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Ligament Tissue Engineering through an Evolutionary Materials Science Methodology

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Introduction

The anterior cruciate ligament contributes to the stability of the knee. Unfortunately, it is the intra-articular ligament that is ripped the most commonly. The ACL has a limited capacity to heal because to poor vascularization and is frequently replaced after severe damage. The search for tissue-engineered ACL repair therapies has been sparked by the absence of suitable alternatives at this time. The scaffolds currently under study range from intricate three-dimensional braided poly-l-lactic acid fibre structures to twisted silk fibre designs. These tissue-engineered constructions aim to promote ligament tissue regeneration while retaining mechanical properties similar to native ligament by using methods such as porous scaffolds, cells, and growth factors [1].

Description

The most frequently injured ligament in the knee is the anterior cruciate ligament. The ACL is the main intra-articular ligament in the knee, and proper kinematics and stability depend on it. The regulates mobility and prevents abnormal motion by supporting the joint and linking the femur to the tibia [2]. It is primarily responsible for supporting and strengthening the knee and preventing excessive anterior femur translation, which can cause a dislocation and bone fracture in the knee. The ACL is a dense, well-organized, cable-like tissue composed of collagen, elastin, proteoglycans, water, and cells. Ligaments consist of collagen molecules, fibrils, fibril bundles, and fascicles that run parallel to the long axis of the tissue. The human ACL's average length is and its cross-sectional area is. The collagen fibrils that make up ligaments have a pattern called a crimp, which is a regular change in direction. In the ACL, this crimp pattern is used repeatedly. The fascicles contain elastin, proteoglycans, and collagen fibrils. The ligament is encircled by a vascularized epiligament sheath. An additional layer of structure is created when the collagenous network is twisted roughly 180 degrees from the femoral attachment site to the tibia attachment site [3].

Ligaments behave diphasically when subjected to tension. Low stress per unit strain, for instance, is a characteristic of the non-linear or toe area. The linear zone comes next and is known for having a higher stress-to-strain ratio. A slight decrease in stress per unit strain in the final section indicates the failure. This is the location of the yield and failure zone. This distinct behavior is caused by the components of the ligament and how they are arranged in the tissue. When force is first applied to the tissue, it transfers to the collagen fibrils. Interfibrillar slippage occurs due to crosslinking. The stress per unit of strain rises as a result. Finally, the ligament's collagen fibers collapse during defibrillation, reducing stress per unit strain and leading to tissue failure. The three stages of ligament healing following an injury are inflammation, cellular proliferation, matrix repair, and remodelling [4]. At this point, serous fluid accumulates in the ligament and surrounding tissues, making the injured area Leukocytes, monocytes, and macrophages are attracted to the wound. During the stage of cellular proliferation and matrix repair, fibroblasts are present, and vascular granulation tissue is produced. A new extracellular matrix is created by synthesizing collagen with a high ratio of collagen. The final stage, which takes several months, is remodeling. At this point, the new extracellular matrix transforms into a hypercellular tissue that is slightly disorganized. The ACL cannot heal because it lacks vascularization. The best healing results when collagen fibers are maintained in a continuous state. Due to a change in the crimp pattern between the new and old extracellular matrix and the loss of tissue structure, the mechanical properties of the new scar tissue and the original ligament differ. When an accident causes a complete ligament midsubstance rupture or detachment, insertion point surgery is required [5]. When the joint is loaded, the articular cartilage gets too much tension, which can cause early osteoarthritis if the ligament doesn't heal.

Conclusion

ACL injuries have traditionally been treated with biological grafts, autografts, and allografts. The patellar, hamstring, or quadriceps tendons of the patient are frequently used as sources of ACL autograft material. Autografts from the hamstring and patella tendons are the choice of the majority of surgeons. Typically, a piece of bone is used to remove the patellar tendon graft material from the patella and the tibia where it is inserted. The "bone–patellar–bone" graft is fed after that. Tissue-engineered alternatives for ACL restoration are becoming increasingly popular due to the limitations of both biological and synthetic grafts. The ideal ACL replacement scaffold ought to be porous, free of inflammatory agents, and biodegradable. It ought to have sufficient mechanical strength, behave mechanically in a manner that is comparable to that of an original ACL, and encourage the growth of ligamentous tissue. The scaffold ought to deteriorate at a rate that makes it possible to add new materials. To overcome the limitations of current treatments, novel ligament repair techniques are required as the number of ACL injuries rises. Tissue engineering-based scaffolds are becoming a viable option for ACL repair. For load-bearing tissue, these scaffolds can be adjusted to provide just the right amount of mechanical support. It has also been demonstrated that tissue-engineered scaffolds enhance cell adhesion, proliferation, and proliferation.

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Conflict of Interest

None

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