Chapter 9 Video Analysis of ACL Injury Mechanisms Using a Model-Based Image-Matching Technique

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 Abstract Model-based image-matching (MBIM) technique has enabled detailed video analysis of injury situations that previously had been limited to simple visual inspection. We have analyzed anterior cruciate ligament (ACL) injury situations from ten analogue and one HD video sequences using the MBIM technique. The knee kinematical patterns were remarkably consistent, with immediate valgus and internal rotation (IR) motion occurring within 40 ms after initial contact (IC), and then external rotation was observed. Peak vertical ground reaction force (GRF) occurred at 40 ms after IC. Based on these results, it is likely that the ACL injury occurred approximately 40 ms after IC. In the one HD video available, 9 mm of abrupt anterior tibial translation at the time of injury was also detected. On the other hand, the hip kinematics were constant with an abducted, flexed and IR position during 40 ms after IC. Based on these results and other previous studies, we propose a new hypothesis for ACL injury mechanisms that valgus loading and lateral compression generate IR motion and anterior translation of the tibia, due to the joint geometry, result in ACL rupture. Moreover, it seems that the hip is relatively 'locked' at IC, cannot absorb energy from GRF and thus the knee is exposed to a larger force, which leads to ACL injury. These results suggest that prevention programs should focus on acquiring a good cutting and landing technique promoting knee flexion and avoiding knee valgus and foot internal rotation, and with greater hip flexion to absorb energy from GRF. Moreover, the fact that the ACL injury occurs within 40 ms after IC suggests that "feed-forward" strategies before landing may be critical, as reflex-based "feed-back" strategies are too slow to prevent ACL injuries.

 Keywords Anterior cruciate ligament • Video analysis • Injury mechanism

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9.1 Introduction

 Anterior cruciate ligament (ACL) injuries commonly occur during sports activities, and the number has increased in recent years. Development of ACL reconstruction procedures has enabled athletes to return to sports and favorable short-term results have been achieved; yet it takes a relatively long period for most athletes to get back to sports activities. It has also been reported that ACL reconstruction cannot prevent progression of osteoarthritis (Oiestad et al. [2009](#page-11-0)). Therefore, the importance of ACL injury prevention has been recognized, and establishment of effective injury prevention programs is needed. As of today, various ACL injury prevention pro-grams have been developed successfully (Caraffa et al. [1996](#page-10-0); Gilchrist et al. 2008; Mandelbaum et al. 2005; Myklebust et al. [2003](#page-11-0); Olsen et al. [2005](#page-11-0)); however, it is not well understood how the different elements in these multicomponent programs play particular roles in preventing the injury. We know that ACL injuries predominantly result from a non-contact mechanism and occur during cutting or one-leg landing maneuvers (Boden et al. [2000](#page-10-0); Krosshaug et al. [2007a](#page-10-0); Olsen et al. 2004). Nevertheless, to develop more targeted injury preventive programs, a more detailed description of the mechanism(s) of non-contact ACL injuries is needed.

9.1.1 Previously Proposed ACL Injury Mechanisms

 Several theories have been proposed regarding the mechanisms for non-contact ACL injury; however, it is still a matter of controversy, with the main opponents favoring either sagittal or non-sagittal plane knee joint loading. DeMorat et al. proposed that aggressive quadriceps loading was responsible, based on a cadaver study which demonstrated that aggressive quadriceps loading could take the ACL to failure (DeMorat et al. [2004](#page-10-0) ; Yu and Garrett [2007](#page-11-0)). In contrast, Mclean et al., based on a mathematical simulation model, argued that sagittal plane loading alone could not produce such injuries (McLean et al. 2004, 2005). A prospective cohort study among female athletes, showing that increased dynamic valgus and high valgus loads increased injury risk, lead Hewett et al. to suggest valgus loading as an impor-tant component (Hewett et al. 2005; Quatman and Hewett [2009](#page-11-0)). Some video analyses also showed that valgus collapse seemed to be the main mechanism among female athletes (Krosshaug et al. [2007a](#page-10-0); Olsen et al. 2004). However, cadaver studies and mathematical simulation have shown that pure valgus motion would not produce ACL injuries without tearing the medial collateral ligament (MCL) first (Mazzocca et al. [2003](#page-11-0); Shin et al. [2009](#page-11-0)).

