CAN OSTEOARTHRITIS AFTER ACL RECONSTRUCTION BE EXPLAINED BY (ALTERING) GRAFT MECHANICAL PROPERTIES?

Janne Spierings (1), Marloes van den Hengel (1), Bert van Rietbergen (1), Keita Ito (1), Jasper Foolen (1)

1. *Orthopaedic Biomechanics, Department of Biomedical Engineering, Eindhoven University of Technology, Eindhoven, The Netherlands.*

Introduction

Each year, 1% of active individuals rupture their Anterior Cruciate Ligament (ACL) [1]. Torn ACLs are often reconstructed using tendon autografts. However, in approximately half of the patients, ACL reconstruction (ACLR) leads to osteoarthritis (OA) within 5 to 15 years [2]. We hypothesize that OA results from a mismatch in mechanical properties between the native ACL and the used tendon grafts. Moreover, due to *in vivo* graft remodeling, graft stiffness decreases and knee laxity increases. It remains unknown however whether these changes contribute to the development of OA. Therefore, this study aims to assess the influence of altering graft mechanical properties on knee kinematics and cartilage loading, as a measure for the risk of developing OA.

Methods

The stance phase of the gait cycle was simulated in 4 subject-specific open-source available finite element models in the FEBio software [3,4]. The material properties of the ACL were adjusted to match currently used grafts, e.g. patellar (PT) and hamstring tendons (HT), at surgery. In addition, hypothetical, but clinically relevant, grafts with a range in decreasing stiffness, increasing transition strain (knee laxity), and a combination of both were implemented to mimic grafts during and after *in vivo* graft remodeling. The effect of these grafts on knee range of motion (anterior tibial translation (ATT) and internal tibial rotation (IR)) and tibial cartilage contact pressure were determined at the point of maximum posterior force on the femur.

Results

Reconstructing the ACL with a PT or HT graft resulted in a decrease in ATT and IR, and a minor relocation of tibial cartilage contact pressure. Moreover, both ATT and IR increased with decreasing graft stiffness and/or increasing knee laxity in 3 out of the 4 patients (Figure 1). A clear relocation in tibial cartilage contact pressure was found with a decreasing graft stiffness and/or an increasing knee laxity in those 3 patients (Figure 2).

Discussion

Currently, ruptured ACLs are mainly reconstructed using PT or HT grafts. Although initially those grafts do not substantially influence knee range of motion and tibial cartilage pressure patterns, a decreasing graft stiffness and increasing knee laxity, as clinically observed during graft remodeling, do. The increase in ATT and IR, both movements the native ACL restricts, indicates changed knee joint mechanics. Together with a relocation of the tibial cartilage contact pressure, this suggests abnormal loading of the knee which can lead to OA [5]. Interestingly, one of the patients did not show altered knee translations or tibial cartilage pressure patterns, indicating that not every patient might be at risk for the development of OA, which is in line with clinical outcomes. Altogether, this model paves the way towards the development of a patient-specific prediction model for ACL-reconstruction-induced OA. Besides, these results suggest the need for novel grafts with mechanical properties that match the native ACL and/or grafts that circumvent *in vivo* graft remodeling.

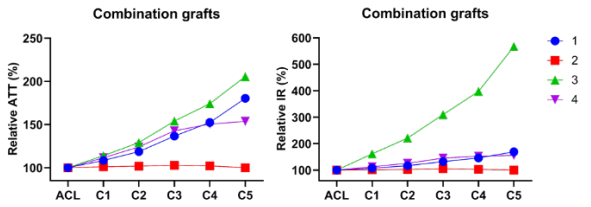

Figure 1: The ATT and IR are increased in grafts with a combination of decreased stiffness and increased knee laxity. With respect to the ACL, C1 has 59% stiffness and 132% laxity; C2 30% stiffness and 152% laxity; C3 19% stiffness and 163% laxity; C4 11% stiffness and 198% laxity; C5 7% stiffness and 290% laxity.

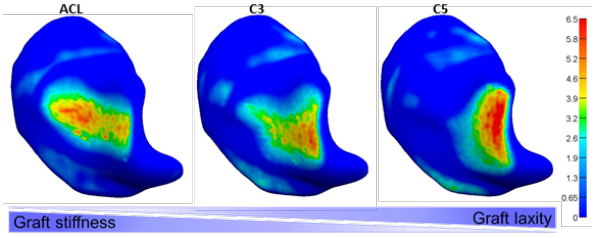

Figure 2: A relocation in tibial cartilage pressure is found with decreasing graft stiffness and increasing knee laxity.

References

1. Ruano JS et al, J Athl Train, 52(6):606-609, 2017.
2. Cheung CC et al, Curr Rev Musculoskelet Med, 13(1): 115–122, 2020.
3. Erdemir A, J Knee Surg, 29(2): 107–116, 2016.
4. Maas SA et al, J Biomech Eng, 134(1): 011005, 2012.
5. Stergiou N et al., Sports Med, 37(7): 601-13, 2007.

Funding

European Union's Horizon 2020 research and innovation programme [grant number 952981].