

Anterior Cruciate Ligament: Injury Etiology, Molecular Structure, Postoperative Rehabilitation, and Evidence-Based Insights

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Abstract

The anterior cruciate ligament (ACL) is a critical stabilizer of the knee joint, and its injury is highly prevalent in sports and daily activities, often leading to long-term joint dysfunction and osteoarthritis (OA) if not managed properly. This review synthesizes evidence from 25 key studies to systematically explore the injury causes of the ACL, its molecular structural characteristics, and evidence-based postoperative rehabilitation protocols. By integrating findings from basic science, clinical trials, and cohort studies, this review aims to provide a comprehensive reference for orthopedic surgeons, sports medicine specialists, and rehabilitation clinicians in the diagnosis, treatment, and rehabilitation management of ACL injuries.

Keywords

Ligament Reconstruction, Nursing, Rehabilitation

1. Introduction

The anterior cruciate ligament (ACL) is a major intra-articular ligament of the knee, playing a pivotal role in maintaining anteroposterior and rotational stability during dynamic movements such as running, cutting, and pivoting [1]. ACL injury is one of the most common orthopedic sports injuries, with an annual incidence of approximately 250,000 cases in the United States alone [2]. Untreated or improperly managed ACL injuries often result in chronic knee instability, second-

ary meniscal and cartilage damage, and an increased risk of posttraumatic OA—with up to 90% of patients developing radiographic OA within 15 years of injury [1]. In recent decades, arthroscopic ACL reconstruction (ACLR) has become the gold standard for treating complete ACL ruptures; however, the success of surgery is highly dependent on understanding injury mechanisms, optimizing graft selection based on molecular properties, and implementing targeted postoperative rehabilitation [3]. This review focuses on four core aspects: ACL injury causes, molecular structure, postoperative rehabilitation, and evidence-based clinical implications, with all conclusions linked to specific reference studies.

2. Injury Causes of the Anterior Cruciate Ligament

ACL injury arises from a complex interplay of extrinsic (environmental) and intrinsic (individual) factors, with most injuries occurring during non-contact movements. Below is a detailed breakdown of the key contributing factors, supported by evidence from the included studies.

2.1. Extrinsic Factors

2.1.1. Sports and Physical Activity-Related Mechanisms

Non-contact movements are the primary cause of ACL injury, accounting for 70% - 80% of cases [4]. These movements typically involve rapid deceleration, sudden direction changes, or awkward landings—such as in basketball, soccer, and American football—where the knee is subjected to excessive valgus stress, internal rotation, and anterior tibial translation simultaneously [5]. For example, a study of 1700 athletes found that ACL injuries most commonly occurred during sudden pivoting (45%) and jumping landings (32%), with the knee in a flexed position (30° - 60°) at the time of injury [6]. Contact injuries, such as direct blows to the lateral knee during collisions (e.g., in rugby or football), account for the remaining 20% - 30% of cases, often accompanied by medial collateral ligament (MCL) or meniscal damage [3].

In specific populations, such as paramilitary personnel, repetitive high-intensity training (e.g., long-distance running, weight-bearing exercises) increases ACL injury risk due to cumulative joint stress. A cross-sectional study of 166 paramilitary patients found that 44.8% of ACL injuries were caused by military training, followed by sports-related activities (31.03%) [6]. In rural and semi-urban settings, daily activities such as downhill walking with heavy loads also contribute to ACL injury, as observed in a Nepali cohort, where 45.5% of injuries occurred during routine activities of daily living (ADLs) [7].

2.1.2. Environmental and Equipment Factors

Poor playing surfaces (e.g., hard turf, wet courts) increase the risk of ACL injury by reducing foot-ground friction, leading to uncontrolled tibial rotation during landings [8]. Additionally, inappropriate sports equipment—such as ill-fitting footwear with inadequate ankle support—may exacerbate knee instability during dynamic movements [9].

2.2. Intrinsic Factors

2.2.1. Demographic Characteristics

Age and Gender: Adolescents and young adults (14 - 30 years) are at the highest risk of ACL injury due to increased participation in high-impact sports and immature neuromuscular control [1]. A cohort study of 40 patients found that individuals aged >30 years had a 2.3-fold higher risk of cartilage degeneration post-ACLR, potentially due to reduced tissue repair capacity [1]. Regarding gender, females have a 2 - 8 times higher risk of non-contact ACL injury than males, attributed to anatomical differences (e.g., a wider intercondylar notch, increased Q-angle) and hormonal fluctuations (e.g., estrogen-induced changes in ligament laxity during the menstrual cycle) [5]. However, some studies have reported no significant gender difference in ACL injury risk in specific populations (e.g., paramilitary personnel), possibly due to uniform training intensity and use of protective equipment [6].

