Determination of Ligament Strain During High Ankle Sprains due to Excessive External Foot Rotation
in Sports

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Abstract   High ankle sprains represent a severe injury in sports. External foot rotation is suspected in these cases, but the mechanism of injury remains unclear. The objective of the current study was to integrate *in vitro* and *in vivo* experiments along with computational models based on rigid bone surfaces and deformable ligaments of the ankle to investigate the external foot rotation injury mechanism with different shoe constraints and ankle positioning. Injuries and the highest strains occurred in the anterior deltoid ligament (ADL) when the foot was held in neutral with athletic tape. Similarly, ADL strains were highest when a football shoe design with a high rotational stiffness was used to constrain the foot. For a flexible shoe, the anterior tibiofibular ligament (ATiFL) strain was increased and ATiFL injury occurred due to increased talar eversion. In human subjects performing a similar movement, the highest strains also occurred in the ATiFL and ADL. The models showed that ATiFL strain was positively correlated with ankle eversion, but eversion decreased strain in the ADL. Finally, the consequence of eversion on ATiFL strain was confirmed in the first cadaver study that consistently generated high ankle sprains in the laboratory.

Keywords   ankle joint, external rotation, injury mechanism, ligament strain, syndesmosis sprain.

I.   INTRODUCTION

Acute ankle sprain is the most common injury in sports [1] and accounts for 10%-30% of sports injuries [2]. Injury to the distal tibiofibular syndesmosis ligaments is commonly referred to as a high ankle sprain or syndesmotic ankle sprain. The distal tibiofibular syndesmosis is comprised of the anterior and posterior tibiofibular ligaments (ATiFL and PTiFL) and the interosseous ligament (IOL) (Fig. 1). Injury to the anterior deltoid ligament (ADL), which consists of the tibionavicular ligament (TiNL) and the anterior tibiotalar ligament (ATiTL) (Fig. 1), is known as a medial ankle sprain. While most ankle sprains are lateral ankle sprains, high and medial ankle sprains are typically more severe. High ankle sprains result in more time off the field [3], require a longer recovery period [4] and a different treatment [5], and often cause chronic ankle dysfunction [6]. In a normal population, high ankle sprains occur in less than 11% of ankle sprains [5], while in professional football the incidence of this more severe type of ankle sprain is approximately 20% [7]. High ankle sprains have historically been under-diagnosed, but more recently a heightened awareness in sports medicine has resulted in more frequent diagnoses of high ankle sprains [8].

The ATiFL is the main structure of the ankle joint preventing excessive fibular movement and external rotation of the talus [9]. In the clinical literature, high ankle sprains are mostly attributed to external foot rotation [4]. However, other studies have indicated that the mechanism of injury involves a combination of dorsiflexion, eversion and external rotation [3],[7]. Although numerous biomechanical studies have been conducted to investigate ankle injuries, most of them are designed to study ankle fractures rather than sprains. Recently, there have been injury mechanism studies of lateral ankle sprain in laboratory settings [1],[10] and based on investigation of game films [11], which have provided insights into the mechanisms of lateral ankle sprains. On the other hand, few biomechanical studies have been conducted on high and medial ankle sprains.

Excessive rotational traction between the shoe and playing surface has been identified as a risk factor for lower extremity injuries [12],[13]. Synthetic turfs generally produce higher ankle joint torques than natural grasses [14], implying an increased risk of injury on artificial turf surfaces. In order to limit or mitigate ankle injuries, it is important to have a good understanding of the intrinsic (age, sex and anthropometry) and extrinsic (shoe/surface traction, bracing and environment) risk factors of a particular athlete, as well as an understanding