 Nevertheless, other simulation studies have suggested that valgus loading would substantially increase ACL force in situations where an anterior tibial shear force is applied (Withrow et al. 2006). Based on MRI findings, Speer et al. reported that bone bruises of the lateral femoral condyle or posterolateral portion of tibial plateau occurred in more than 80 % of acute ACL non-contact injuries. They concluded that valgus in combination with internal rotation and/or anterior tibial translation occurred at the time of ACL injuries (Speer et al. [1992](#page-11-0)). Furthermore, it has been shown that valgus loading induces a coupled motion of valgus and internal tibial rotation (Matsumoto 1990; Matsumoto et al. [2001](#page-11-0)).

 Although both cadaver studies and MRI studies have suggested that internal rotation is present in ACL injury situations, video analyses have suggested that valgus in combination with external rotation is the most frequent motion pattern (Olsen et al. 2004; Ebstrup and Bojsen-Moller [2000](#page-10-0)).

9.1.2 Research Approaches to Injury Mechanisms

 As mentioned above, several different approaches have been used to investigate ACL injury mechanisms, including athlete interviews, clinical studies, laboratory motion analysis, video analysis, cadaver studies, and mathematical simulations (Krosshaug et al. 2005). Among these, video analysis of injury tapes is the only method available to extract kinematic data from actual injury situations. However, video analyses have so far been limited to simple visual inspection (Boden et al. [2000](#page-10-0); Olsen et al. 2004; Cochrane et al. 2007), and the accuracy has been shown to be poor, even among experienced researchers (Krosshaug et al. 2007b). In addition, simple visual inspection is not sufficient to extract time course data for joint angles, velocities and accelerations; therefore, it is difficult to determine the point of ACL rupture.

9.1.3 Development of Model-Based Image-Matching Technique

 Model-based image-matching (MBIM) has been developed as an alternative to simple visual inspection, in order to extract joint kinematics from video recordings using one or more uncalibrated cameras (Krosshaug and Bahr 2005). The detailed procedures of the MBIM technique have been described in the literature, as well as in the previous section of this chapter. The idea underpinning this technique is that matching a skeleton model to the background video sequences provides an estimate of the actual three-dimentional body kinematics using the commercially available program Poser® and Poser® Pro Pack (Curious Labs Inc., Santa Cruz, California, USA). This technique has been validated in non-injury situations in a laboratory environment (Krosshaug and Bahr 2005) and also has been found to be feasible for use in actual ACL injury situations (Krosshaug et al. [2007c](#page-10-0)). In the following we propose a new description of the mechanism for non-contact ACL injury based on analyses using the MBIM technique. In addition, suggestions for injury programs based on the proposed mechanism are introduced.

9.2 Biomechanics in Non-contact ACL Injury

Ten ACL injury situations from women's team handball $(n=7)$ and basketball $(n=3)$, recorded with at least two analogue cameras during TV broadcasts, were analyzed using MBIM technique (Fig. 9.1). All the players were handling the ball in the injury situation; seven were in possession of the ball at the time of injury, two had shot and one had passed the ball. In six cases, there was player-to-player contact with an opponent at the time of injury, all of them to the torso being pushed or held. There was no direct contact to the knee. The injury situations could be classified into two groups; seven cases occurred when cutting and three during one-leg landings.

