

## Hip extension, knee flexion paradox: A new mechanism for non-contact ACL injury

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### Abstract:

Considering that an athlete performs at-risk sports activities countless times throughout the course of his or her career prior to the instance of anterior cruciate ligament (ACL) injury, one may conclude that non-contact ACL injury is a rare event. Nevertheless, the overall number of non-contact ACL injuries, both in the US and worldwide, remains alarming due to the growing number of recreational and professional athletes participating in high-risk activities. To date, numerous non-contact ACL injury mechanisms have been proposed, but none provides a detailed picture of sequence of events leading to injury and the exact cause of this injury remains elusive. In this perspective article, we propose a new conception of non-contact ACL injury mechanism that comprehensively integrates risk factors inside and outside the knee joint. The proposed mechanism is robust in the sense that it is biomechanically justifiable and addresses a number of confounding issues related to ACL injury.

**Keywords:** Anterior cruciate ligament (ACL) | Injury | Mechanism | Quadriceps | Valgus

### Article:

#### 1. Introduction

The mechanics leading to non-contact anterior cruciate ligament (ACL) injury are complex and difficult to monitor. Non-contact ACL injury is most prevalent in the younger population (Griffin et al., 2006), and the negative effects of an ACL tear are lifelong. Patients with torn ACLs are at increased risk of developing osteoarthritis (OA), regardless of whether the ACL is reconstructed (Daniel et al., 1994). As a result, prevention is considered the ideal approach to address the negative consequences of ACL injury. The need for effective prevention strategies requires that emphasis first be placed on how and why these injuries occur, i.e. what is the mechanism of non-contact ACL injury?

In its simplest conception, ACL injury occurs when stress on the ligament exceeds its failure strength (Slauterbeck et al., 2006). However, this simplistic relationship belies the true complexity of the injury as many factors converge to affect both ACL strength and the loads applied to it. Numerous mechanisms of injury have been suggested and studied exhaustively over the past decades. These mechanisms primarily address how injurious loads are produced, and include but are not limited to:

- (i) anterior shear force mechanisms—a major contributor to the anterior shear force is the contraction of quadriceps muscles resulting in significant anterior tibial translation at low knee flexion angles (Berns et al., 1992, Markolf et al., 1995, Fleming et al., 2001, Arms et al., 1984, Draganich and Vahey, 1990, Beynnon et al., 1995 and Demorat et al., 2004),
- (ii) axial compressive load mechanism (Li et al., 1998 and Meyer and Haut, 2005),
- (iii) hyperextension mechanism (Markolf et al., 1990 and Boden et al., 2000),
- (iv) valgus collapse mechanism—owing either to pure abduction of the distal tibia relative to the femur or to tibiofemoral internal/external rotations (Hewett et al., 2005 and McLean et al., 2005; Krosshaug et al., 2007, Shin et al., 2009 and Chaudhari and Andriacchi, 2006; Quatman and Hewett, 2009),
- (v) internal rotation of the tibia (Markolf et al., 2004 and Fleming et al., 2001),
- (vi) combined valgus and anterior shear (Markolf et al., 1995),
- (vii) combined valgus and internal tibial torque (Kanamori et al., 2002),
- (viii) valgus and external tibial torque (Ireland, 1999), and finally,
- (ix) valgus, anterior tibial shear, and axial torque about the long axis of the tibia (Quatman and Hewett, 2009).

The mechanical and/or structural properties of the ACL are not considered important in these mechanisms partly because it is presumed, perhaps precipitately, that little can be done to alter ACL size and strength.

Although many differences exist among the above mechanisms, there are also commonalities. In almost all of the ACL injury mechanism literature (with the exception of Ireland, 1999), sagittal plane hip kinematics are ignored as a direct contributor to ACL loading. ACL loading is often treated as a purely single joint (tibiofemoral) phenomenon. This is evident in an exhaustive review of existing non-contact ACL injury mechanisms by Shimokochi and Shultz (2008) in which the direct contribution of sagittal hip kinetics/kinematics to ACL loading never appears. It is also frequently assumed that excessive muscle-generated forces or torques cause ACL injury, but never the opposite. A lack of adequately protective co-contraction of both knee and hip muscles is seldom considered as a cause of ACL injury, despite being more plausible. Lastly, ACL injury risk factors are frequently disjointed from the injury mechanisms. That is, while mechanisms have been theorized and risk factors identified, few risk factors have been directly implicated in any particular injury mechanism. Viable risk factors must exert influence on either the biomechanical loads placed on the ACL or the resistance to these loads as provided by the ligament.

In this perspective article, we propose a new non-contact mechanism of injury that is inherently different from extant mechanisms and provides a more complete picture of the events leading to

injury. It is important to note that neither are we discounting existing non-contact ACL injury mechanisms, nor are we suggesting that the proposed injury mechanism is the only viable one. We use the perspective article venue to express our thoughts because admittedly we do not have experimental validation for some of our assertions. Nevertheless, this forum affords us the opportunity to express our theories about non-contact ACL injury mechanisms based on our understanding of the biomechanics of the lower extremity. Our hope is that this proposed mechanism will provide a new way of looking at an old problem.

