INTRODUCTION:
Rupture of the anterior cruciate ligament (ACL) is a common acute injury which is associated with an increased risk for developing osteoarthritis (OA) in the affected knee [1]. The cartilage degeneration in ACL deficiency likely depends on a combination of endogenous and environmental risk factors [2]. In the current clinical practice though, the immediate outcome of ACL deficiency in the physically active population is often ACL reconstruction, rather than cartilage degeneration. With excellent postoperative stability and the ability to return to athletic activities [3], each year ~100,000 patients opt for reconstruction of the ACL in the United States [4]. However, despite the reliable surgical results, no long-term difference in OA prevalence has been detected between those patients that opted for conservative treatment and those that opted for the surgical reconstruction [2, 5].

We recently found that ACL deficiency shifted the articular contact location to smaller regions of thinner cartilage, and increased the magnitude of cartilage contact deformation, providing insight in the possible underlying biomechanical dimension of cartilage degeneration [6]. In the present study, we used a dual fluoroscopic and magnetic resonance imaging (MRI) technique to re-analyze the cartilage contact biomechanics of the tibiofemoral joint during in-vivo weight-bearing flexion of the knee as was described in detail previously in our study sample of eight patients with an isolated ACL rupture [6], now six months following clinically successful reconstruction of the ACL. We hypothesized that ACL reconstruction is unable to correct the abnormal tibiofemoral cartilage contact biomechanics caused by ACL injury.

METHODS:
Eight patients (6 men and 2 women; age range 19-38 years old) with an acute, isolated ACL rupture in one knee and the contralateral knee intact were included in the study.

Both knees were imaged with an MR scanner to create 3D meshed models of the knees. Each knee model included the bony geometry of the tibia and femur, and tibial and femoral cartilage layers. The models were digitally manipulated until the projections of the models matched the outlines of series of orthogonally placed fluoroscopic images, which were acquired as the patient performed a single-leg lunge. Fluoroscopic images were obtained pre-operatively and six months following ACL reconstruction. Reconstruction was performed in the standard fashion using a 10mm bone-patellar tendon-bone (BPTB) autograft.

Cartilage contact location was defined as the location of peak cartilage deformation, referenced to Cartesian coordinate systems on the tibial plateau. The size of the tibiofemoral contact area was determined by computing the area of tibial cartilage that intersected the femoral cartilage deformation, referenced to Cartesian coordinate systems on the tibial plateau. The location of cartilage contact was defined as the location of peak cartilage contact deformation, providing insight in the possible underlying biomechanical dimension of cartilage degeneration [6]. In the present study, we used a dual fluoroscopic and magnetic resonance imaging (MRI) technique to re-analyze the cartilage contact biomechanics of the tibiofemoral joint during in-vivo weight-bearing flexion of the knee as was described in detail previously in our study sample of eight patients with an isolated ACL rupture [6], now six months following clinically successful reconstruction of the ACL. We hypothesized that ACL reconstruction is unable to correct the abnormal tibiofemoral cartilage contact biomechanics caused by ACL injury.

RESULTS:

**Location of cartilage contact.** ACL reconstruction did not restore the lateral and posterior shift of the cartilage contact location that was measured in ACL-deficient knees to normal at 0° and 15° of flexion (P < 0.05). Between 30° and 90°, no significant differences in cartilage contact location were found between the knee conditions (P > 0.05).

**Size of the contact area.** The cartilage contact areas after ACL reconstruction were significantly smaller than the normal cartilage contact areas at 0° of flexion (P < 0.05). Between 15° and 90° of flexion, no statistically significant differences in size of contact area were observed between the knee conditions (P > 0.05).

**Cartilage thickness at the contact area.** We previously found that the cartilage thickness at the location of maximum cartilage contact deformation in ACL-deficient knees was significantly lower as compared with healthy contralateral knees. In the present study, the maximum cartilage contact deformation continued to occur at areas where the cartilage thickness was significantly lower than the normal condition between 0° and 30° of flexion on the medial compartment and between 0° and 15° of flexion on the lateral compartment (P < 0.05).

**Magnitude of cartilage contact deformation.** In the medial compartment, ACL reconstruction reduced the increased magnitude of cartilage contact deformation to normal levels at 30° and 60° of flexion (P > 0.05). However, significant differences persisted between the healthy contralateral knees and the ACL-reconstructed knees at 0° and 15° of flexion (P < 0.05) (Fig. 2A). Similarly, normal levels of cartilage contact deformation were found at flexion angles between 15° and 90° in the lateral compartment of the ACL-reconstructed knees (P > 0.05), but at 0° of flexion a significant increase in cartilage contact deformation persisted after ACL reconstruction (P < 0.05) (Fig. 2B).

DISCUSSION:
Based on the limitations of the present methodology, the formulation of inclusive insight into the pathogenesis of OA following ACL reconstruction remained beyond our reach: the assessment of underlying physiochemical activities, occult loss of the structural integrity of cartilage that was already present prior to the surgery, or regional variations in the mechanical properties of articular cartilage has not been incorporated in the study design. Nevertheless, the present study is the first to provide data on the persistent abnormal in-vivo cartilage contact biomechanics following reconstruction of the ACL – one of the most commonly performed surgeries in sports medicine.

REFERENCES: