitations associated with them would be present. Furthermore, although this study used a relatively small sample size the large effect sizes suggest that the findings are still meaningful and clinically significant. While effort was made to place the markers for strain analysis at standardized locations, it was often difficult to locate the transition between the meniscus and ALL and therefore, the data presented here are approximations of this strain. Finally, the testing set-up resulted in the ALL being loaded in a non-physiological way, however this was the only way to ensure the infra- and supra-meniscal were being loaded in a similar manner allowing for a direct comparison.

4.5 Conclusion

The results of this study indicate that the ALL-meniscal construct that includes the infra-meniscal fibers is significantly stronger and stiffer than the construct that includes the supra-meniscal fibers. When examining the ALL-meniscus complex, the majority of the strain experienced in a pull-to-failure test occurs within the ALL and not the meniscus.
4.6 References


5 Overall Discussion and Conclusions

5.1 Overall Discussion

The purpose of this thesis was to determine the relationship between the ALL and the lateral meniscus and how they interact with each other. I aimed to determine the role that the ALL and LMPR exerted in controlling internal rotation in an ACL deficient knee, as well as how the supra- and infra- meniscal parts of the ALL and their attachment to the lateral meniscus differed in their biomechanical properties. Given that sectioning studies examining the ALL only section the infra-meniscal fibers, it was important to determine the biomechanical properties of the parts of the ALL to determine if sectioning only one part could be affecting the biomechanical testing of the ALL as a whole. Overall the following hypotheses were tested:

**Hypothesis 1:** It is hypothesized that the ALL and lateral meniscus will work synergistically, and that a tear to the LMPR will have the same effect on anterolateral laxity and an ALL tear in the ACL deficient knee

**Result 1:** I failed to reject hypothesis 1, in that sectioning either the ALL or the LMPR resulted in a significant increase in internal rotation in the ACL deficient knee. The ALL did however appear to exert greater control in limiting internal rotation at higher degrees of flexion, while the LMPR exerted greater control in extension.

**Hypothesis 2:** It is hypothesized that the supra-meniscal part of the ALL will have more tensile strength and the connection between the femur and meniscus will be able to withstand more load than the connection between the tibia and the meniscus.

**Result 2:** I rejected hypothesis 2 because it was found that the meniscal construct with the infra-meniscal fibers was both stronger and stiffer than its supra-meniscal counterpart.

The results of these studies indicate that there is both an anatomical and functional link between the ALL and lateral meniscus. The study presented in Chapter 3 demonstrated a functional link between the ALL and the LMPR, as internal rotation did not increase when the alternate structure was sectioned. This postulation is based upon the anatomic
connection of the ALL to the rim of the meniscus, and how that may translate into a functional connection between the ALL and LMPR. The study presented in Chapter 4 demonstrated that a strong connection exists between the two structures, as the ALL-meniscal construct only ruptured at the interface between the structures in one out of nine cases. The strain occurring at the meniscal interface was also minimal, indicating a strong connection between the structures. The result of the infra-meniscal fibers having more tensile strength than the supra-meniscal fibers suggests that future biomechanical sectioning studies of the ALL as a whole should section the ALL superior and inferior to the meniscus. This will ensure that the difference in tensile properties between the two parts of the ALL does not confound biomechanical results.

The histological analysis of the ALL was performed to determine if the biomechanical differences observed between the supra- and infra-meniscal fibers of the ALL could be attributed to a difference in collagen organization. However, the results of the analysis indicated that similarities in the collagen organization exist between the supra- and infra-meniscal fibers, therefore the microscopic composition of the ALL likely cannot account for the differences in peak force that was observed. Additionally, cross sectional area was also calculated to determine if the stronger infra-meniscal fibers could be a result of a larger area of tissue and therefore more collagen fibers. However, both the supra- and infra-meniscal fibers presented with the same cross sectional area, and therefore the stress in the infra-meniscal construct was also greater than that experienced by the supra-meniscal construct. Therefore, cross sectional area cannot account for the observed disparity in peak force. The difference in peak force may be due to the fact that the ALL-meniscal construct was used as opposed to sections that consisted of ALL fibers only. The ALL-meniscal interface and the connection that exists between these two structures may differ and may affect the results of the biomechanical testing in a way that is unknown at this time.