## **2. A proposal for a new mechanism of non-contact ACL injury**

We propose that ACL injury occurs because of the concurrence of specific neuromuscular events, external loads due to ground contact/impact, and certain subject-specific anatomical disadvantages. Landing from a jump will be used as an example, however the proposed mechanism may be applied to all deceleration tasks. The theorized mechanism is that non-contact ACL injury occurs when the following factors converge: (1) delayed or slow co-activation of quadriceps and hamstrings muscles, (2) a dynamic ground reaction force applied while the knee is near full extension, (3) a shallow medial tibial plateau and a steep posterior tibial slope, and (4) a stiff landing due to incompatible hip and knee flexion velocities. The concurrence of the above conditions would lead to an unnatural state in which the hip joint experiences a tendency to extend relative to a knee joint that is forced to flex: this is the so-called relative hip extension-knee flexion paradox which would be highly injurious to the ACL in the presence of the other aforementioned conditions.

Below, we explore our proposed mechanism in more detail and discuss how it addresses a number of questions yet unanswered in the literature.

### **2.1. Delayed or slow co-activation of quadriceps and hamstring muscles**

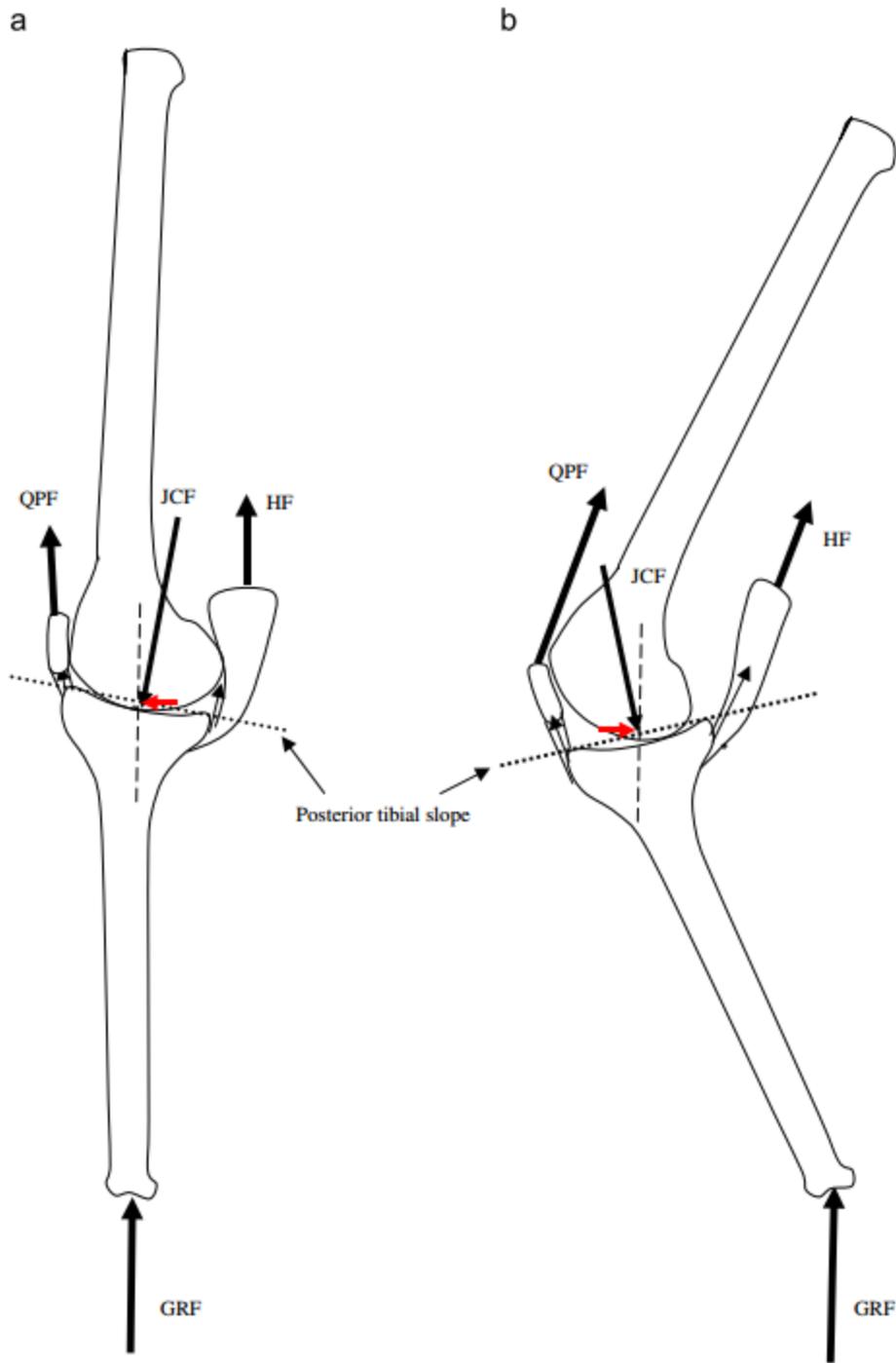
It is well known that co-contraction of the quadriceps and hamstring muscles provides active protection for the knee and its passive restraints (Markolf et al., 1978, Goldfuss et al., 1973 and Wojtys et al., 2002). Appropriate muscle activation creates a condition of ‘active stability’ for the knee (stable control through application of active restraints). We suggest that a loss of active tibiofemoral stability, resulting in increased reliance on passive structures, is a necessary (but not sufficient) condition for ACL injury (Markolf et al., 1978, Goldfuss et al., 1973 and Wojtys et al., 2002). This ‘passive stability’ may occur at a critical time during athletic activity when quadriceps and hamstring groups are either not active, are active but at inadequate levels, or are reflexively silenced due to sudden perturbation. This deficiency in activation results in a joint that is reliant on passive restraint (Louie and Mote, 1987) and also inadequately compressed at the time of ground contact. Lack of muscular protection could occur over a short period of time due to delays in muscle activation. It is reported, for instance, that fatigue could create a delay in knee flexor/extensor muscle activation (Nyland et al., 1994). A delay in co-activation of quadriceps and hamstring muscles is the keystone of the currently proposed mechanism and could occur due to a variety of factors including response to stimulus, tendon tap—a sudden decrease in load against which a muscle is contracting, or a sudden increase in load in an antagonist (Shahani and Young, 1973 and Shultz et al., 2000).

