

Simulation of Anterior Cruciate Ligament Deficiency in a Musculoskeletal Model with Anatomical Knees

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Abstract: Abnormal knee kinematics and meniscus injury resulting from anterior cruciate ligament (ACL) deficiency are often implicated in joint degeneration even though changes in tibio-femoral contact location after injury are small, typically only a few millimeters. Ligament reconstruction surgery does not significantly reduce the incidence of early onset osteoarthritis. Increased knowledge of knee contact mechanics would increase our understanding of the effects of ACL injury and help guide ACL reconstruction methods. Presented here is a cadaver specific computational knee model combined with a body-level musculoskeletal model from a subject of similar height and weight as the cadaver donor. The knee model was developed in the multi-body framework and includes representation of the menisci. Experimental body-level measurements provided input to the musculoskeletal model. The location of tibio-menisco-femoral contact as well as contact pressures were compared for models with an intact ACL, partial ACL transection (posterolateral bundle transection), and full ACL transection during a muscle driven forward dynamics simulation of a dual limb squat. During the squat, small changes in femur motion relative to the tibia for both partial and full ACL transection push the lateral meniscus in the posterior direction at extension. The central-anterior region of the lateral meniscus then becomes “wedged” between the tibia and femur during knee flexion. This “wedging” effect does not occur for the intact knee. Peak contact pressure and contact locations are similar for the partial tear and complete ACL transection during the deep flexion portion of the squat, particularly on the lateral side. The tibio-femoral contact location on the tibia plateau shifts slightly to the posterior and lateral direction with ACL transection.

Keywords: Anterior Cruciate Ligament, Meniscus, Anterior Cruciate Ligament Deficiency, Musculoskeletal Model, Joint Loading, Biomechanics.

1. INTRODUCTION

It is estimated that 27 million adults in the United States have clinical osteoarthritis with knee osteoarthritis being the most prevalent form affecting 28% of adults over age 45 and 37% of adults over age 60 [1]. Although the prevalence and debilitating nature of osteoarthritis is well documented, the etiology of this chronic disease is not completely understood. The most significant cause of osteoarthritis in young adults is joint trauma including tears of the menisci or ligaments. For example, 12 years after rupture of the anterior cruciate ligament (ACL), 51% of a female population with a mean age of 31 years had radiographic evidence of osteoarthritis [2]. Similarly, 43% of patients with intact ACLs and isolated limited meniscectomy had radiographic evidence of osteoarthritis after 16 years [3]. In addition, the severity of osteoarthritis correlates with the severity of meniscal injury and the amount of tissue removed [4].

Abnormal knee kinematics and meniscus injury resulting from ACL deficiency are often implicated in joint degeneration [5] while ACL reconstruction surgeries attempt to restore normal tibio-femoral motion and prevent long-term consequences [6]. The observed differences in

tibio-femoral kinematics between normal and ACL deficient knees are small, typically only a few millimeters [7, 8]. Studies have also shown that ACL reconstruction does not significantly reduce the incidence of early onset osteoarthritis [2]. Increased knowledge of tibiofemoral joint contact mechanics would increase our understanding of the effects of ACL injury and help guide ACL reconstruction methods. Medical imaging has been used to quantify motion and cartilage contact in normal and ACL deficient knees including knee loading in a magnetic resonance imaging (MRI) tunnel [9] and MRI combined with fluoroscopic images captured during a weight-bearing lunge [10]. Computational models capable of predicting tibio-menisco-femoral contact mechanics during movement would also provide a valuable tool for increasing our understanding of normal joint mechanics as well as knee injury and joint degeneration.

The main objective of this study was to develop a muscle driven forward dynamics model of the lower extremities in a dual limb squat, validate the computational model against experimental data and then simulate the same motion in two different ACL deficient conditions. The model combined a cadaver anatomical knee with anthropometric and motion data from a female subject of similar height and weight as the cadaver donor. The model was developed in the multibody framework and it was validated against measurements of the ground reaction forces and surface electromyography (EMG) taken during the squat motion.

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The multibody model included representation of the menisci with an intact ACL, transected posterolateral ACL bundle, and full ACL transection. The contact pressures on the tibia plateau for the intact and ACL deficient cases were compared during a muscle driven forward dynamics simulation.