 The knee kinematical patterns were remarkably consistent among the ten cases (Fig. 9.2). The knee was relatively straight, with a flexion angle of 23° (range, 11–30°), at initial contact (IC) and had increased by 24° (95 % CI, 19–29°, p < 0.001) 40 ms later. The knee abduction angle was neutral, 0° (range, −2° to 3°) at IC, but had increased by 12° (95 % CI, 10–13°, p < 0.001) 40 ms later. As for knee rotation angle, the knee was externally rotated 5° (range, -5° to 12°) at IC,

 Fig. 9.1 An example of a video matched in Poser, three-camera basketball injury situation 50 ms after IC. The *two top panels* and *left bottom panel* show the customized skeleton model and the basketball court model superimposed on and matched with the background video image from three cameras with different angles. The *right bottom two panels* show the skeleton model from a side view created in Poser

 Fig. 9.2 Time sequences of the mean knee angles (°) (*black line*) of the 10 cases with 95 % confidence intervals (CI) (*grey area*). Time 0 indicates IC and the dotted vertical line indicates the time point 40 ms after IC

but abruptly rotated internally by 8° (95 % CI, 2–14°, p=0.037) during the first 40 ms. From 40 ms to 300 ms after IC, however, we observed an external rotation of 17 \degree (95 % CI, 13–22 \degree , p < 0.001). In addition, the estimated peak vertical ground reaction force (GRF) was 3.2 times body weight (95 % CI, 2.7–3.7), and occurred at 40 ms (range, 0–83) after IC. On the other hand, the hip kinematics was relatively constant at a 20 $^{\circ}$ abducted, 50 $^{\circ}$ flexed and 30 $^{\circ}$ IR position during 40 ms after IC (Fig. [9.3](#page-5-0)).

 However, a limitation of the above mentioned analysis was how accurate the joint kinematics and timing of peak GRF could be estimated from the relatively low frame rate (50 or 60 Hz) and low quality images (768×576 pixels) in analog video sequences, and therefore we were unable to assess the anterior translation of the tibia. However, a noncontact ACL injury situation in a male footballer was available which had been recorded using four high-definition (HD, 1080i) cameras, including two high-speed recordings (100 and 300 Hz). In this case, the 26-year old male elite football player suffered a noncontact ACL injury to his right knee during a national

 Fig. 9.3 Time sequences of the mean hip angles (°) (*black line*) of the 10 cases with 95 % CI (*grey area*). Time 0 indicates IC and the dotted vertical line indicates the time point 40 ms after IC

team match, when he tried to stop after having passed the ball with his right leg. This case was analyzed using the MBIM technique to describe the more detailed joint kinematics, including tibial translations (Fig. [9.4](#page-6-0)). Knee kinematics in this case were strikingly consistent with the previous analyses of the ten cases (Fig. [9.5 \)](#page-6-0). The knee was flexed 35 $^{\circ}$ at IC, with initial extension (26 $^{\circ}$ of flexion) until 20 ms after IC, after which flexion angle continued to increase. The knee abduction angle was neutral at IC, but had increased by 21° 30 ms later. The knee was externally rotated 11 \degree at IC, but abruptly rotated internally by 21 \degree during the first 30 ms, then changed its direction to external rotation after this. In addition, anterior tibial translation was able to be detected; it started to occur at 20 ms after IC, where the knee was the most extended, and by 30 ms after IC approximately 9 mm of anterior translation had occurred. The translations plateaued by 150 ms, and then shifted back to a reduced position between 200 ms and 240 ms after IC.

 Fig. 9.4 A soccer injury situation recorded using HD cameras. Each panel shows the customized skeleton model and the football pitch model superimposed on and matched with the background video image from each camera. Overview camera and rear camera had an effective frame rate after being deinterlaced of 50 Hz, frontal camera 100 Hz and side camera 300 Hz

 Fig. 9.5 Time sequences of knee joint angles (left axis) and anterior tibial translation (right axis) in the soccer case. Time θ (*a*) indicates IC and the *dotted vertical lines* (*b*) and (*c*) indicate the time point 20 and 30 ms after IC, respectively