This ‘passive stability’ is similar to the concept of ligament dominance discussed by Hewett et al. (2002) defined as, “the absence of muscle control of mediolateral knee motion, [when the subject] allows the ground reaction force to control the direction of motion of the lower extremity joints, and the ligament to take on a significant percentage of the force.” In our interpretation of Hewett et al.’s ligament dominance concept, the subject always allows, as a matter of conditioned response or perhaps congenitally, dynamic support of loads through exclusive or dominant use of ligaments. In contrast, the ‘passive stability’ referred to here is not conscious or voluntary, and is exclusively due to temporary neuromuscular delay in co-contraction of quadriceps and hamstring muscles at ground contact. We suggest that all participants, male or female, are susceptible to this delay. This distinction is crucial when one considers prevention programs.

## **2.2. Application of an impulsive ground reaction force while the knee is near full extension**

When there is a delay in co-activation of the quadriceps and hamstrings muscles, active restraint is absent or insufficient and the joint will rely primarily on passive restraints. If there is a lack of adequate co-contraction a few milliseconds prior to foot contact, the ensuing ground reaction force becomes the key force in loading the tibia in all planes, specifically the sagittal plane. Dynamic or impulsive ground reaction forces up to 5.7 times body weight have been reported during jump landing in recreational athletes (Seegmiller and McCaw, 2003). Thus, for a 75 kg subject, the dynamic ground reaction force could exceed 4000 N. The ground reaction force will concurrently elicit joint compressive forces, JCFs, of similar magnitude in the medial and lateral tibial compartments. The JCFs are contact forces that, in the absence of friction, act perpendicular to the medial/lateral tibial plateau. Near full extension, the JCFs acting on the posteriorly tilted tibial plateau have a large shear component that induces anterior tibial translation (see Fig. 1a). Here, the sagittal kinematics of the ankle becomes crucial as they influence the magnitude of the ground reaction force. For instance, Boden et al. (2009) suggest that landing in a flat-footed position (reduced plantar flexion) increases ground reaction force.

In the literature, there are numerous assertions that the ACL is the primary restraint against anterior tibial translation at low flexion angles (Grood et al., 1981, Butler et al., 1980, Markolf et al., 1976 and Markolf et al., 1978). If this is true, during landing near full extension, with a delay in co-contraction of quadriceps and hamstring muscles, there is seemingly no protective mechanism to stop anterior tibial translation other than the ACL. However, this begs the question, “If delayed co-activation of quadriceps and hamstring muscles (for instance, due to fatigue) and a dynamic ground reaction force are all that is required for injury, why are higher ACL injury rates not observed?” The answer to the above question is that, according to literature, there are many other mechanisms by which the ACL is protected from injury, even if a deficiency in muscular protection occurs. Several of these protective mechanisms will be discussed in later sections.



**Fig. 1.**

The role of the slope and orientation of the tibial plateau on the direction of the joint compressive force (JCF). (a) At full extension JCF is leaning anteriorly and (b) at moderate flexion JCF is leaning posteriorly. QPF, quadriceps patellar tendon force; HF, hamstring force; GRF, ground reaction force.