2. MATERIALS AND METHODS

2.1. Computational Knee Model

A previously developed anatomical computational knee model was used for this study. The multibody model was created in MD Adams (MSC Software Corporation, Santa Ana, CA) and has been previously described [11]. Only a brief summary is provided here. Knee geometries (tibia, femur, articular cartilage and menisci) were derived from magnetic resonance images (MRI) of a fresh frozen cadaver knee (55 years old, female, left knee, 170 cm height, 72 kg mass). After MRI, the cadaver knee was mounted in a dynamic knee simulator (Kansas Knee Simulator, University of Kansas, Lawrence, KS) and manipulated through a walk cycle. During testing, the forces applied by the actuators of the machine were recorded and bone motion was measured using rigid body markers attached to the femur, tibia, and patella with an Optotrak 3020 system (Northern Digital Inc., Waterloo, Ontario). For calculation of ligament zero-load lengths, the motion of the tibia and femur rigid body markers was recorded while the joint was manually moved through its full range of motion with a minimum force applied (as judged by the experimenter). After testing, the knee was disarticulated and the insertion sites for the anterior cruciate ligament (ACL), posterior cruciate ligament (PCL), medial collateral ligament (MCL), lateral collateral ligament (LCL), patellar tendon, menisci horn attachments, and menisci transverse ligament were measured with an Optotrak digitizing probe.

In the computational model, ligament bundles were represented as one-dimensional non-linear springs. The model included two bundles for the ACL [12] and PCL [13] and three bundles for the MCL and LCL [14]. Non-linear splines were used to describe the force-displacement curve of each ligament including the non-linear “toe” region. The splines were derived from the ligament force as a function of strain, the length of each ligament in the position it was constructed, and the measured zero-load length. The zero-load length of each bundle was determined by calculating the maximum straight-line distance between insertion and origin sites throughout the experimentally measured full range of motion and then applying a correction percentage [11]. The force-length relationship for each ligament is described by: [15, 16]

$$f = \begin{cases} \frac{1}{4}k\varepsilon^2/\varepsilon_l & 0 \leq \varepsilon \leq 2\varepsilon_l \\ k(\varepsilon - \varepsilon_l) & \varepsilon > 2\varepsilon_l \\ 0 & \varepsilon < 0 \end{cases} \quad (1)$$

$$\varepsilon = \left(\frac{l - l_0}{l_0} \right) \quad (2)$$

where k is a stiffness parameter, ε_l is a spring parameter assumed to be 0.03, ε is the engineering strain of each ligament bundle, l is ligament bundle length, and l_0 is the zero-load length. Values of k for each ligament bundle came from Wismans *et al.* and Blankevoort *et al.* [15, 16]. Each 1-D spring also included a parallel damper and a damping coefficient of 0.5 Ns/mm was used for each ligament bundle.

As described in Guess, Liu *et al.*, [11], the medial and lateral tibia plateau cartilage geometries were divided into multiple hexahedral rigid bodies. The size of each hexahedral element's cross-section in the transverse plane of the tibia was 4 x 4 mm and a total of 61 lateral and 72 medial elements were created. Each cartilage element was connected to tibia bone with a fixed joint located at the center of each tibia cartilage-bone interface. A deformable contact constraint was defined between each tibia cartilage element and the femur cartilage geometry. The contact model used for all articulating surfaces in the knee was defined as:

$$F_c = k\delta^n + B(\delta)\dot{\delta} \quad (3)$$

where F_c is the contact force, δ is the interpenetration of geometries, $\dot{\delta}$ is the velocity of interpenetration, k is a spring constant, n is the compliance exponent, and $B(\delta)$ is a damping coefficient. The contact parameters defined between each cartilage rigid body and the femur cartilage were derived by optimizing the contact parameters to match contact pressure predictions of a finite element model [11]. Specifically, the contact parameters were optimized to minimize the difference between pressure predictions of the multibody model and that of an identically loaded and constrained finite element model. The resulting parameters were $k = 140 \text{ N/mm}^{1/1.3}$, $n = 1.3$, and $B = 5 \text{ Ns/mm}$.