9.3 Timing of Non-contact ACL Injury

 It has not been possible to determine the exact timing of ACL injury from video analysis based on simple visual inspection (Boden et al. [2000](#page-10-0); Krosshaug et al. [2007a](#page-10-0); Olsen et al. [2004](#page-11-0)). However, this may be possible by using the MBIM technique, by assessing abnormal joint configurations, sudden changes in joint angular motion and timing of GRFs. The extracted knee kinematics during ACL injuries using the MBIM technique showed that sudden increase of valgus and internal rotation angle occurred within the first 40 ms after IC. These periods also correspond to the average peak vertical GRF in these cases. Moreover, in the case recorded using HD cameras, abrupt anterior tibial translation reached 9 mm in 30 ms after IC, which corresponds to the maximum anterior translation in intact knees (Jakob et al. 1987; Meyer and Haut 2008). Based on these results, together with the previous studies showing that the ACL was strained shortly (approximately 40 ms) after IC in simulated landing (Withrow et al. [2006](#page-11-0); Shin et al. 2007), it seems likely that the injury occurs within 40 ms for the majority of these cases.

9.4 Mechanism for Non-contact ACL Injury

 As already mentioned, valgus collapse in combination with external rotation (i.e. knee in, toe out) has frequently been identified as an ACL injury mechanism based on simple visual inspection of injury video tapes. However, it has been discussed as to whether these kinematics actually represent the cause for ACL injuries or simply are a result of the ACL being torn (Olsen et al. [2004](#page-11-0); Ebstrup and Bojsen-Moller [2000 \)](#page-10-0). Our results using the MBIM technique showed that immediate valgus motion occurred within 40 ms after IC. The abrupt internal rotation also occurred during the first 40 ms after IC, then external rotation was observed, which seems to have occurred after the ACL was torn. In addition, anterior tibial translation started a little after IC, and increased abruptly until when the injury might have occurred. The discrepancy between the previous studies and our results could be that the abrupt internal rotation and anterior tibial translation observed using the MBIM technique analysis are likely not easily detected from visual inspection alone; the external rotation that occurs afterwards is more pronounced and therefore easier to observe. The internal-to-external rotation sequence with anterior tibial translation has also been reported previously. In a recent cadaver study, the application of pure compressive loads led to anterior tibial translation and internal tibial rotation of up to 8° , followed by a sudden external rotation of 12° (Meyer and Haut 2008). The combination of internal tibial rotation and anterior tibial translation is probably caused by the joint surface geometry. The concave geometry of the medial tibia facet combined with the slightly convex lateral tibia facet may cause the lateral femoral condyle to slip back. This may also explain why ACL-injured patients tend to have greater posterior lateral tibial plateau slopes than uninjured controls (Brandon et al. [2006](#page-10-0); Stijak et al. [2008](#page-11-0); Hashemi et al. [2010](#page-10-0)).

 Fig. 9.6 The proposed non-contact ACL injury mechanism. (**a**) An unloaded knee. (**b**) When valgus loading is applied, the MCL becomes taut and lateral compression occurs. (**c**) This compressive load causes a lateral femoral posterior displacement, probably due to the posterior slope of lateral tibial plateau, and the tibia translates anteriorly and rotates internally, resulting in ACL rupture. (d) After the ACL is torn, the primary restraint to anterior translation of the tibia is gone. This causes the medial femoral condyle to also be displaced posteriorly, resulting in external rotation of the tibia

Combining the results obtained using the MBIM technique with previous findings, the following hypothesis for the mechanism of non-contact ACL injury is pro-posed (Fig. 9.6) (Oiestad et al. [2009](#page-11-0)): when valgus loading is applied, the MCL becomes taut and lateral compression occurs (Caraffa et al. [1996](#page-10-0)). This compressive load causes a lateral femoral posterior displacement, probably due to the posterior slope of lateral tibial plateau, and the tibia translates anteriorly and rotates internally, resulting in ACL rupture (Gilchrist et al. 2008). After the ACL is torn, the primary restraint to anterior translation of the tibia is gone. This causes the medial femoral condyle to also be displaced posteriorly, resulting in external rotation of the tibia. This external rotation may be exacerbated by the typical movement pattern when athletes plant and cut, where the foot typically rotates externally relative to the trunk.