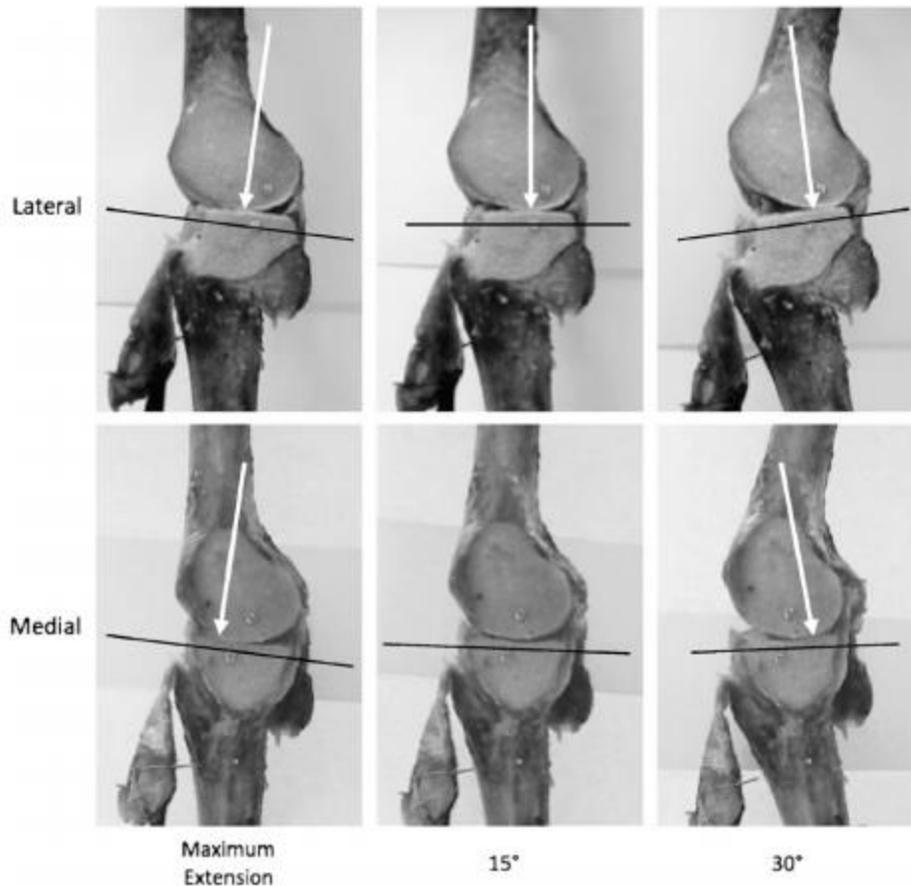
## **2.3. Shallow medial tibial plateau and steep posterior tibial slope**

### **2.3.1. Posterior slope of the tibial plateau and its orientation relative to the femur**

It has recently been established that the posterior slope of the tibial plateau is different on the medial and lateral aspects, and the slope ranges from  $-1^{\circ}$  to  $14^{\circ}$  in normal subjects (Stijak et al., 2008; Hashemi et al., 2008a and Hashemi et al., 2010). The medial slope is schematically represented by the dashed line in Fig. 1a. The corresponding JCF is perpendicular to the surface of the tibia and has an anterior shear component (red arrow). The shear component produces anterior tibial translation in conjunction with the anterior component of the quadriceps force acting through the patellar tendon. Thus, subjects with steeper tibial slope will have a larger shear force resulting in potentially higher anterior tibial translation. On the contrary, subjects with milder tibial slopes may innately avoid excessive anterior tibial translation (mild slopes may be protective).

The protective nature of milder tibial slopes is hidden in the fact that the direction of the JCF changes with knee flexion. We demonstrate this schematically by showing the orientation of the tibial plateau after a certain amount of knee flexion in Fig. 1b. As knee flexion increases, the combined tibial and femoral movements will cause the plateau to angulate behind the femoral condyles. This is represented by the dashed line in Fig. 1b, which now has an anteriorly directed slope relative to the femoral condyles (compare the slope of the dashed lines in Fig. 1a and b relative to the femur). As a result, in Fig. 1b, the JCF, which is again perpendicular the plateau, will be directed posteriorly (compare to JCFs in Fig. 1a and b). This creates a posteriorly directed shear force shown by the red arrow in Fig. 1b, which will resist anterior tibial translation.

To elucidate this further, medial and lateral sagittal image of a cadaveric knee at three different measured flexion angles of  $\sim 0^{\circ}$  (maximum extension),  $15^{\circ}$  and  $30^{\circ}$  are shown in Fig. 2. It is clear that near full extension of the JCF on the lateral aspect of the tibial plateau is tilted anteriorly but this changes to a posterior orientation as the knee is flexed (Fig. 2a). A similar observation can be made on the medial side (Fig. 2b). In this figure, the shift in the contact point between the femur and tibia from anterior, near full extension, to posterior, at  $30^{\circ}$  of flexion is also noteworthy. It is clear from these figures that the corresponding JCFs have very different directions based on the amount of knee flexion. In addition to the change in direction, it is also important to note that the JCF is often significantly larger than the muscle forces during physical activities and could therefore have a large anterior/posterior component. To support this assertion, we refer to two studies: Wilk et al. (1996) reported the generation of posteriorly directed shear forces in flexion angles ranging from  $12^{\circ}$  to  $104^{\circ}$  in squatting and  $18^{\circ}$  to  $104^{\circ}$  in leg press. The large joint compressive forces reported (6139 N in squats and 5762 N in leg press) must be directed posteriorly, as shown in Fig. 2b, to create an overall posteriorly directed shear force in the presence of anteriorly directed patellar tendon (quadriceps) forces. Lutz et al. (1993) report similar findings, showing posteriorly directed shear forces acting on the tibia in closed kinetic chain exercises at flexion angles of  $30-90^{\circ}$ .