The anatomic dissection of the ALL indicated that it originated posterior and proximal to the LCL origin, with fibers of both the ALL and LCL blending to create a common origin point which is supported by previous studies (Caterine et al., 2014; M. I. Kennedy et al., 2015; Kosy et al., 2016; Runer et al., 2015). Past research has also noted that the popliteal
tendon is in close relationship to the lateral meniscus and ALL (Brody et al., 2007; Vincent et al., 2012); however during anatomic dissection it was found that the popliteal tendon was tightly adhered to the ALL and a scalpel was required to resect the popliteal tendon from the deep fibers of the ALL. Overall, 14 knees were dissected for examination of the ALL, and 12 were found to have an ALL present. This prevalence, (86%), is in accordance with Dodds et al., (2014) who reported that approximately 83% of knees had an ALL. However, it is important to note that the two knees that did not present with an ALL were a matched pair from the same cadaveric donor, and may be artificially decreasing the prevalence of the ALL. If one of paired knees is removed, the prevalence increases to 93%, which is more in line with the majority of the anatomic studies that report a 97%-100% prevalence and use unpaired knees (Campos et al., 2001; Caterine et al., 2014; Claes et al., 2013; Helito et al., 2013; Helito, et al., 2014; Patella et al., 2002; Terry et al., 1993; Vincent et al., 2012). This finding may also suggest that even in rare cases when a patient does not present with an ALL, symmetry can be expected and there seems to be no effect of laterality for the ALL. Lastly, the study by De Maeseneer et al., (2014) that found the ITB to be attached to every Segond fracture fragment that the ALL was attached to, differs from the results in Chapter 4. The pull-to-failure of the infra-meniscal fibers of the ALL resulted in one Segond fracture and this supports past literature indicating that the ITB is not essential for causing a Segond fracture, and the ALL is in fact capable of causing one in isolation (Claes et al., 2013; Helito et al., 2013; M. I. Kennedy et al., 2015; Parsons et al., 2015; Porrino et al., 2015; Van der Watt et al., 2015)

5.2 Future Directions

Future studies should continue to investigate the effect of the ALL and the LMPR in controlling both internal rotation and anterior translation, however this should be performed with a robotic knee examination device to ensure standardization of testing, and minimize human error during the application of forces. Additionally, serial sectioning of the ALL should be carried out in an ACL deficient knee, with sectioning of both the supra- and infra-meniscal fibers to determine if they have a different role in controlling internal rotation. In addition, the effect of sectioning the ALL and LMPR
should be investigated in an ACL intact knee. The study presented in Chapter 4 examines the ALL-meniscal construct as a whole, and the meniscal and bone interactions could be influencing the results observed. It would be beneficial to section the supra- and infra-meniscal fibers from their attachments and perform tensile testing on sections of ALL tissue only to determine if the infra-meniscal fibers are stronger, or if the observed difference is a result of a complex interaction between the ALL and meniscus. Related to that, cross-sections of the supra- and infra-meniscal fiber insertions into the lateral meniscus should be examined to determine if they are equivalent.

5.3 Conclusions

As a result of these investigations, the ALL and LMPR have been discovered to play a synergistic role in controlling internal rotational laxity, with the ALL exerting a greater control of internal rotation in higher degrees of flexion. These findings highlight the importance of carefully inspecting the meniscal roots and the ALL at the time of ACL reconstruction or in the presence of increased anterolateral instability. The fibers of the ALL have also been determined to have unique mechanical properties, and the infra-meniscal fibers and their attachment to the lateral meniscus offer more tensile strength and were calculated to be stiffer than their supra-meniscal counterparts.
5.4 References


Appendices

Appendix A: Authorization for publication of the work to Scholarship@Western

I grant, on behalf of UWO, the non-exclusive right to distribute my submission publicly as part of the University of Western Ontario Institutional Repository, Scholarship@Western.
### Appendix B: Copyright permission for use of material provided by John Wiley and Sons

**JOHN WILEY AND SONS LICENSE TERMS AND CONDITIONS**

Mar 24, 2016

---

This Agreement between Gillian Corbo ("You") and John Wiley and Sons ("John Wiley and Sons") consists of your license details and the terms and conditions provided by John Wiley and Sons and Copyright Clearance Center.