9.5 The Role of the Hip in Preventing ACL Injury

 The lower extremities act as a kinetic chain during dynamic tasks and the control of hip motion substantially affects the knee motion. Researchers have studied the relationships between hip biomechanics and ACL injury. As for hip biomechanics being a risk factor for ACL injury, Decker et al. ([2003 \)](#page-10-0) reported that, in drop landing, energy absorption at the hip joint, and hip flexion angles at IC were less in females than in males. Schmitz et al. (2007) reported that, in single-leg landing, energy absorption at the hip and total hip flexion displacement were smaller in females, whereas peak vertical GRF was larger in females. Yu et al. (2006) also reported that hip flexion angular velocity at IC was negatively correlated with peak vertical GRF in a stop-jump task. When it comes to ACL injury mechanisms, Heshemi et al.

(2007) reported that, in a cadaver study, a restricted flexion of the hip at 20° combined with low quadriceps and hamstrings force levels in simulated single-leg landing were found to be conducive to ACL injury. A video analysis has shown that ACL injured subjects' hip flexion and abduction angles were constant during 100 ms after IC, whereas uninjured control subjects' hip flexion increased by 15° in cutting/ landing maneuvers (Boden et al. 2009). Our study using MBIM technique also showed that hip kinematics was constant during 40 ms after IC in an abducted, flexed and internally-rotated position, which seems to play a significant role in the mechanism of ACL injury. In this regard, Hashemi et al. (2011) have proposed a mechanism called "hip extension, knee flexion paradox", i.e. that a mismatch between hip and knee flexion in landing is the cause of ACL injury. In normal conditions, both the knee and the hip flex together in landings, whereas in unbalanced landings, the knee is forced to flex while the hip is forced to extend, and the tibia will undergo anterior translation, which will increase the risk of ACL injury.

There are some possible causes of hip/knee mismatch (Oiestad et al. [2009](#page-11-0)): in the sagittal plane, an upright or backward-leaning trunk position at IC makes the center of mass posterior to knee, and increased GRF may encourage more knee flexion than hip flexion, and relatively act to extend hip (Caraffa et al. 1996). In other planes insufficient hip abductor/external rotator strength or activation would lead to adducted/internally-rotated position of the hip, causing knee valgus (Gilchrist et al. [2008](#page-10-0)). Large hip internal rotation at IC seen in our video analysis could also be an explanation; ACL injured patients could have limited range of motion in internal rotation, and the hip joint may be locked at a large internally-rotated position. As a matter of fact, hip dysplasia has also been reported to be a risk factor of ACL injury (Yamazaki et al. 2011).

 For these reasons, it seems that the hip joint is relatively locked at IC, cannot absorb energy from GRF and the knee joint is thus exposed to larger forces, which may lead to ACL injury. Therefore, it is important that prevention efforts should focus not only on the knee joint, but also on the hip joint.

9.6 ACL Injury Prevention Based on the Proposed Mechanisms

Based on the mechanisms clarified using MBIM technique, prevention strategy for ACL injury can be proposed as follows (Oiestad et al. [2009](#page-11-0)): as the kinematics when ACL injury is happening is knee valgus and internal rotation with the hip being locked, it is important to acquire a good cutting and landing technique with knee flexion, avoiding knee valgus and foot internal rotation, and with hip flexion to absorb energy from GRF, avoiding hip internal rotation (Caraffa et al. [1996](#page-10-0)). As ACL injuries occur approximately 40 ms after IC, it is likely that a "feed-back" strategy, i.e. ACL prevention program focusing on training after landing cannot prevent ACL injury; it takes at least 150–200 ms to react after landing at risk. Prevention efforts should focus on a "feed-forward" strategy before landing, i.e. training muscular activation and neural control during the pre-landing phase.