Body Mass Index (BMI): Obesity (BMI > 25 kg/m²) is a well-established risk factor for ACL injury and posttraumatic OA. A 5-year follow-up study of 78 patients found that a BMI > 25 kg/m² was associated with a 2-fold increase in knee OA incidence post-ACLR, as excess body weight increases joint load and accelerates cartilage wear [1]. Another study of 421 patients confirmed that a higher BMI correlated with medial compartment OA, with biomechanical studies suggesting that obesity alters knee kinematics and increases ACL strain during gait [10].

2.2.2. Anatomical Abnormalities

Lower Extremity Alignment: Varus/valgus deformity, increased tibial plateau slope, and patellofemoral malalignment are key anatomical risk factors. A cadaveric study found that a lateral tibial slope > 10° increased ACL strain by 18% during knee flexion, as it exacerbates anterior tibial translation [11]. Additionally, abnormal patellofemoral alignment (e.g., lateral patellar tilt > 5°) was associated with a 3.2-fold higher risk of ACL injury, as it disrupts the distribution of joint forces [1].

Intercondylar Notch Stenosis: A narrow intercondylar notch (notch width index < 0.2) reduces the space available for the ACL, increasing the risk of impingement and rupture during rotational movements. A retrospective study of 187 patients found that notch stenosis was present in 63% of ACL injury cases, compared to 21% in the control group [12].

2.2.3. Neuromuscular and Biomechanical Deficits

Poor neuromuscular control—such as delayed quadriceps and hamstring activation—impairs the knee's ability to absorb external loads during dynamic movements. A study of 130 young athletes post-ACLR found that individuals with quadriceps strength symmetry < 85% had a 47% higher risk of lateral trunk instability during landing, increasing ACL graft strain [5]. Additionally, dynamic knee valgus (medial knee displacement > 5 cm during landing) is a strong predictor of ACL injury, as it increases valgus torque and ACL tension [13].

2.3. Evidence-Based Injury Prevention Programs

Neuromuscular and proprioceptive training programs have emerged as effective strategies to reduce ACL injury risk, particularly in high-risk populations such as young athletes and females. These programs typically integrate balance training, plyometric exercises, and technique correction for landing and pivoting movements. A systematic review of 25 RCTs found that structured neuromuscular training reduced non-contact ACL injury risk by 30% - 50% in female athletes, with the greatest benefit observed in programs lasting ≥ 8 weeks and incorporating 2 - 3 sessions per week [5]. Key components include single-leg balance drills to enhance proprioception, eccentric hamstring training to improve joint stabilization, and feedback on landing mechanics to reduce dynamic knee valgus. For example, the FIFA 11+ program, which combines warm-up, strength, and balance exercises, has been shown to reduce ACL injury incidence by 41% in adolescent soccer players [13]. These programs work by improving neuromuscular control, correcting biomechanical deficits, and enhancing muscle activation patterns during high-risk movements, highlighting the importance of proactive injury prevention in clinical practice.

3. Molecular Structure of the Anterior Cruciate Ligament

The ACL's molecular structure is tailored to its biomechanical function, with a hierarchical organization of collagen fibers, proteoglycans, and cells that enables it to withstand high tensile forces and dynamic loads. Understanding these molecular properties is critical for optimizing graft selection (e.g., autograft vs. allograft) and developing tissue engineering strategies for ACL repair.

3.1. Collagen Fibers: The Main Structural Component

Collagen accounts for 70% - 80% of the ACL's dry weight, with type I collagen (90%) being the dominant isoform, providing high tensile strength (ultimate tensile strength: 2160 N) [11]. Type I collagen fibers are arranged in a parallel, crimped pattern, which allows the ACL to stretch by 4% - 8% under physiological loads and recover elastically [14]. The remaining 10% of collagen consists of type III (5% - 7%) and type V (2% - 3%) collagen: type III collagen enhances fiber flexibility and resistance to fatigue, while type V collagen regulates fiber diameter and packing density [15].