Multibody models of the menisci were created by radially sectioning the lateral and medial menisci geometries into 17 elements each and assigning mass properties to every segment based on its volume and a density of 1100 kg/m^3 [17]. The meniscus rigid body elements were connected to neighboring elements by the following stiffness matrix:

$$\begin{bmatrix} F_\theta \\ F_r \\ F_z \\ T_\theta \\ T_r \\ T_z \end{bmatrix} = \begin{bmatrix} K_\theta & K_{\theta r} & K_{\theta z} & 0 & 0 & 0 \\ K_{\theta r} & K_r & K_{rz} & 0 & 0 & 0 \\ K_{\theta z} & K_{rz} & K_z & 0 & 0 & 0 \\ 0 & 0 & 0 & T_\theta & 0 & 0 \\ 0 & 0 & 0 & 0 & T_r & 0 \\ 0 & 0 & 0 & 0 & 0 & T_z \end{bmatrix} \begin{bmatrix} \theta \\ r \\ z \\ a \\ b \\ c \end{bmatrix} \quad (4)$$

where $F_{\theta,r,z}$ and $T_{\theta,r,z}$ are the translational and torsional forces between elements acting in the circumferential, radial, and axial directions. K_θ , K_r , K_z , $K_{\theta r}$, $K_{\theta z}$, K_{rz} , T_θ , T_r , and T_z are the stiffness matrix parameters, θ , r , and z are relative translational displacements and a , b , and c are relative rotational displacements. The stiffness matrix parameters are shown in Table 1. Values for the stiffness parameters were derived through an optimization process that minimized the displacement error between identically loaded finite element and multibody menisci models [18].

Each meniscus was attached to the tibia *via* 4 horn attachments (2 posterior and 2 medial per meniscus)

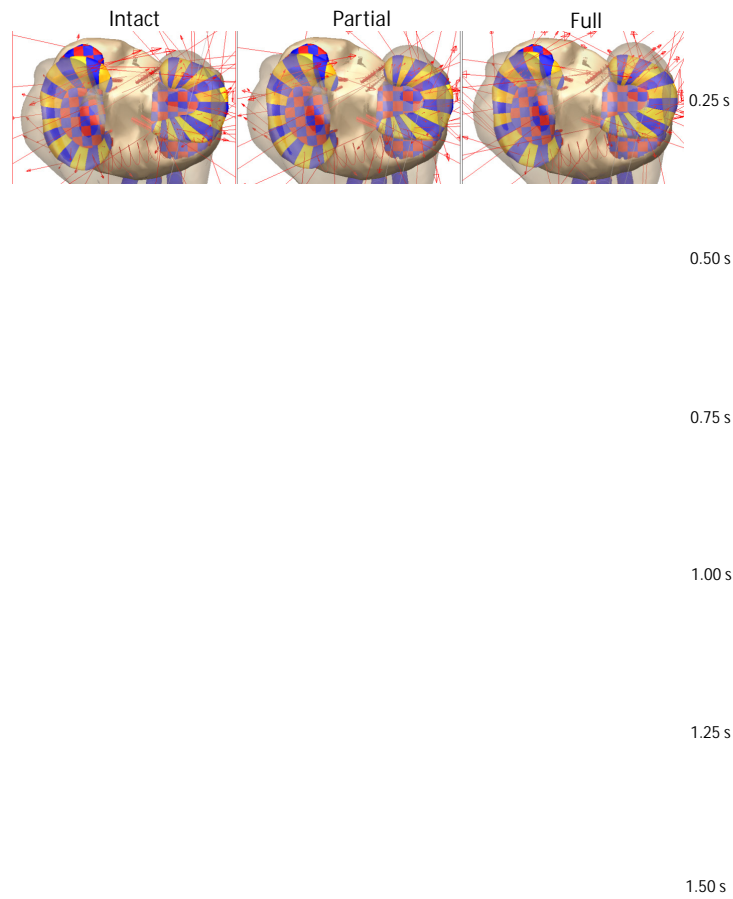


Fig. (11). Tibia plateau for the three model versions at 0.25 second intervals during the forward dynamics simulation. The shaded areas result from the interpenetration of geometries and are the contact patch.