of the inciting event (injury mechanism) associated with the onset of injury [15]. Injury mechanisms can be studied with various approaches [16], including; cadaver, surrogate, *in vivo* and *in silico* (computer simulation) (Fig. 2). This "multifaceted, multidisciplinary, integration of *in vivo*, *in vitro* and *in silico* methods" has been recently advocated for the topic of determining the mechanism of injury for anterior cruciate ligament sprains [17].
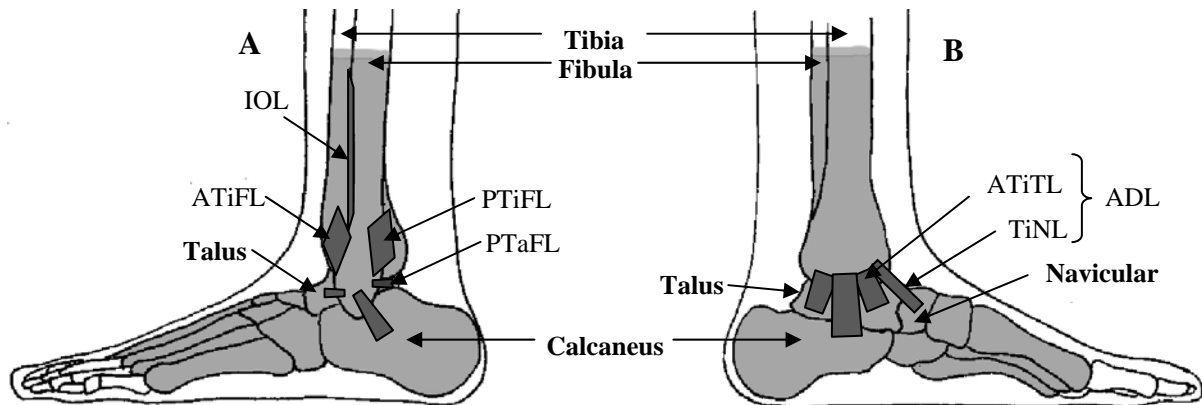

Fig. 1. Lateral (A) and medial (B) views of selected ankle anatomy. Bones are highlighted in bold and ligament abbreviations are as follows; IOL: interosseous ligament, ATiFL: anterior tibiofibular ligament, PTiFL: posterior tibiofibular ligament, PTaFL: posterior talofibular ligament, ATiTL: anterior tibiotalar ligament, TiNL: tibionavicular ligament, ADL: anterior deltoid ligament.
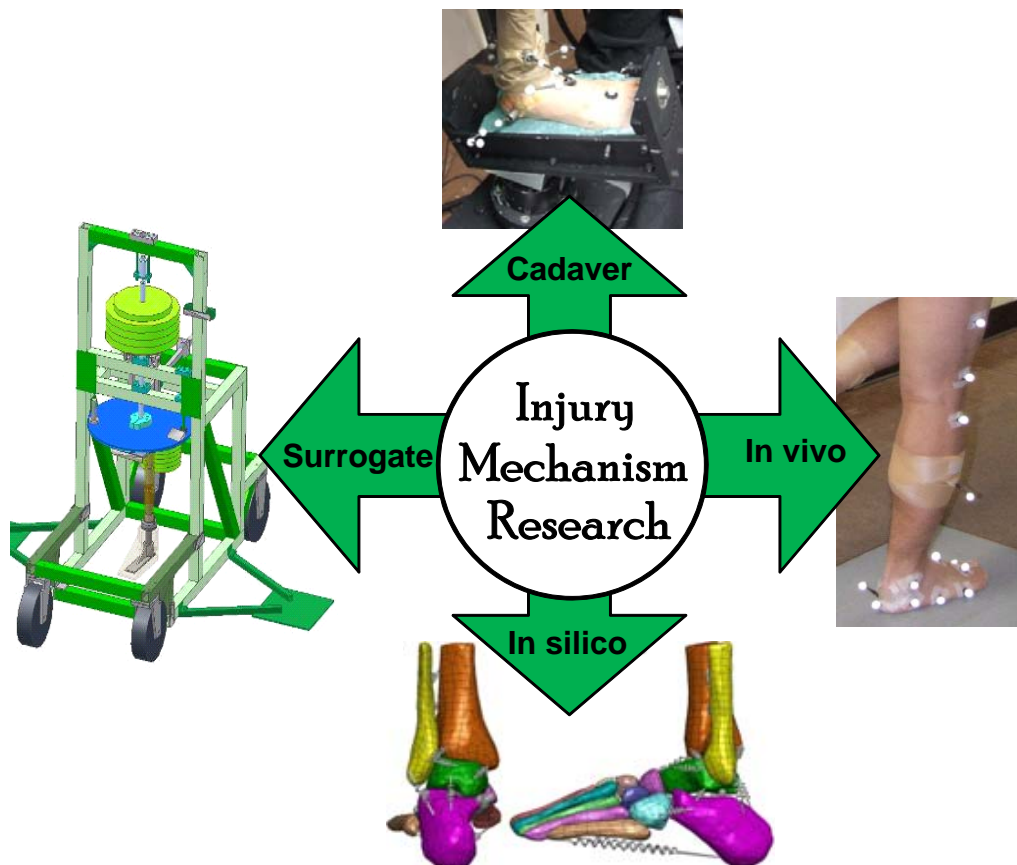

Fig. 2. Research approaches for studying sports related injury mechanisms.

Although surrogate devices can be used to measure the shoe-surface torque for many different conditions [14], one disadvantage of such studies is that artificial devices are used to represent the ankle. Consequently, it is not possible to determine ligament strains, which is more realistic in determining ankle injury [18]. The advantage of human cadaver tests is that forces and motions can be controlled and measured with great precision [19]. Dissection after testing documents the ligament and bone injuries. On the other hand, cadaver studies may not represent injury events in living humans. Computer simulation of musculoskeletal joints and

limbs can also provide useful information about joint mechanics. Validated models can be predictive tools to understand normal joint function and serve as clinical tools for predicting and helping prevent sports injuries. While finite element models yield useful information about stresses and strains in bones and ligaments [20],[21] an advantage of rigid multi-body models is the ability to rapidly solve for motion-based mechanics in large structures [22]. Liacouras and Wayne [23] developed a 3D computational approach to model the lower leg in order to simulate cadaver ligament sectioning studies of syndesmotic injury and ankle inversion stability. A recent study [24] followed a similar approach to develop a computational ankle model based on a generic computed tomography (CT) scan and validated it against two well-documented cadaver studies for measurements of ankle ligament strains [25] and ankle joint torques [26]. Simulations of *in vivo* human movements using computational models also offer a way to solve for motion-based mechanics and determine ligament strains.

The objective of the current study was to use multiple research approaches (Fig. 2) to investigate the external rotation ankle injury mechanism with different shoe constraints and ankle positioning in order to achieve a more comprehensive understanding of the relationships between joint biomechanics and the observed injury mechanism. The hypothesis was that, when real-world kinematics of the ankle from sports scenarios were applied to cadaver and human subjects, ligament strains could be predicted using a validated ankle computer model whose results would correlate with ankle injuries in the form of either high (ATiFL) or medial (ADL) ankle ligament sprains.

II. METHODS

Experimental Testing

The effect of foot constraint was investigated in cadaver ankles, either by restraining the calcaneus tightly with potting and screws [26] or loosely with athletic tape [27]. Dynamic external rotation experiments were conducted on lower limbs from seven male cadavers (age 40±11 years). The tibia and fibula were sectioned 15 cm distal to the center of the knee and the proximal ends of each shaft were potted in a rectangular aluminum sleeve. The foot was everted to the maximum extent of the range of motion (10-15°) in all tests. A biaxial testing machine was used to perform the test with the axis of rotation aligned at the ankle center between the medial and lateral malleoli. Compressive preloads of two to three times body weight were applied through the tibia prior to the application of an external torque. A dynamic angular rotation was input with a position controlled waveform. The rotation was increased by increments of five degrees in successive tests until gross ligament injury or bone fracture was observed based on an audible "pop" and corresponding drop in the torque signal. Between successive tests, the ankles were checked for damage and the orientation of the foot was reviewed. After failure, the mode of injury, failure torque and foot rotation were documented for each specimen. Motion analysis of the talus during external rotation was performed on four pairs of ankles using a Vicon motion capture system (OMG plc., Oxford, UK).