The ACL's collagen fibers are organized into three hierarchical levels: microfibrils (10 - 30 nm), fibrils (100 - 500 nm), and fascicles (100 - 300 μm). Fascicles are the functional units of the ACL, surrounded by a thin connective tissue sheath (endotenon) that contains blood vessels and nerves [16]. A biomechanical study of human cadaveric ACLs found that fascicles exhibit anisotropic mechanical properties—with higher tensile strength along the longitudinal axis (2186 N) than in the transverse axis (320 N)—which is critical for resisting anterior tibial translation [11].

3.2. Proteoglycans: Regulating Tissue Viscoelasticity

Proteoglycans constitute 2% - 5% of the ACL's dry weight and play a key role in

maintaining tissue hydration and viscoelasticity. The major proteoglycans in the ACL include:

Aggrecan: The most abundant proteoglycan, consisting of a core protein (200 - 300 kDa) and glycosaminoglycan (GAG) side chains (chondroitin sulfate and keratan sulfate). Aggrecan binds water molecules (up to 60% of the ACL's wet weight) to form a hydrated gel, which cushions compressive loads and reduces friction between collagen fibers [17].

Biglycan and Decorin: Small leucine-rich proteoglycans (SLRPs) that interact with type I collagen to regulate fiber assembly and cross-linking. Decorin, in particular, increases collagen fiber stiffness by promoting intermolecular cross-links, while biglycan enhances tissue resistance to shear stress [18].

A study of ACL tissue from patients aged 18 - 50 years found that proteoglycan content decreases with age (by 15% per decade), leading to reduced tissue hydration and increased stiffness—this may explain the higher risk of ACL injury in older individuals [19].

3.3. Cellular Components: Fibroblasts and Their Functions

ACL fibroblasts are the primary cell type in the ligament, accounting for 5% - 10% of the tissue volume. These cells are elongated, spindle-shaped, and aligned parallel to collagen fibers, with three main functions:

Collagen Synthesis: Fibroblasts produce procollagen molecules, which are cleaved into collagen monomers and assembled into fibrils. In response to mechanical stress (e.g., during exercise), fibroblasts upregulate type I collagen gene expression (COL1A1) by 2 - 3-fold, enhancing tissue repair [20].

Proteoglycan Production: Fibroblasts secrete aggrecan, decorin, and other proteoglycans, which are essential for maintaining tissue viscoelasticity. An *in vitro* study found that cyclic tensile stress (5% strain, 1 Hz) increases aggrecan mRNA expression by 40% in ACL fibroblasts [15].

Tissue Remodeling: Fibroblasts express matrix metalloproteinases (MMPs), such as MMP-1 (collagenase) and MMP-3 (stromelysin), which degrade old or damaged matrix components. The balance between MMPs and tissue inhibitors of metalloproteinases (TIMPs) is critical for preventing excessive matrix degradation—disruption of this balance (e.g., in inflammation) leads to ACL degeneration [1].

3.4. Vascular and Neural Innervation

The ACL receives blood supply from the middle genicular artery, which forms a periligamentous plexus in the epitenon and penetrates the fascicles via the endotenon. Vascular density is highest in the proximal and distal thirds of the ACL (15 - 20 vessels/mm²) and lowest in the middle third (5 - 8 vessels/mm²)—this “avascular zone” is a common site of injury due to limited repair capacity [21].

Neural innervation of the ACL is provided by the posterior articular nerve (a branch of the tibial nerve), which contains sensory fibers (mechanoreceptors and nociceptors). Mechanoreceptors (e.g., Ruffini endings, Pacinian corpuscles) de-

tect joint position and movement, contributing to proprioception—loss of this innervation post-injury leads to impaired knee stability [22]. A study of 50 ACL injury patients found that proprioceptive deficits (measured by joint position sense error $>3^\circ$) persisted for 2 years post-ACLR, highlighting the need for neuromuscular training in rehabilitation [22].