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References

- Boden BP, Dean GS, Feagin JA Jr, Garrett WE Jr (2000) Mechanisms of anterior cruciate ligament injury. Orthopedics 23:573–578
- Boden BP, Torg JS, Knowles SB, Hewett TE (2009) Video analysis of anterior cruciate ligament injury: abnormalities in hip and ankle kinematics. Am J Sports Med 37:252–259
- Brandon ML, Haynes PT, Bonamo JR, Flynn MI, Barrett GR, Sherman MF (2006) The association between posterior-inferior tibial slope and anterior cruciate ligament insufficiency. Arthroscopy 22:894–899
- Caraffa A, Cerulli G, Projetti M, Aisa G, Rizzo A (1996) Prevention of anterior cruciate ligament injuries in soccer. A prospective controlled study of proprioceptive training. Knee Surg Sports Traumatol Arthrosc 4:19–21
- Cochrane JL, Lloyd DG, Buttfield A, Seward H, McGivern J (2007) Characteristics of anterior cruciate ligament injuries in Australian football. J Sci Med Sport 10:96–104
- Decker MJ, Torry MR, Wyland DJ, Sterett WI, Richard SJ (2003) Gender differences in lower extremity kinematics, kinetics and energy absorption during landing. Clin Biomech (Bristol, Avon) 18:662–9
- DeMorat G, Weinhold P, Blackburn T, Chudik S, Garrett W (2004) Aggressive quadriceps loading can induce noncontact anterior cruciate ligament injury. Am J Sports Med 32:477–483
- Ebstrup JF, Bojsen-Moller F (2000) Anterior cruciate ligament injury in indoor ball games. Scand J Med Sci Sports 10:114–116
- Gilchrist J, Mandelbaum BR, Melancon H et al (2008) A randomized controlled trial to prevent noncontact anterior cruciate ligament injury in female collegiate soccer players. Am J Sports Med 36:1476–1483
- Hashemi J, Chandrashekar N, Jang T, Karpat F, Oseto M, Ekwaro-Osire S (2007) An alternative mechanism of non-contact anterior cruciate ligament injury during jump-landing: in-vitro simulation. Exp Mech 47:347–354
- Hashemi J, Chandrashekar N, Mansouri H et al (2010) Shallow medial tibial plateau and steep medial and lateral tibial slopes: new risk factors for anterior cruciate ligament injuries. Am J Sports Med 38:54–62
- Hashemi J, Breighner R, Chandrashekar N et al (2011) Hip extension, knee flexion paradox: a new mechanism for non-contact ACL injury. J Biomech 44:577–585
- Hewett TE, Myer GD, Ford KR et al (2005) Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. Am J Sports Med 33:492–501
- Jakob RP, Staubli HU, Deland JT (1987) Grading the pivot shift. Objective tests with implications for treatment. J Bone Joint Surg Br 69:294–299
- Krosshaug T, Bahr R (2005) A model-based image-matching technique for three-dimensional reconstruction of human motion from uncalibrated video sequences. J Biomech 38:919–929
- Krosshaug T, Andersen TE, Olsen OE, Myklebust G, Bahr R (2005) Research approaches to describe the mechanisms of injuries in sport: limitations and possibilities. Br J Sports Med 39:330–339
- Krosshaug T, Nakamae A, Boden BP et al (2007a) Mechanisms of anterior cruciate ligament injury in basketball: video analysis of 39 cases. Am J Sports Med 35:359–367
- Krosshaug T, Nakamae A, Boden B et al (2007b) Estimating 3D joint kinematics from video sequences of running and cutting maneuvers–assessing the accuracy of simple visual inspection. Gait Posture 26:378–385
- Krosshaug T, Slauterbeck JR, Engebretsen L, Bahr R (2007c) Biomechanical analysis of anterior cruciate ligament injury mechanisms: three-dimensional motion reconstruction from video sequences. Scand J Med Sci Sports 17:508–519
- Mandelbaum BR, Silvers HJ, Watanabe DS et al (2005) Effectiveness of a neuromuscular and proprioceptive training program in preventing anterior cruciate ligament injuries in female athletes: 2-year follow-up. Am J Sports Med 33:1003–1010