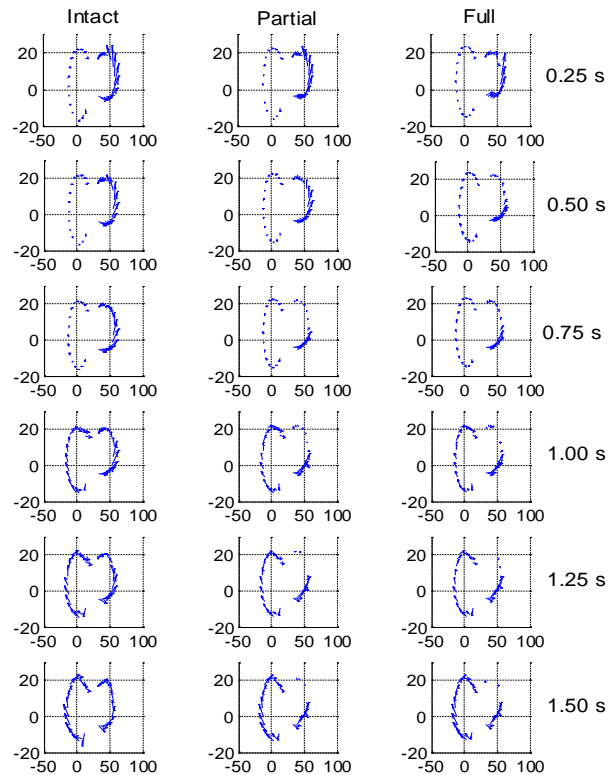


Fig. (12). Internal forces in the menisci rigid bodies for the three model versions at 0.25 second intervals during the forward dynamics simulation.

between the natural knee model compressive force and the measured prosthetic compressive force may be explained by the fact that the musculoskeletal model was a combination of a generic skeletal model based on gait subject height and weight and an MRI derived anatomical knee. The stitching of the geometries could have resulted in slight misalignments of the muscle lines of actions that in turn caused higher loads on the cartilage contacts. This issue will be investigated further in future models where the geometries of all bones will be derived from one subject's MRI data.

Peak contact pressure and contact locations are similar for the partial tear (posterolateral bundle transection) and complete ACL transection cases at flexion, particularly on the lateral side. For the squat simulations, the tibio-femoral contact location shifts to the posterior and lateral direction with ACL transection. This displacement is only a few millimeters and is larger at extension. The change in peak contact and contact patch location on the tibia plateau is consistent with experimental measurements and observations [9, 10, 23]. For both the partial and full transection simulations, small changes in femur motion relative to the tibia push the lateral meniscus in the posterior direction, exposing it to "wedging". This "wedging" effect increases the loads on the meniscus and it is possible that the increased loading damages the tissue and increases the risk of cartilage degeneration during ACL deficiency [24, 25].

For all simulations only the lower body was modeled. As seen in Fig. (2), the upper body of the test subject leans forward during the squat, allowing the body's center of pressure to stay between its base of support. During the forward dynamics simulations, a LifeMOD tracking agent was used to maintain balance during the squat. The tracking agent is a 6-axis spring located between a "dummy" rigid body and the pelvis. The "dummy" rigid body is driven by a motion constraint that follows pelvis motion measured during the inverse dynamics simulation. If the forward dynamics pelvis motion closely follows the inverse dynamics pelvis motion, the tracking agent will have minimal influence on the model. Also, the tracking agent imparts no force in the vertical direction, regardless of pelvis motion. Measured ground reaction forces are symmetrical in the medial-lateral and anterior-posterior directions, but the simulation results include asymmetry in both axes (Fig. 4). For example, both the right and left knee have posterior shear forces while the knee was flexing and an anterior ground reaction shear force when the knee was extending. The model pushes against the tracking agent when these asymmetries occur and these forces will have an influence on muscle force prediction. Misalignment of the knee relative to the hip and ankle and slight misalignment between the left and right sides may be responsible for the predicted ground reaction force asymmetries. Previous work that used a hinge joint for the right knee showed significant asymmetries in the vertical ground reaction forces during the squat [26]. Changing the right knee from a hinge joint to an anatomical joint eliminated the vertical asymmetry. In addition, the vertical ground reaction force predictions may be further improved by including representation of the upper body. In the current model simulations, vertical ground reaction forces were greater for the left foot than the right.

Experimentally, the right foot had a greater vertical reaction force (Fig. 4). Ongoing work in our lab has shown that including representation of the upper body improves prediction of ground reaction forces, particularly the vertical force distribution between the right and left leg. Future musculoskeletal models will include representation of the upper body.