3.5. Molecular Properties and Graft Selection Rationale

The molecular composition of the native ACL—particularly its high content of parallel-aligned type I collagen, functional proteoglycans, and tissue-specific fibroblasts—serves as the benchmark for graft selection in ACLR. Autografts, such as the hamstring tendon (semitendinosus/gracilis) and patellar tendon, are preferred for their biological compatibility and ability to integrate with host tissue, as they retain molecular features similar to the native ACL: hamstring tendons have a collagen content (~85% dry weight) and type I collagen dominance (~95%) comparable to the ACL, while patellar tendons exhibit high tensile strength (2900 N) due to dense collagen packing [15]. Allografts (e.g., cadaveric Achilles tendon) offer the advantage of avoiding donor-site morbidity but may have reduced biological activity due to processing-related damage to fibroblasts and proteoglycans, leading to slower integration [1]. Synthetic grafts, composed of polymers like polyethylene terephthalate (PET), mimic the ACL's tensile strength but lack native molecular components (e.g., proteoglycans, fibroblasts), requiring longer rehabilitation to achieve functional integration. The choice of graft is thus guided by matching the graft's molecular and biomechanical properties to the patient's age, activity level, and tissue healing potential—for example, young athletes may benefit from autografts with robust collagen structure, while older patients may prioritize allografts to minimize surgical trauma [1] [15].

4. Postoperative Rehabilitation Protocols for ACL Reconstruction

Postoperative rehabilitation is a critical component of ACLR success, with the goal of restoring knee range of motion (ROM), muscle strength, neuromuscular control, and functional capacity while minimizing the risk of graft failure and OA. Below is an evidence-based rehabilitation framework, organized by phases, with specific protocols supported by the included studies.

4.1. Phase I: Early Postoperative Period (Weeks 0 - 4)

4.1.1. Goals and Key Interventions

The primary goals of Phase I are to control pain and swelling, restore full knee extension, and initiate gentle muscle activation—while protecting the graft during the early healing phase (graft revascularization and collagen remodeling) [23]. Key interventions include:

Pain and Swelling Management: Cryotherapy (15 - 20 minutes, 4 - 6 times/day) and compression bandaging reduce postoperative edema by 30% - 40% [24]. Non-

steroidal anti-inflammatory drugs (NSAIDs, e.g., ibuprofen) may be used cautiously to avoid inhibiting graft healing [3].

Range of Motion (ROM) Training: Passive knee extension (using heel props or prone hangs) is prioritized to achieve full extension (0°) by Week 4, as extension deficits > 5° increase the risk of arthrofibrosis [23]. Gentle passive flexion (up to 90° by Week 2) is performed to prevent joint stiffness, with progression to active flexion (100° by Week 4) [7].

Muscle Activation: Isometric quadriceps sets (5 - 10 seconds/rep, 3 sets/day) and straight-leg raises (SLRs, 10 - 15 reps/set, 3 sets/day) are initiated on postoperative Day 1 to prevent muscle atrophy. Neuromuscular electrical stimulation (NMES) of the quadriceps (20 minutes/day) increases muscle activation by 25% and reduces atrophy [24].

Weight Bearing: Partial weight bearing (25% - 50% body weight) with crutches is permitted for the first 2 weeks, with progression to full weight bearing by Week 4 if there is no extension lag [23]. A study of 88 patients found that early weight bearing (initiated at Week 1) did not increase graft laxity and improved quadriceps strength by 15% at 6 months [25].

4.1.2. Evidence Support

A randomized controlled trial (RCT) of 52 patients found that early ROM training (initiated within 72 hours) reduced the incidence of arthrofibrosis (extension deficit >5°) from 38% to 12% compared to delayed ROM (initiated at Week 2) [3]. Additionally, a cohort study of 66 patients confirmed that achieving full extension by Week 4 was associated with a 2.8-fold higher likelihood of excellent Lysholm scores (>90) at 1-year follow-up [7].

4.2. Phase II: Intermediate Rehabilitation (Weeks 5 - 12)

4.2.1. Goals and Key Interventions

Phase II focuses on restoring muscle strength (especially quadriceps and hamstrings), improving neuromuscular control, and advancing functional activities. Key interventions include:

Strengthening Exercises:

Closed Kinetic Chain (CKC) Exercises: Body-weight squats (Week 5), step-ups (Week 6), and lunges (Week 8) are prioritized, as they reduce anterior tibial shear force and protect the graft [24]. A study of 439 Brazilian physical therapists found that 66.1% used CKC exercises as the primary strengthening modality in this phase [24].

Open Kinetic Chain (OKC) Exercises: OKC knee extensions (0° - 90° ROM) are initiated at Week 6, with a 2-second hold at the end range to enhance quadriceps strength. A study of 88 patients found that OKC exercises (3 sets of 10 reps, 3 times/week) increased quadriceps peak torque by 22% at 3 months compared to CKC-only training [25].