**Fig. 2.**

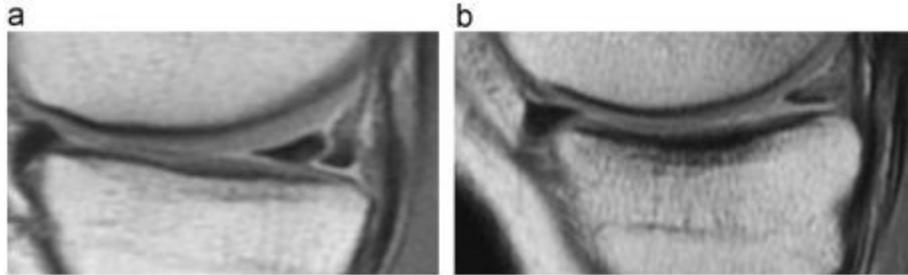
Sagittal images of a knee with shaved lateral and medial condyles: (a) exposed lateral sagittal views at three different flexion angles and (b) exposed medial sagittal views at three different flexion angles. Note that the tibial plateau is directed at substantially different orientations as a function of the flexion angle. White arrows show the approximate JCF directions while the thin dark lines show the approximate orientation of the plateau at the given flexion angles.

Biomechanically, it could be argued that this posteriorly directed component of the JCF plays an equal, if not greater ACL-protective role than the posteriorly directed component of the hamstrings force. Subjects with mild tibial slope will benefit from this protection after very small amounts of knee flexion. On the contrary, subjects with steep tibial slopes will experience this added benefit only after moderate knee flexion.

### **2.3.2. Shallow medial tibial plateau depth**

It has also recently been shown that the depth of the medial tibial concavity may be a more critical risk factor in anterior cruciate ligament injury than the slope (Hashemi et al., 2010). Those subjects with shallow or flat medial tibial plateaus, such as the one shown in Fig. 3a are at 3× greater risk of injuring their ACLs for a 1 mm decrease in the depth of concavity. Deeper plateaus, such as the one in Fig. 3b, provide more stable seating of the medial femoral condyle on the tibial plateau. One could conclude that deeper medial plateaus are protective of the ACL

under weight-bearing because the convex medial femoral condyle will fit snugly with the concave medial tibial plateau (even if muscular protection is insufficient). At low flexion angles this interlocking mechanism, together with the action of the menisci, may act as the primary restraint against anterior tibial translation. Perhaps, it is for this reason that subjects with a flat medial tibial plateau are far more susceptible to ACL injuries than those with deeper medial plateaus (Hashemi et al., 2010).



**Fig. 3.**

Sagittal MRI images of two knees in the middle of articulating region. (a) A knee with a shallow MTD of almost 0 mm and (b) with a deep MTD of approximately 4.5 mm (adapted from Hashemi et al. (2010), AJSM).

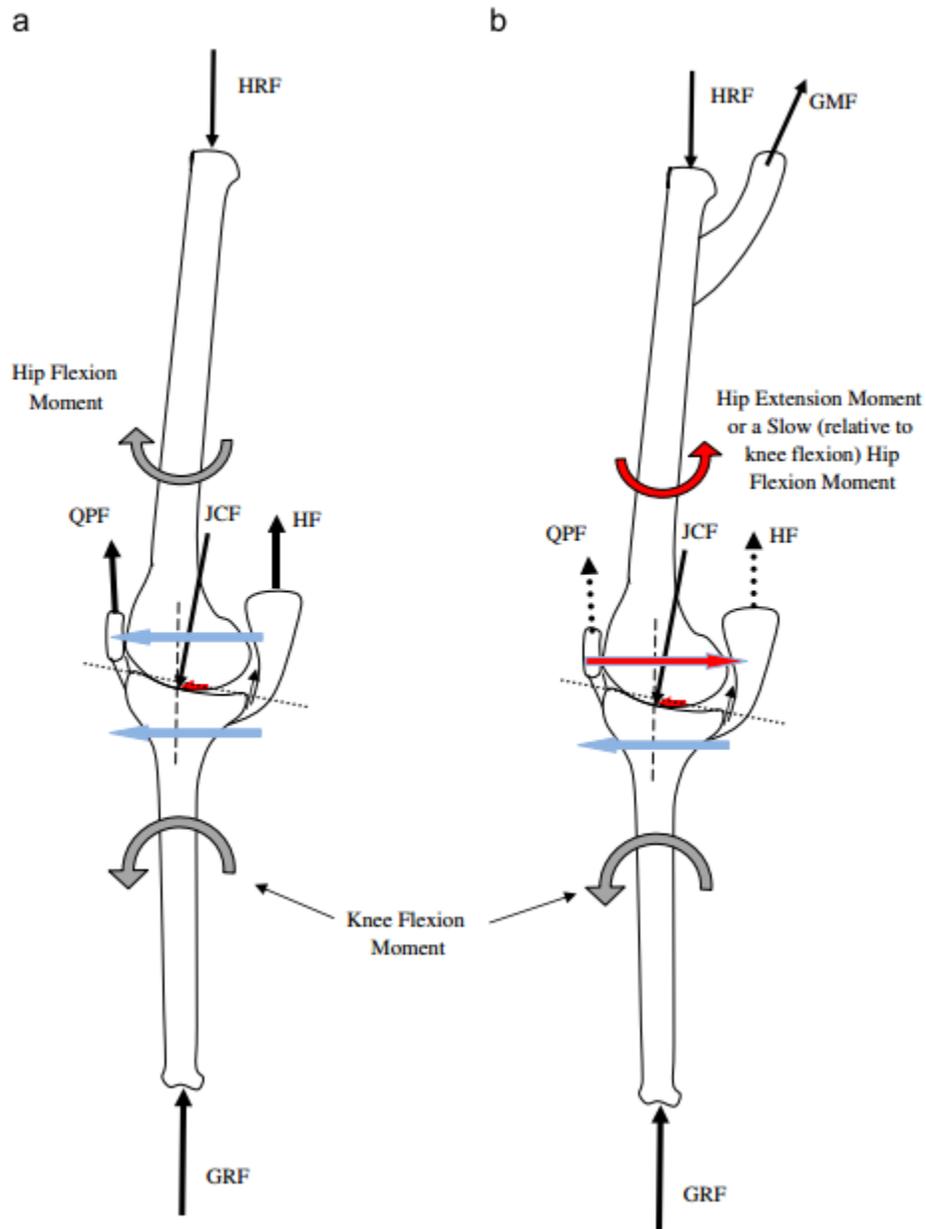
Returning to the discussion of delayed muscular protection, even if a delay in co-activation occurs and the knee is not afforded muscular protection, the ‘locking’ of the femoral condyle in the medial tibial plateau – as occurs naturally, under weight-bearing – may offer robust protection against anterior tibial translation. Therefore, the tibial plateau offers protection to the ACL in two ways: (i) by angulating behind the femur at large flexion angles (Fig. 1 and Fig. 2), and (ii) by providing a locking mechanism between the femur and the tibia at low flexion angles. It is for these reasons that neuromuscular delay in co-activation of the quadriceps and hamstring muscles is a necessary but not sufficient condition for ACL injury.