<table>
<thead>
<tr>
<th>License Number</th>
<th>3835540017469</th>
</tr>
</thead>
<tbody>
<tr>
<td>License date</td>
<td>Mar 24, 2016</td>
</tr>
<tr>
<td>Licensed Content Publisher</td>
<td>John Wiley and Sons</td>
</tr>
<tr>
<td>Licensed Content Publication</td>
<td>Journal of Anatomy</td>
</tr>
<tr>
<td>Licensed Content Title</td>
<td>Anatomy of the anterolateral ligament of the knee</td>
</tr>
<tr>
<td>Licensed Content Author</td>
<td>Steven Claes, Evie Vereecke, Michael Maes, Jan Victor, Peter Verdonk, Johan Bellemans</td>
</tr>
<tr>
<td>Licensed Content Date</td>
<td>Aug 1, 2013</td>
</tr>
<tr>
<td>Pages</td>
<td>8</td>
</tr>
<tr>
<td>Type of use</td>
<td>Dissertation/Thesis</td>
</tr>
<tr>
<td>Requestor type</td>
<td>University/Academic</td>
</tr>
<tr>
<td>Format</td>
<td>Electronic</td>
</tr>
<tr>
<td>Portion</td>
<td>Figure/table</td>
</tr>
<tr>
<td>Number of figures/tables</td>
<td>3</td>
</tr>
<tr>
<td>Original Wiley figure/table number(s)</td>
<td>Figure 1, Figure 3, Figure 4</td>
</tr>
<tr>
<td>Will you be translating?</td>
<td>No</td>
</tr>
<tr>
<td>Title of your thesis / dissertation</td>
<td>The Relationship Between the Anterolateral Ligament and the Lateral Meniscus</td>
</tr>
<tr>
<td>Expected completion date</td>
<td>Apr 2016</td>
</tr>
</tbody>
</table>
Expected size (number of pages) 80
Requestor Location  Gillian Corbo
301-1265 Richmond St.
London, ON N6A 3M1
Canada
Attn: Gillian Corbo
Billing Type  Invoice
Billing Address  Gillian Corbo
301-1265 Richmond St.
London, ON N6A 3M1
Canada
Attn: Gillian Corbo
Total 0.00 CAD
Terms and Conditions

TERMS AND CONDITIONS
This copyrighted material is owned by or exclusively licensed to John Wiley & Sons, Inc. or one of its group companies (each a "Wiley Company") or handled on behalf of a society with which a Wiley Company has exclusive publishing rights in relation to a particular work (collectively "WILEY"). By clicking "accept" in connection with completing this licensing transaction, you agree that the following terms and conditions apply to this transaction (along with the billing and payment terms and conditions established by the Copyright Clearance Center Inc., ("CCC's Billing and Payment terms and conditions"), at the time that you opened your RightsLink account (these are available at any time at http://myaccount.copyright.com).

Terms and Conditions

• The materials you have requested permission to reproduce or reuse (the "Wiley Materials") are protected by copyright.

• You are hereby granted a personal, non-exclusive, non-sub licensable (on a stand-alone basis), non-transferable, worldwide, limited license to reproduce the Wiley Materials for the purpose specified in the licensing process. This license, and any CONTENT (PDF or image file) purchased as part of your order, is for a one-time use only and limited to any maximum distribution number specified in the license. The first instance of republication or reuse
granted by this license must be completed within two years of the date of the grant of this license (although copies prepared before the end date may be distributed thereafter). The Wiley Materials shall not be used in any other manner or for any other purpose, beyond what is granted in the license. Permission is granted subject to an appropriate acknowledgement given to the author, title of the material/book/journal and the publisher. You shall also duplicate the copyright notice that appears in the Wiley publication in your use of the Wiley Material. Permission is also granted on the understanding that nowhere in the text is a previously published source acknowledged for all or part of this Wiley Material. Any third party content is expressly excluded from this permission.