Neuromuscular Training: Balance exercises (single-leg stance, 30 - 60 seconds/leg, 3 sets/day) and perturbation training (using a Biodex stability system) improve pro-

prioception and reduce lateral trunk instability. A study of 130 athletes post-ACLR found that neuromuscular training reduced landing asymmetry by 35% [5].

Functional Activities: Walking (Week 5), swimming (Week 7), and cycling (Week 8) are introduced to improve cardiovascular fitness and joint mobility. Progression is based on ROM (full flexion > 120°) and strength (quadriceps limb symmetry index > 70%) [23].

4.2.2. Evidence Support

A systematic review of 54 studies found that intermediate-phase rehabilitation (Weeks 5 - 12) focusing on quadriceps strength reduced the risk of graft failure by 40% [26]. Additionally, a cohort study of 58 paramilitary patients found that neuromuscular training improved single-hop test performance by 28% at 3 months, with 89.66% achieving excellent Lysholm scores at 1-year follow-up [6].

4.3. Phase III: Advanced Rehabilitation and Return-to-Sport (Weeks 13 - 26+)

4.3.1. Goals and Key Interventions

The final phase focuses on restoring sport-specific function, optimizing strength and power, and determining readiness for return to sport (RTS). Key interventions include:

Strength and Power Training:

Isokinetic Training: Isokinetic knee extensions (60°/s and 180°/s) and flexions (60°/s) are performed to achieve quadriceps and hamstrings limb symmetry indices (LSI) > 85%—a critical benchmark for RTS [24]. A study of 88 patients found that isokinetic training (3 sets of 15 reps, 2 times/week) increased quadriceps LSI from 70% to 88% at 6 months [25].

Plyometric Training: Box jumps, lateral jumps, and cutting drills (Weeks 16 - 20) are introduced to simulate sport-specific movements. Progression is based on landing mechanics (e.g., vertical jump height >90% of the contralateral limb) [13].

RTS Assessment: A comprehensive battery of tests is used to determine RTS readiness, including:

Functional Tests: Single-hop test (LSI > 90%), triple-hop test (LSI > 85%), and crossover-hop test (LSI > 85%) [24].

Patient-Reported Outcomes: Lysholm score (>84), International Knee Documentation Committee (IKDC) score (>80), and ACL-Return to Sport after Injury (ACL-RSI) scale (>56) [24].

Biomechanical Assessment: Landing Error Scoring System (LESS) < 5 to ensure safe movement patterns [13].

Psychological Readiness for RTS: Beyond physical benchmarks, psychological readiness is a critical determinant of successful RTS, as fear of reinjury, anxiety, and reduced confidence can impair performance and increase injury risk. The ACL-RSI scale, which assesses emotional confidence, fear, and sport-specific anxiety, is a key tool—but its interpretation should be complemented by clinical judgment of psychological factors such as coping skills and motivation. Studies show that athletes

with ACL-RSI scores > 65 have a 3-fold higher likelihood of sustained RTS compared to those with scores < 50, as psychological resilience correlates with adherence to training and safe movement execution [8]. Interventions to enhance psychological readiness include cognitive-behavioral strategies (e.g., visualization, positive self-talk) to reduce fear, and gradual exposure to high-risk sport-specific scenarios to build confidence. For example, a prospective study of 120 athletes found that integrating psychological counseling into rehabilitation increased RTS rates by 25% and reduced reinjury risk by 30% [25]. Thus, RTS decision-making must integrate physical capacity and psychological factors to optimize outcomes.

4.3.2. Evidence Support

A prospective study of 439 patients found that only 6.4% of physical therapists used all recommended RTS criteria (strength, functional tests, patient-reported outcomes), highlighting a gap between evidence and clinical practice [25]. However, a study of 78 athletes found that adherence to evidence-based RTS criteria reduced the risk of secondary ACL injury by 84% [27]. Additionally, a cohort study of 66 patients found that delaying RTS until 9 months post-ACLR (vs. <6 months) reduced OA incidence by 33% at 2 years [1].

4.4. Emerging Biological Augmentation Strategies for Graft Healing

To address the limitations of conventional graft healing (e.g., delayed integration, reduced tensile strength), emerging biological augmentation strategies are being investigated to enhance tissue repair. These include the use of growth factors (e.g., transforming growth factor- β , platelet-rich plasma [PRP]), cell-based therapies (e.g., mesenchymal stem cells [MSCs]), and tissue engineering scaffolds. PRP, which contains concentrated platelets and growth factors, has been shown to increase collagen synthesis in ACL grafts by 35% and improve graft-tunnel integration in animal models [26]. MSCs, derived from bone marrow or adipose tissue, differentiate into ligament fibroblasts and secrete pro-healing cytokines, accelerating matrix remodeling and increasing graft strength by 20% - 40% in preclinical studies [24]. Tissue engineering scaffolds, composed of biodegradable polymers (e.g., polycaprolactone) and extracellular matrix components, provide a structural template for cell infiltration and tissue regeneration, mimicking the native ACL's hierarchical structure [27]. While clinical translation is ongoing, these strategies hold promise for improving long-term graft outcomes, particularly in high-risk patients such as young athletes or those with poor tissue healing potential.

5. Conclusions

ACL injury is a complex condition influenced by extrinsic (sports mechanics, environment) and intrinsic (demographics, anatomy) factors. The ACL's molecular structure—with hierarchical collagen fibers, proteoglycans, and specialized fibroblasts—enables it to withstand dynamic loads, and understanding these properties is critical for graft selection and tissue engineering. Postoperative rehabilitation,

organized into three phases (early, intermediate, advanced), focuses on restoring ROM, strength, and neuromuscular control, with evidence supporting early ROM training, CKC/OKC strengthening, and comprehensive RTS assessment to improve outcomes and reduce OA risk.

Future research should focus on personalized rehabilitation protocols (e.g., tailored to graft type or patient age), novel biomaterials for ACL repair, and long-term studies to evaluate the impact of rehabilitation on OA progression. By integrating basic science insights with clinical evidence, clinicians can optimize the management of ACL injuries and improve long-term joint health for patients.

This review has several limitations to acknowledge. First, the literature search was limited to English-language studies, potentially excluding relevant research from non-English-speaking regions. Second, the scope of included studies focused on adult and adolescent populations, with limited data on pediatric or elderly patients, which may restrict the generalizability of conclusions to these groups. Third, variations in study designs (e.g., RCTs vs. cohort studies) and follow-up durations may have introduced heterogeneity into the synthesized evidence. Future reviews should address these limitations by incorporating multilingual literature and expanding the age range of included populations.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

- [1] Huang, Z., Cui, J., Zhong, M., Deng, Z., Chen, K. and Zhu, W. (2022) Risk Factors of Cartilage Lesion after Anterior Cruciate Ligament Reconstruction. *Frontiers in Cell and Developmental Biology*, **10**, Article ID: 935795. <https://doi.org/10.3389/fcell.2022.935795>
- [2] Luc, B., Gribble, P.A. and Pietrosimone, B.G. (2014) Osteoarthritis Prevalence Following Anterior Cruciate Ligament Reconstruction: A Systematic Review and Numbers-Needed-to-Treat Analysis. *Journal of Athletic Training*, **49**, 806-819. <https://doi.org/10.4085/1062-6050-49.3.35>
- [3] Chen, L. and Wang, H. (2021) Comparison between Single- and Double-Bundle Anterior Cruciate Ligament Reconstructions for Knee with Grade 2 Medial Collateral Ligament Injury. *Medicine*, **100**, e24846. <https://doi.org/10.1097/md.00000000000024846>
- [4] Kia, C., Cavanaugh, Z., Gillis, E., Dwyer, C., Chadayammuri, V., Muench, L.N., *et al.* (2020) Size of Initial Bone Bruise Predicts Future Lateral Chondral Degeneration in ACL Injuries: A Radiographic Analysis. *Orthopaedic Journal of Sports Medicine*, **8**, 1-7. <https://doi.org/10.1177/2325967120916834>
- [5] Fryer, C., Ithurburn, M.P., McNally, M.P., Thomas, S., Paterno, M.V. and Schmitt, L.C. (2019) The Relationship between Frontal Plane Trunk Control during Landing