#### **2.4. A stiff landing due to incompatible hip and knee flexion velocities in the sagittal plane**

Let us now assume that during landing from a jump, a delay in co-activation has occurred, the knee is near full extension, and the subject’s medial tibial plateau is shallow with a steep posterior slope. This results in a supercritical situation in which the ACL *does* act as the primary restraint against anterior tibial translation. However, it might still be argued that far more ACLs should be injured because these conditions likely occur regularly in sport. Is there another protective mechanism? The answer is yes. According to basic kinematics, if the existing neuromuscular and anatomical states allow the knee and the hip to flex together through harmonious co-flexion of the hip and knee, then anterior translation of the tibia relative to the femur will remain low leading to low ACL loads. The question now becomes, “What could prevent this harmonious co-flexion of the hip and the knee?”

During a jump landing activity – the activity that causes the highest percentage of ACL injuries – the force that works to flex both the knee and hip is the ground reaction force. This force is also related to the magnitude of the joint compressive force. In addition to the ground reaction force, another factor that influences knee flexion is the ability of the hip to flex. During foot contact,

the ground reaction force causes the knee to flex (gray CCW arrow on tibia in Fig. 4a), and the ensuing joint compressive force at the knee will force the hip to flex (gray CW arrow in the femur in Fig. 4a). Under normal muscle activation, because these flexions are mutually dependent, one cannot separate knee flexion from hip flexion; as the hip flexes, so does the knee. This co-flexion is protective because it assures that the tibia and the femur can harmoniously roll and glide on each other in the direction shown by light blue arrows in Fig. 4a.



**Fig. 4.**

Hip extension knee flexion paradox. (a) Normal condition at full extension, neuromuscular control assures that both knee and hip joints flex together and (b) abnormal condition under which knee is forced to flex but hip is forced to extend. HRF, hip reaction force; GMF, gluteus maximus force. (not a free body diagram).

Under the conditions described above, when co-flexion of the hip and the knee is interrupted or impaired, i.e., the knee will flex (gray CCW arrow on tibia in Fig. 4b) but the hip flexes more slowly than the knee (red CCW arrow on the femur in Fig. 4b), the tibia will undergo anterior translation, resisted solely by the ACL. This can happen while landing from a jump or during plant-and-cut maneuvers. To reiterate, when there are temporarily insufficient muscle forces to resist anterior tibial translation and the hip is not flexing as fast as required by the knee, the only remaining possibility is for the tibia to translate anteriorly relative to the femur (see opposing blue arrows in Fig. 4b); the ACL will most likely fail. However, if the hip co-flexes with the knee at an acceptable rate, even though the knee is passively stable, the relative movement between the tibia and the femur will be reduced and the ACL will remain safe.

The question that is immediately raised is, “What could keep the hip from flexing at an appropriate rate (or even cause it to extend) during foot contact?” One possibility is that, in our scenario, the quadriceps and hamstring muscles have delayed co-activation and there is instability; the only remaining major hip extensor is the gluteus maximus. Isolated and strong application of the gluteus maximus (see Fig. 4b), may keep the hip from flexing at a proper rate or perhaps encourage hip extension (Hashemi et al., 2007). This paradoxical situation is not observed during normal athletic activities and can only happen when co-activation is compromised. Yu et al. (2006), in an especially noteworthy study, suggest that the rate of hip flexion, and not hip flexion angle at landing, influences ACL loading. This is a thought-provoking conclusion that in part led to the proposed mechanism.

A second possibility that may create a mismatch between hip flexion and knee flexion is the position of the trunk at the time of ground contact. In a situation where the trunk is upright or leaning backward at the time of ground contact, the center of mass (COM) will be positioned posterior the knee, and the ensuing increase in ground reaction force will likely encourage more knee flexion than hip flexion, and may act to extend the hip. Conversely, a more forward leaning trunk (COM anterior the hip), is more likely to encourage both hip and knee flexion (Shimokochi et al., 2009 and Koyanagi et al., 2006). Further, Kulas et al. (2008) compared changes in hip and knee kinematics when a load was applied to the trunk in individuals who adopted a more extended trunk posture to those who adopted a more flexed trunk posture during landing. They reported a decrease in peak hip flexion and angular impulses in response to the applied load in those who adopted a more extended trunk. Those who adopted a more flexed trunk experienced increases in peak hip flexion and angular impulses. Their findings in the trunk-flexed group versus extended group are consistent with those of Blackburn and Padua, 2008 who reported greater relative increases in hip flexion versus knee flexion both at initial contact ( $3^\circ$  versus  $6^\circ$ ) and at peak loading ( $22^\circ$  versus  $31^\circ$ ) when subjects performed a controlled drop landing with the trunk more flexed as compared to their preferred landing strategy. Reduced hip flexion, when combined with greater knee flexion, has been shown to be predictive of greater anterior shear forces during the initial landing phase of a drop jump (Shultz et al., 2009). This suggests that the relative trunk position at the ground contact has the potential to either reduce (forward trunk) or facilitate (extended trunk) a hip and knee flexion dysynchrony and influence risk of ACL failure.

### **3. Discussion**

With the simultaneous occurrence of the four conditions, listed above, we have proposed a non-contact ACL injury mechanism that is biomechanically justifiable and plausible. The proposed mechanism is different from current ACL injury mechanisms in the following ways: (i) the injury-causing force is strictly the ground reaction force and not excessive muscle forces or torques, (ii) the mechanism considers and explains the concurrent kinematics of the hip and knee, and (iii) it connects variables external to the knee itself – muscle forces, ground reaction force, fatigue – to the structure of the knee (tibial plateau geometry). We believe that the proposed mechanism is viable because it answers a number of outstanding questions as follows.

#### **3.1. There is a clear inciting event**

Considering that athletes perform at-risk activities including landing from a jump, lateral pivoting, and deceleration activities countless times during their careers, why does the ACL fail only at a particular instance? Any suggested mechanism must have a persuasive answer to this question. In our mechanism, the ‘inciting event’ is a delay in co-activation of the quadriceps and hamstring muscles due to fatigue or other factors. None of the existing ACL injury mechanisms offers a clear explanation of the inciting event.

#### **3.2. The timing requirements for ACL injury are met**

It has been reported that ACL injury occurs between 17 and 50 ms after initial contact (Krosshaug et al., 2007). A major distinguishing feature of our mechanism is that it is not based on an excessive force or torque concept. For instance, an excessive quadriceps force of 4500 N (DeMorat et al., 2004), a valgus torque of 125–210 N m (Seering et al., 1980), or an internal rotational torque of 35–80 N m (Seering et al., 1980) are needed to induce ACL rupture in the sagittal, coronal, and transverse planes, respectively. Can such excessive levels of force and torque be generated in the 17–50 ms time period after impact? The answer to this question is not yet clear. In-vivo studies that measure the temporal changes in the ground reaction force, angular accelerations and velocities of the femur and the tibia, and coronal position of the tibiofemoral joint are needed to determine the time history of valgus and internal tibial torques.

Can our mechanism produce ACL injuries in the proposed time frame of 17–50 ms after initial contact? Since our mechanism requires only a delay in muscular activation and suggests that it is the ground reaction force that is loading the ACL, then the timing of the peak ground reaction force should be consistent with the actual timing of the injury. According to Seegmiller and McCaw (2003), during landing from a jump in recreational athletes, the first peak of the ground reaction force ranging magnitude from just under one body weight (BW) to around 2.4 BW occurs at times ranging from 10 to 18 ms after first contact. The second peak in the ground reaction force is of magnitudes ranging from just under 2.2 BW to around 5.7 BW and occurs at times ranging from 42 to 65 ms after initial contact. These findings show that both the magnitude and timing of the ground reaction force are consistent with causing non-contact ACL injury in the reported 17–50 ms window after initial contact. Based on our review of the literature, none of the existing non-contact ACL injury mechanisms discuss this constraint.

### **3.3. The contribution of passive knee laxity to the injury mechanism is explained**

It is generally believed that passive knee laxity (anterior–posterior, internal–external, and varus–valgus measured under non-weight bearing conditions) plays a role in ACL injury (Woodford-Rogers et al., 1994 and Uhorchak et al., 2003). However, exactly how knee laxity – measured under *non-weight-bearing* conditions and *minimal muscle forces* – contributes to various existing non-contact ACL injury mechanisms, *under weight-bearing* and *excessive muscle forces*, is not well-explained.

In our proposed mechanism, knee laxity plays a crucial role because of the delay in co-activation of the quadriceps and hamstring muscles. A knee with inherently high levels of laxity will certainly be more susceptible to anterior tibial translation in the absence of muscular protection. Passive anterior and rotational laxities will allow greater joint movements (femur relative to the tibia) and these large movements could compromise the relative positioning of the femur and tibia in the articulating region to the “point of no return (Ireland, 1999)”. In a general sense, laxity should only matter when muscular protection is not offered or its onset is delayed. Thus, in our mechanism, laxer knees will almost certainly be at higher risk of ACL injury.