• With respect to the Wiley Materials, all rights are reserved. Except as expressly granted by the terms of the license, no part of the Wiley Materials may be copied, modified, adapted (except for minor reformatting required by the new Publication), translated, reproduced, transferred or distributed, in any form or by any means, and no derivative works may be made based on the Wiley Materials without the prior permission of the respective copyright owner. For STM Signatory Publishers clearing permission under the terms of the STM Permissions Guidelines only, the terms of the license are extended to include subsequent editions and for editions in other languages, provided such editions are for the work as a whole in situ and does not involve the separate exploitation of the permitted figures or extracts. You may not alter, remove or suppress in any manner any copyright, trademark or other notices displayed by the Wiley Materials. You may not license, rent, sell, loan, lease, pledge, offer as security, transfer or assign the Wiley Materials on a stand-alone basis, or any of the rights granted to you hereunder to any other person.

• The Wiley Materials and all of the intellectual property rights therein shall at all times remain the exclusive property of John Wiley & Sons Inc, the Wiley Companies, or their respective licensors, and your interest therein is only that of having possession of and the right to reproduce the Wiley Materials pursuant to Section 2 herein during the continuance of this Agreement. You agree that you own no right, title or interest in or to the Wiley Materials or any of the intellectual property rights therein. You shall have no rights hereunder other than the license as provided for above in Section 2. No right, license or interest to any trademark, trade name, service mark or other branding ("Marks") of WILEY or its licensors is granted hereunder, and you agree that you shall not assert any such right, license or interest with respect thereto.

• NEITHER WILEY NOR ITS LICENSORS MAKES ANY WARRANTY OR REPRESENTATION OF ANY KIND TO YOU OR ANY THIRD PARTY, EXPRESS, IMPLIED OR STATUTORY, WITH RESPECT TO THE MATERIALS OR THE ACCURACY OF ANY INFORMATION CONTAINED IN THE MATERIALS, INCLUDING, WITHOUT LIMITATION, ANY IMPLIED WARRANTY OF MERCHANTABILITY,
ACCURACY, SATISFACTORY QUALITY, FITNESS FOR A PARTICULAR PURPOSE, USABILITY, INTEGRATION OR NON-INFRINGEMENT AND ALL SUCH WARRANTIES ARE HEREBY EXCLUDED BY WILEY AND ITS LICENSORS AND WAIVED BY YOU.

- WILEY shall have the right to terminate this Agreement immediately upon breach of this Agreement by you.

- You shall indemnify, defend and hold harmless WILEY, its Licensors and their respective directors, officers, agents and employees, from and against any actual or threatened claims, demands, causes of action or proceedings arising from any breach of this Agreement by you.

- IN NO EVENT SHALL WILEY OR ITS LICENSORS BE LIABLE TO YOU OR ANY OTHER PARTY OR ANY OTHER PERSON OR ENTITY FOR ANY SPECIAL, CONSEQUENTIAL, INCIDENTAL, INDIRECT, EXEMPLARY OR PUNITIVE DAMAGES, HOWEVER CAUSED, ARISING OUT OF OR IN CONNECTION WITH THE DOWNLOADING, PROVISIONING, VIEWING OR USE OF THE MATERIALS REGARDLESS OF THE FORM OF ACTION, WHETHER FOR BREACH OF CONTRACT, BREACH OF WARRANTY, TORT, NEGLIGENCE, INFRINGEMENT OR OTHERWISE (INCLUDING, WITHOUT LIMITATION, DAMAGES BASED ON LOSS OF PROFITS, DATA, FILES, USE, BUSINESS OPPORTUNITY OR CLAIMS OF THIRD PARTIES), AND WHETHER OR NOT THE PARTY HAS BEEN ADVISED OF THE POSSIBILITY OF SUCH DAMAGES. THIS LIMITATION SHALL APPLY NOTWITHSTANDING ANY FAILURE OF ESSENTIAL PURPOSE OF ANY LIMITED REMEDY PROVIDED HEREIN.