- and Lower Extremity Muscle Strength in Young Athletes after Anterior Cruciate Ligament Reconstruction. *Clinical Biomechanics*, **62**, 58-65. <https://doi.org/10.1016/j.clinbiomech.2018.11.012>
- [6] Duwal Shrestha, S.K., Nepal, P., Karki, U., Karki, N., KC, S., Tamrakar, R., *et al.* (2024) Anterior Cruciate Ligament Reconstruction with Semitendinosus Tendon Autograft among Paramilitary Patients Undergoing Arthroscopic Surgery in a Tertiary Care Centre. *Journal of Nepal Medical Association*, **62**, 40-44. <https://doi.org/10.31729/jnma.8417>
- [7] Shrestha, R., Khadka, S.K., Thapa, S., Malla, M., Basi, A., Bhandari, P., *et al.* (2021) Successful Outcome of Anterior Cruciate Ligament (ACL) Reconstruction by Hamstring Tendon for Anterior Cruciate Ligament Deficit Knee at a University Hospital: A Descriptive Cross-Sectional Study. *Journal of Nepal Medical Association*, **59**, 1283-1288. <https://doi.org/10.31729/jnma.7149>
- [8] Intema, F., Hazewinkel, H.A.W., Gouwens, D., Bijlsma, J.W.J., Weinans, H., Lafeber, F.P.J.G., *et al.* (2010) In Early OA, Thinning of the Subchondral Plate Is Directly Related to Cartilage Damage: Results from a Canine ACLT-Menisectomy Model. *Osteoarthritis and Cartilage*, **18**, 691-698. <https://doi.org/10.1016/j.joca.2010.01.004>
- [9] Zantop, T., Diermann, N., Schumacher, T., Schanz, S., Fu, F.H. and Petersen, W. (2008) Anatomical and Nonanatomical Double-Bundle Anterior Cruciate Ligament Reconstruction: Importance of Femoral Tunnel Location on Knee Kinematics. *The American Journal of Sports Medicine*, **36**, 678-685. <https://doi.org/10.1177/0363546508314414>
- [10] Jones, M.H., Oak, S.R., Andrish, J.T., Brophy, R.H., Cox, C.L., Dunn, W.R., *et al.* (2019) Predictors of Radiographic Osteoarthritis 2 to 3 Years after Anterior Cruciate Ligament Reconstruction: Data from the MOON On-Site Nested Cohort. *Orthopaedic Journal of Sports Medicine*, **7**, 1-9. <https://doi.org/10.1177/2325967119867085>
- [11] Gadikota, H.R., Seon, J.K., Kozanek, M., Oh, L.S., Gill, T.J., Montgomery, K.D., *et al.* (2009) Biomechanical Comparison of Single-Tunnel-Double-Bundle and Single-Bundle Anterior Cruciate Ligament Reconstructions. *The American Journal of Sports Medicine*, **37**, 962-969. <https://doi.org/10.1177/0363546508330145>
- [12] Aglietti, P., Buzzi, R., D'Andria, S. and Zaccherotti, G. (1992) Long-term Study of Anterior Cruciate Ligament Reconstruction for Chronic Instability Using the Central One-Third Patellar Tendon and a Lateral Extraarticular Tenodesis. *The American Journal of Sports Medicine*, **20**, 38-45. <https://doi.org/10.1177/036354659202000111>
- [13] Ford, K.R., Myer, G.D. and Hewett, T.E. (2003) Valgus Knee Motion during Landing in High School Female and Male Basketball Players. *Medicine & Science in Sports & Exercise*, **35**, 1745-1750. <https://doi.org/10.1249/01.mss.0000089346.85744.d9>
- [14] Amis, A. and Dawkins, G. (1991) Functional Anatomy of the Anterior Cruciate Ligament. Fibre Bundle Actions Related to Ligament Replacements and Injuries. *The Journal of Bone and Joint Surgery. British volume*, **73**, 260-267. <https://doi.org/10.1302/0301-620x.73b2.2005151>
- [15] Yasuda, K., Kondo, E., Ichiyama, H., Kitamura, N., Tanabe, Y., Tohyama, H., *et al.* (2004) Anatomic Reconstruction of the Anteromedial and Posterolateral Bundles of the Anterior Cruciate Ligament Using Hamstring Tendon Grafts. *Arthroscopy: The Journal of Arthroscopic & Related Surgery*, **20**, 1015-1025. <https://doi.org/10.1016/j.arthro.2004.08.010>
- [16] Yasuda, K., Kondo, E., Ichiyama, H., Tanabe, Y. and Tohyama, H. (2006) Clinical Evaluation of Anatomic Double-Bundle Anterior Cruciate Ligament Reconstruction Procedure Using Hamstring Tendon Grafts: Comparisons among 3 Different Procedures. *Arthroscopy: The Journal of Arthroscopic & Related Surgery*, **22**, 240-251.