Matsumoto H (1990) Mechanism of the pivot shift. J Bone Joint Surg Br 72:816–821

- Matsumoto H, Suda Y, Otani T, Niki Y, Seedhom BB, Fujikawa K (2001) Roles of the anterior cruciate ligament and the medial collateral ligament in preventing valgus instability. J Orthop Sci 6:28–32
- Mazzocca AD, Nissen CW, Geary M, Adams DJ (2003) Valgus medial collateral ligament rupture causes concomitant loading and damage of the anterior cruciate ligament. J Knee Surg 16:148–151
- McLean SG, Huang X, Su A, Van Den Bogert AJ (2004) Sagittal plane biomechanics cannot injure the ACL during sidestep cutting. Clin Biomech (Bristol, Avon) 19:828–38
- McLean SG, Andrish JT, van den Bogert AJ (2005) Aggressive quadriceps loading can induce noncontact anterior cruciate ligament injury. Am J Sports Med 33:1106; author reply −7
- Meyer EG, Haut RC (2008) Anterior cruciate ligament injury induced by internal tibial torsion or tibiofemoral compression. J Biomech 41:3377–3383
- Myklebust G, Engebretsen L, Braekken IH, Skjolberg A, Olsen OE, Bahr R (2003) Prevention of anterior cruciate ligament injuries in female team handball players: a prospective intervention study over three seasons. Clin J Sport Med 13:71–78
- Oiestad BE, Engebretsen L, Storheim K, Risberg MA (2009) Knee osteoarthritis after anterior cruciate ligament injury: a systematic review. Am J Sports Med 37:1434–1443
- Olsen OE, Myklebust G, Engebretsen L, Bahr R (2004) Injury mechanisms for anterior cruciate ligament injuries in team handball: a systematic video analysis. Am J Sports Med 32:1002–1012
- Olsen OE, Myklebust G, Engebretsen L, Holme I, Bahr R (2005) Exercises to prevent lower limb injuries in youth sports: cluster randomised controlled trial. BMJ 330:449
- Quatman CE, Hewett TE (2009) The anterior cruciate ligament injury controversy: is "valgus collapse" a sex-specific mechanism? Br J Sports Med 43:328–335
- Schmitz RJ, Kulas AS, Perrin DH, Riemann BL, Shultz SJ (2007) Sex differences in lower extremity biomechanics during single leg landings. Clin Biomech (Bristol, Avon) 22:681–8
- Shin CS, Chaudhari AM, Andriacchi TP (2007) The influence of deceleration forces on ACL strain during single-leg landing: a simulation study. J Biomech 40:1145–1152
- Shin CS, Chaudhari AM, Andriacchi TP (2009) The effect of isolated valgus moments on ACL strain during single-leg landing: a simulation study. J Biomech 42:280–285
- Speer KP, Spritzer CE, Bassett FH 3rd, Feagin JA Jr, Garrett WE Jr (1992) Osseous injury associated with acute tears of the anterior cruciate ligament. Am J Sports Med 20:382–389
- Stijak L, Herzog RF, Schai P (2008) Is there an influence of the tibial slope of the lateral condyle on the ACL lesion? A case-control study. Knee Surg Sports Traumatol Arthrosc 16:112–117
- Withrow TJ, Huston LJ, Wojtys EM, Ashton-Miller JA (2006) The effect of an impulsive knee valgus moment on in vitro relative ACL strain during a simulated jump landing. Clin Biomech (Bristol, Avon) 21:977–83
- Yamazaki J, Muneta T, Ju YJ, Morito T, Okuwaki T, Sekiya I (2011) Hip acetabular dysplasia and joint laxity of female anterior cruciate ligament-injured patients. Am J Sports Med 39:410–414
- Yu B, Garrett WE (2007) Mechanisms of non-contact ACL injuries. Br J Sports Med 41(Suppl 1):i47–i51
- Yu B, Lin CF, Garrett WE (2006) Lower extremity biomechanics during the landing of a stop-jump task. Clin Biomech (Bristol, Avon) 21:297–305