### **3.4. The abnormal knee kinematics (mostly valgus collapse) during ACL injury can be explained**

It is frequently observed in video evidence that during ACL injury, the knee of the subject undergoes abnormal kinematics including extensive or abnormal valgus and internal/external rotations (Krosshaug et al., 2007). This makes a case that, in fact, valgus is a viable mechanism of ACL injury. How do we delineate the often observed valgus collapse in relation to our proposed mechanism?

The first explanation may be that the valgus collapse occurs after the ACL has already been injured (Yu and Garrett, 2007). The medial and lateral slopes of the tibial plateau may be substantially different within a subject (Stijak et al., 2008 and Hashemi et al., 2008a). For instance Hashemi et al. (2008a) report that the medial and lateral tibial slopes may be classified into three categories: (1) medial and lateral slopes within the subject are similar in 21% of the subjects ( $1^\circ$  or less of difference); (2) the lateral slope within the subject (LTS) is steeper in 55% of the subjects ( $2^\circ$  or more of difference); and (3) the medial slope within the subject (MTS) is steeper in 23% of the subjects ( $2^\circ$  or more of difference). We believe that once ACL injury has commenced due to the proposed mechanism, subjects in category 1 (similar MTS and LTS) will exhibit a purely translational (anterior) mode of injury. Subjects in category 2 (LTS steeper than MTS) will be more susceptible to internal rotation of the tibia and valgus-related kinematics (McLean et al., 2010). Subjects in category 3 will be more susceptible to external rotation of the tibia and valgus-related kinematics. In other words, the valgus collapse, either accompanied by internal or external rotation, is simply a side effect of ACL injury and the geometry of the tibial plateau.

Second, the valgus mechanism may be a viable ACL injury mechanism causing direct injury to the ACL. However, a delay in co-contraction of the quadriceps and hamstring as well as a delay in hip abductor activation is needed for a valgus-collapse theory to work. A delay in co-

contraction of the quadriceps and hamstrings is necessary to make the tibiofemoral joint lax and a delay or weakness in hip abduction muscles is needed (no hip abduction muscle means more hip adduction and thus knee abduction). Under this situation, there is no resistance against valgus kinematics and the ACL will tear, likely accompanied by extensive damage to other soft-tissues. One important additional point is that the location of foot contact must be substantially lateral to the axis of the body to create the high external valgus moments needed for ACL injury (Hewett et al., 2009). A similar mechanism was offered by Ireland (1999), where the author proposed the “point of no return,” after which valgus collapse occurs.

Lastly, the addition of an external knee abduction moment could exacerbate our proposed mechanism by effectively reducing the ‘interlocking’ of the medial plateau and condyle. When a knee abduction moment is present, a greater share of the contact force is carried by the lateral compartment, which is less stable due to this convex-on-convex interaction. Less of the contact force is carried by the medial compartment thus limiting the ability of the convex-on-concave medial tibiofemoral articulation to act as a constraint on anterior translation. This will superimpose excessive anterior tibial translation with mild or moderate valgus movement.

#### **4. Implication to sex-based disparity in ACL injury rates**

It is also of interest to speculate as to how this mechanism of injury might delineate the sex-based disparity in ACL injuries. Notwithstanding any anthropometric differences between sexes, as well as any size and strength differences in the ACL itself, according to our proposed mechanism, subjects possessing a union of certain anatomical and neuromuscular characteristics will be more susceptible to knee instability, hip extension-knee flexion paradox, and possibly ACL injury.

These characteristics are as follows:

- (1) The anatomical features that would expose the subject to (i) increased disruption in the knee/hip co-flexion and (ii) reduction in intrinsic protection of the ACL.
  - The steeper tibial slope in females (Hashemi et al., 2008a) will produce larger anterior shear components of the JCF and more importantly, will require greater knee flexion for the orientation of tibial plateau to become protective of the ACL (see Fig. 1).
  - The reported knee joint anterior/posterior laxity of female subjects may predispose females to ACL injury under inadequate muscular protection (Rozzi et al., 1999 and Boden et al., 2000).
- (2) Fatigue (Nyland et al., 1994) and neuromuscular factors would result in delayed activation or co-contraction of quadriceps and hamstring muscles in the subject. Furthermore, Moore et al. (2002) suggest that females have a decreased ability to stiffen the joint in response to mechanical perturbations when fatigued.
- (3) Lower muscle stiffness in women (Granata et al., 2002) could potentially produce a slower ascent in protective muscle load (Kibler and Livingston, 2001).
- (4) ACL-injured females, as suggested by Hewett et al. (2005), showed more valgus alignment and significantly higher ground reaction forces over a shorter stance time (the definition of a stiff landing).

Finally, even under the rare circumstance that an ACL is exposed to an injurious load, it may yet resist failure if it has a sufficient size (length, diameter, and volume) and strength. A female's smaller ACL size (Chandrashekar et al., 2005), lower ACL strength (Chandrashekar et al., 2006), and less-efficient ACL ultrastructure (Hashemi et al., 2008b) may collectively be a final disadvantage in leading to this injury. Indeed, additional study will be required to determine whether sex influences susceptibility to injury primarily by determining ACL size and strength or rather by affecting the likelihood that an injurious load will be applied.

### **Conflict of interest statement**

None of the authors report any conflict of interest.

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