- Should any provision of this Agreement be held by a court of competent jurisdiction to be illegal, invalid, or unenforceable, that provision shall be deemed amended to achieve as nearly as possible the same economic effect as the original provision, and the legality, validity and enforceability of the remaining provisions of this Agreement shall not be affected or impaired thereby.

- The failure of either party to enforce any term or condition of this Agreement shall not constitute a waiver of either party's right to enforce each and every term and condition of this Agreement. No breach under this agreement shall be deemed waived or excused by either party unless such waiver or consent is in writing signed by the party granting such waiver or consent. The waiver by or consent of a party to a breach of any provision of this Agreement shall not operate or be construed as a waiver of or consent to any other or subsequent breach by such other party.

- This Agreement may not be assigned (including by operation of law or
otherwise) by you without WILEY's prior written consent.

- Any fee required for this permission shall be non-refundable after thirty (30) days from receipt by the CCC.

- These terms and conditions together with CCC's Billing and Payment terms and conditions (which are incorporated herein) form the entire agreement between you and WILEY concerning this licensing transaction and (in the absence of fraud) supersedes all prior agreements and representations of the parties, oral or written. This Agreement may not be amended except in writing signed by both parties. This Agreement shall be binding upon and inure to the benefit of the parties successors, legal representatives, and authorized assigns.

- In the event of any conflict between your obligations established by these terms and conditions and those established by CCC's Billing and Payment terms and conditions, these terms and conditions shall prevail.

- WILEY expressly reserves all rights not specifically granted in the combination of (i) the license details provided by you and accepted in the course of this licensing transaction, (ii) these terms and conditions and (iii) CCC's Billing and Payment terms and conditions.

- This Agreement will be void if the Type of Use, Format, Circulation, or Requestor Type was misrepresented during the licensing process.

- This Agreement shall be governed by and construed in accordance with the laws of the State of New York, USA, without regards to such state's conflict of law rules. Any legal action, suit or proceeding arising out of or relating to these Terms and Conditions or the breach thereof shall be instituted in a court of competent jurisdiction in New York County in the State of New York in the United States of America and each party hereby consents and submits to the personal jurisdiction of such court, waives any objection to venue in such court and consents to service of process by registered or certified mail, return receipt requested, at the last known address of such party.

**WILEY OPEN ACCESS TERMS AND CONDITIONS**

Wiley Publishes Open Access Articles in fully Open Access Journals and in Subscription journals offering Online Open. Although most of the fully Open Access journals publish open access articles under the terms of the Creative Commons Attribution (CC BY) License only, the subscription journals and a few of the Open Access Journals offer a choice of Creative Commons Licenses. The license type is clearly identified on the article.

**The Creative Commons Attribution License**
The Creative Commons Attribution License (CC-BY) allows users to copy, distribute and transmit an article, adapt the article and make commercial use of the article. The CC-BY license permits commercial and non-

**Creative Commons Attribution Non-Commercial License**

The Creative Commons Attribution Non-Commercial (CC-BY-NC) License permits use, distribution and reproduction in any medium, provided the original work is properly cited and is not used for commercial purposes. (see below)

**Creative Commons Attribution-Non-Commercial-NoDerivs License**

The Creative Commons Attribution Non-Commercial-NoDerivs License (CC-BY-NC-ND) permits use, distribution and reproduction in any medium, provided the original work is properly cited, is not used for commercial purposes and no modifications or adaptations are made. (see below)

**Use by commercial "for-profit" organizations**

Use of Wiley Open Access articles for commercial, promotional, or marketing purposes requires further explicit permission from Wiley and will be subject to a fee. Further details can be found on Wiley Online Library [http://olabout.wiley.com/WileyCDA/Section/id-410895.html](http://olabout.wiley.com/WileyCDA/Section/id-410895.html)