Combining a cadaver knee with *in vivo* gait measurements from a subject of similar height and weight allows *in vitro* validation of the knee model, but it is also a limitation of the study. Additionally, the same measured motion from a subject with an intact ACL was used for the inverse dynamics solution of all three model versions. ACL deficient subjects demonstrate altered gait patterns [27] and it is possible that the kinematics of the dual limb squat would also be different. This altered motion could affect simulation predictions at the knee. Finally, only a limited number of muscle activations were measured with EMG and maximum voluntary muscle contraction was not measured.

The primary objective of this study was to create a musculoskeletal model of the lower extremities that included anatomical representation of the knees, validate the model with experimental data, and then simulate two ACL deficient conditions for a simple motion (squat). The current study is a first step in the development of anatomically correct subject specific musculoskeletal models of the lower extremities to study joint function. Future refinements of this modeling method will include finer segmentations of the menisci and cartilage structures and validation of the models in other dynamic motions.

CONFLICT OF INTEREST

The authors have no conflict of interest with the presented work.

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SUPPLEMENTARY MATERIAL

Supplementary material is available on the publisher's web site along with the published article.

REFERENCES

- [1] R.C. Lawrence, D.T. Felson, C.G. Helmick, L.M. Arnold, H. Choi, R.A. Deyo, S. Gabriel, R. Hirsch, M.C. Hochberg, G.G. Hunder, J. M. Jordan, J.N. Katz, H.M. Kremers, and F. Wolfe, "Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II," *Arthritis. Rheum.*, vol. 58, pp. 26-35, Jan 2008.
- [2] L.S. Lohmander, A. Ostengren, M. Englund, and H. Roos, "High prevalence of knee osteoarthritis, pain, and functional limitations in female soccer players twelve years after anterior cruciate ligament injury," *Arthritis Rheum.*, vol. 50, pp. 3145-52, Oct 2004.