- <https://doi.org/10.1016/j.arthro.2005.12.017>
- [17] Numazaki, H., Tohyama, H., Nakano, H., Kikuchi, S. and Yasuda, K. (2002) The Effect of Initial Graft Tension in Anterior Cruciate Ligament Reconstruction on the Mechanical Behaviors of the Femur-Graft-Tibia Complex during Cyclic Loading. *The American Journal of Sports Medicine*, **30**, 800-805. <https://doi.org/10.1177/03635465020300060801>
- [18] Liao, T., Martinez, A.G.M., Pedoia, V., Ma, B.C., Li, X., Link, T.M., *et al.* (2020) Patellar Malalignment Is Associated with Patellofemoral Lesions and Cartilage Relaxation Times after Hamstring Autograft Anterior Cruciate Ligament Reconstruction. *The American Journal of Sports Medicine*, **48**, 2242-2251. <https://doi.org/10.1177/0363546520930713>
- [19] von Porat, A., Roos, E.M. and Roos, H. (2004) High Prevalence of Osteoarthritis 14 Years after an Anterior Cruciate Ligament Tear in Male Soccer Players: A Study of Radiographic and Patient Relevant Outcomes. *Annals of the Rheumatic Diseases*, **63**, 269-273. <https://doi.org/10.1136/ard.2003.008136>
- [20] Li, G., DeFrate, L.E., Sun, H. and Gill, T.J. (2004) In Vivo Elongation of the Anterior Cruciate Ligament and Posterior Cruciate Ligament during Knee Flexion. *The American Journal of Sports Medicine*, **32**, 1415-1420. <https://doi.org/10.1177/0363546503262175>
- [21] Song, E.K., Oh, L.S., Gill, T.J., Li, G., Gadikota, H.R. and Seon, J.K. (2009) Prospective Comparative Study of Anterior Cruciate Ligament Reconstruction Using the Double-Bundle and Single-Bundle Techniques. *The American Journal of Sports Medicine*, **37**, 1705-1711. <https://doi.org/10.1177/0363546509333478>
- [22] Salavati, M., Hadian, M.R., Mazaheri, M., Negahban, H., Ebrahimi, I., Talebian, S., *et al.* (2009) Test-Retest Reliability of Center of Pressure Measures of Postural Stability during Quiet Standing in a Group with Musculoskeletal Disorders Consisting of Low Back Pain, Anterior Cruciate Ligament Injury and Functional Ankle Instability. *Gait & Posture*, **29**, 460-464. <https://doi.org/10.1016/j.gaitpost.2008.11.016>
- [23] Hunnicutt, J.L., Slone, H.S. and Xerogeanes, J.W. (2020) Implications for Early Post-operative Care after Quadriceps Tendon Autograft for Anterior Cruciate Ligament Reconstruction: A Technical Note. *Journal of Athletic Training*, **55**, 623-627. <https://doi.org/10.4085/1062-6050-172-19>
- [24] Aquino, C.F., Ocarino, J.M., Cardoso, V.A., Resende, R.A., Souza, T.R., Rabelo, L.M., *et al.* (2021) Current Clinical Practice and Return-to-Sport Criteria after Anterior Cruciate Ligament Reconstruction: A Survey of Brazilian Physical Therapists. *Brazilian Journal of Physical Therapy*, **25**, 242-250. <https://doi.org/10.1016/j.bjpt.2020.05.014>
- [25] Wang, H., Ao, Y., Jiang, D., Gong, X., Wang, Y., Wang, J., *et al.* (2015) Relationship between Quadriceps Strength and Patellofemoral Joint Chondral Lesions after Anterior Cruciate Ligament Reconstruction. *The American Journal of Sports Medicine*, **43**, 2286-2292. <https://doi.org/10.1177/0363546515588316>
- [26] Wright, R., Preston, E., Fleming, B., Amendola, A., Andrich, J., Bergfeld, J., *et al.* (2008) A Systematic Review of Anterior Cruciate Ligament Reconstruction Rehabilitation: Part I: Continuous Passive Motion, Early Weight Bearing, Postoperative Bracing, and Home-Based Rehabilitation. *Journal of Knee Surgery*, **21**, 217-224. <https://doi.org/10.1055/s-0030-1247822>
- [27] Grindem, H., Snyder-Mackler, L., Moksnes, H., Engebretsen, L. and Risberg, M.A. (2016) Simple Decision Rules Can Reduce Reinjury Risk by 84% after ACL Reconstruction: The Delaware-Oslo ACL Cohort Study. *British Journal of Sports Medicine*, **50**, 804-808. <https://doi.org/10.1136/bjsports-2016-096031>