**Other Terms and Conditions:**

v1.10 Last updated September 2015

Questions? [customercare@copyright.com](mailto:customercare@copyright.com) or +1-855-239-3415 (toll free in the US) or +1-978-646-2777.
Appendix C: Copyright permission for use of material provided by American Roentgen Ray Society

<table>
<thead>
<tr>
<th>American Roentgen Ray Society</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gillian Corbo</td>
</tr>
<tr>
<td>University of Western Ontario</td>
</tr>
<tr>
<td>301-1265 Richmond St</td>
</tr>
<tr>
<td>London ON N6A 3M1</td>
</tr>
<tr>
<td>04/07/16</td>
</tr>
<tr>
<td>Invoice No: C    09017</td>
</tr>
<tr>
<td>Canada</td>
</tr>
</tbody>
</table>

Thank you for your request for permission to reproduce the following material from the *American Journal of Roentgenology*.

<table>
<thead>
<tr>
<th>AJR</th>
<th>Brody et al.</th>
<th>The Meniscal Roots: Gross Anatomic Correlation with 3-T</th>
<th>4</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td>AJR</td>
<td>MRI Findings Fig 1ABCD</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Order Total $0.00

Permission is effective when signed below by ARRS authorized representative. Payment if applicable, may be made by check or credit card. Tax is not included in the price, if your country requires a tax, you will have to calculate the tax separately. Permission will not be granted until invoice is paid in full. Please return this form with payment.

The following conditions apply:

1. Use of the following credit line: Reprinted with permission from the *American Journal of Roentgenology*.
2. One-time, non-exclusive use only to include on-line versions and/or CD-ROMS. This permission does not include revisions or future editions.
3. Translations of copyrighted text is prohibited.

We regret that ARRS cannot supply original or digital material for reproduction.

ARRS authorized signature: ___________________________ Date 4/7/16

Payment Options

Check (made out to ARRS in U.S. funds drawn on a U.S. bank)

_____ Visa _____ American Express _____ Master Card

Card # ___________________________ Exp. Date ___________________________

Signature: ___________________________ Phone # ___________________________

Send remittance to: 44211 Slatestone Court Leesburg, VA 20176-5109
Or fax Credit Card # to 703-729-5913
Curriculum Vitae

Name: Gillian Gabriela Corbo

Post-secondary Education and Degrees:

University of British Columbia
Vancouver, British Columbia, Canada
2009-2014 B.Kin

The University of Western Ontario
London, Ontario, Canada
2014-2016 M.Sc. Clinical Anatomy

Honours and Awards:

Wayne Davis Anatomy Excellence Award
2015

NSERC CGS-M
2013 (Awarded & Declined); 2015

Ontario Graduate Scholarship
2015 (Awarded & Declined)

Western Graduate Research Scholarship
2014 – 2016

Dean’s Commendation for Academic Excellence
2013

Hugh M. Brock Education Abroad Scholarship
2013

Head of Class Award
2012

TREK Excellence Scholarship
2010; 2012

Kinesiology Faculty Scholarship
2012

William B. McNulty Kinesiology Scholarship
2012
Faces of Today Leadership Award
2011

Human Kinetics Faculty Scholarship
2010

President’s Entrance Scholarship
2009

**Related Work**

**Teaching Assistant**
The University of Western Ontario
2014-2016
- Gross Anatomy – 1st & 2nd Year Medicine
- Functional Human Anatomy - Occupational Therapy 9524
- Functional Human Anatomy – Physical Therapy 9501
- Systemic Human Anatomy 3319

Anatomy Outreach Instructor
The University of Western Ontario
2014-2016
- Anatomy & Radiology Contouring Bootcamp
- Clinical Anatomy Outreach Program

**Conference Presentations:**

Canadian Bone and Joint Conference 2016

Experimental Biology 2016

London Health Research Day 2016
Anatomy & Cell Biology Research Day

Publications:


Certifications and Professional Memberships:

Western Certificate in University Teaching and Learning
2015

American Association of Anatomists Graduate Student Member
2015-2016