- [3] M. Englund, E.M. Roos, and L.S. Lohmander, "Impact of type of meniscal tear on radiographic and symptomatic knee osteoarthritis: a sixteen-year followup of meniscectomy with matched controls," *Arthritis Rheum*, vol. 48, pp. 2178-87, Aug 2003.
- [4] M. Englund and L.S. Lohmander, "Risk factors for symptomatic knee osteoarthritis fifteen to twenty-two years after meniscectomy," *Arthritis. Rheum.*, vol. 50, pp. 2811-9, Sep 2004.
- [5] L.S. Lohmander, P.M. Englund, L.L. Dahl, and E.M. Roos, "The long-term consequence of anterior cruciate ligament and meniscus injuries: osteoarthritis," *Am. J. Sports. Med.*, vol. 35, pp. 1756-69, Oct 2007.
- [6] C. van Eck, Z. Working, and F. Fu, "Current concepts in anatomic single- and double-bundle anterior cruciate ligament reconstruction," *Phys. Sportsmed.*, vol. 39, pp. 140-8, May 2011.
- [7] L.E. DeFrate, R. Papannagari, T.J. Gill, J.M. Moses, N.P. Pathare, and G. Li, "The 6 degrees of freedom kinematics of the knee after anterior cruciate ligament deficiency: an in vivo imaging analysis," *Am. J. Sports. Med.*, vol. 34, pp. 1240-6, Aug 2006.
- [8] A.D. Georgoulis, A. Papadonikolakis, C.D. Papageorgiou, A. Mitsou, and N. Stergiou, "Three-dimensional tibiofemoral kinematics of the anterior cruciate ligament-deficient and reconstructed knee during walking," *Am. J. Sports. Med.*, vol. 31, pp. 75-9, Jan-Feb 2003.
- [9] J.M. Scarvell, P.N. Smith, K.M. Refshauge, H. Galloway, and K. Woods, "Comparison of kinematics in the healthy and ACL injured knee using MRI," *J. Biomech.*, vol. 38, pp. 255-62, Feb 2005.
- [10] S.K. Van de Velde, J.T. Bingham, A. Hosseini, M. Kozanek, L.E. DeFrate, T.J. Gill, and G. Li, "Increased tibiofemoral cartilage contact deformation in patients with anterior cruciate ligament deficiency," *Arthritis. Rheum.*, vol. 60, pp. 3693-702, Dec 2009.
- [11] T.M. Guess, H. Liu, S. Bhashyam, and G. Thiagarajan, "A multibody knee model with discrete cartilage prediction of tibiofemoral contact mechanics," *Computer Methods in Biomechanics and Biomedical. Engineering.*, pp. 1-15, 2011.
- [12] V.B. Duthon, C. Barea, S. Abrassart, J.H. Fasel, D. Fritschy, and J. Menetrey, "Anatomy of the anterior cruciate ligament," *Knee. Surg. Sports. Traumatol. Arthrosc.*, vol. 14, pp. 204-13, Mar 2006.
- [13] A.A. Amis, A.M. Bull, C.M. Gupta, I. Hijazi, A. Race, and J.R. Robinson, "Biomechanics of the PCL and related structures: posterolateral, posteromedial and meniscofemoral ligaments," *Knee Surg. Sports. Traumatol. Arthrosc.*, vol. 11, pp. 271-81, Sep 2003.
- [14] S.E. Park, L.E. DeFrate, J.F. Suggs, T.J. Gill, H.E. Rubash, and G. Li, "The change in length of the medial and lateral collateral ligaments during in vivo knee flexion," *Knee.*, vol. 12, pp. 377-82, Oct 2005.
- [15] L. Blankevoort, J.H. Kuiper, R. Huiskes, and H.J. Grootenboer, "Articular contact in a three-dimensional model of the knee," *J. Biomech.*, vol. 24, pp. 1019-31, 1991.
- [16] J. Wismans, F. Veldpaus, J. Janssen, A. Huson, and P. Struben, "A three-dimensional mathematical model of the knee-joint," *J. Biomech.*, vol. 13, pp. 677-85, 1980.
- [17] D.C. Fithian, M.A. Kelly, and V.C. Mow, "Material properties and structure-function relationships in the menisci," *Clin. Orthop.*, pp. 19-31., 1990.
- [18] T.M. Guess, G. Thiagarajan, M. Kia, and M. Mishra, "A subject specific multibody model of the knee with menisci," *Med. Eng. Phys.*, vol. 32, pp. 505-15, Jun 2010.
- [19] T.L. Donahue, M.L. Hull, M.M. Rashid, and C.R. Jacobs, "A finite element model of the human knee joint for the study of tibiofemoral contact," *J. Biomech. Eng.*, vol. 124, pp. 273-80, Jun 2002.
- [20] J.P. Smith, 3rd and G.R. Barrett, "Medial and lateral meniscal tear patterns in anterior cruciate ligament- deficient knees. A prospective analysis of 575 tears," *Am. J. Sports. Med.*, vol. 29, pp. 415-9., 2001.
- [21] J.B. Duncan, R. Hunter, M. Purnell, and J. Freeman, "Meniscal injuries associated with acute anterior cruciate ligament tears in alpine skiers," *Am. J. Sports. Med.*, vol. 23, pp. 170-2, Mar-Apr 1995.
- [22] A. Mundermann, C.O. Dyrby, D.D. D'Lima, C.W. Colwell, Jr., and T.P. Andriacchi, "In vivo knee loading characteristics during activities of daily living as measured by an instrumented total knee replacement," *J. Orthop. Res.*, vol. 26, pp. 1167-72, Sep 2008.
- [23] J.M. Scarvell, P.N. Smith, K.M. Refshauge, H.R. Galloway, and K. R. Woods, "Association between abnormal kinematics and degenerative change in knees of people with chronic anterior cruciate ligament deficiency: a magnetic resonance imaging study," *Aust. J. Physiother.*, vol. 51, pp. 233-40, 2005.
- [24] A. Meunier, M. Odensten, and L. Good, "Long-term results after primary repair or non-surgical treatment of anterior cruciate ligament rupture: a randomized study with a 15-year follow-up," *Scand. J. Med. Sci. Sports.*, vol. 17, pp. 230-7, Jun 2007.
- [25] J.K. Seon, E.K. Song, and S.J. Park, "Osteoarthritis after anterior cruciate ligament reconstruction using a patellar tendon autograft," *Int. Orthop.*, vol. 30, pp. 94-8, Apr 2006.
- [26] T. Guess, "Forward dynamics simulation using a natural knee with menisci in the multibody framework," *Multibody. System. Dynamics.*, pp. 1-17.
- [27] R. Ferber, L.R. Osternig, M.H. Woollacott, N.J. Wasielewski, and J.H. Lee, "Gait mechanics in chronic ACL deficiency and subsequent repair," *Clin. Biomech. (Bristol, Avon).*, vol. 17, pp. 274-85, May 2002.

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