The effect of shoe rotational stiffness (determined to be the rate at which torque is developed under external shoe rotation) was investigated in a second cadaver experiment by applying external rotation to the foot using a football shoe with a flexible or a rigid rotational stiffness design [28]. This experiment was based on interesting rotational traction data from various shoe-surface interfaces using a surrogate ankle designed to have a biofidelic external rotation response [29]. Briefly, four football shoe designs were tested and compared in terms of rotational stiffness. Then lower limbs from six additional male cadavers (age 56±12 years) were externally rotated 30 deg using two selected football shoe designs with the most flexible and rigid responses. Motion capture was performed to track the movement of the talus with a reflective marker array screwed into the bone (Fig. 3). A compression pre-load (1500 N) and a pre-torque (2 Nm) were applied along the axis of the tibia. Internal tibial rotations (external foot rotations) of 30° were input in position control at a frequency of 1 Hz (0.5 seconds to peak rotation). Maximum torques were documented for each specimen.

An *in vivo* study was performed to document kinematics and kinetics of human subjects during a scenario that was comparable to the cadaver experiments [30]. Briefly, six male subjects (age 27±6 years; body mass 81.2±17 kg) with no history of lower extremity injury were recruited. After obtaining informed consent, the subjects stood with their right foot planted on the force plate (Fig. 4) and performed a single-legged internal rotation of their body (external rotation of the foot) at a self-determined maximum effort. A Vicon motion capture system was used to capture motion at 100 Hz from a marker set defined by the "Oxford Foot Model" (OFM). An AMTI force plate (Advanced Medical Technology, Inc., Watertown, MA) captured ground reaction

data at 1000 Hz. The hind foot motion relative to the tibia was obtained from four trials using commercial kinetic (Plug-in Gait, OMG plc., Oxford, UK) and kinematic (Dynamic OFM) packages.

Fig. 3. Human cadaver ankle mounted in a servo-hydraulic testing machine. Vicon motion capture was used to track motion of the talus [28].

Fig. 4. Marker set of the Oxford Foot Model. The subjects performed a single-legged, external rotation of the foot, i.e. internal rotation of the body [30].

The effect of eversion was investigated in a final cadaver experiment by applying external rotation to ankles either in an everted or neutral position [31]. This experiment was based on a preliminary video analysis of high ankle sprains in collegiate football [32]. The injury mechanism was identified as dorsiflexion and eversion of the foot by a blow to the lateral aspect of the shank and knee while the player's foot was planted in external rotation. Lower limbs that had previously been used in the shoe stiffness experiments underwent external foot rotation. All limbs were positioned in 20° of dorsiflexion and restrained with elastic athletic tape. Right limbs were in neutral while left limbs were everted 20°. Talus motion relative to the tibia was measured using motion capture. Dynamic external rotation (1 Hz) was applied with repeated increments of 10° until gross failure. The mode of injury, failure torque and foot rotation were documented for each specimen.

Computational Modeling

A generic three-dimensional, multibody computational ankle model was developed based on CT scans and validated in a previous study [24] for measurements of ankle ligament strain under physiologic loads [25] and ankle joint torques at failure [26]. Briefly, the model was created based on Digital Imaging and Communications in Medicine (DICOM) files from a computed tomography (CT) scan of a cadaveric ankle with a voxel resolution of 0.6 mm. The CT images were imported into Materialise's Interactive Medical Imaging Control System (MIMICS) (Materialise, Ann Arbor, MI). MIMICS was used to eliminate all soft tissues from images, yielding a three-dimensional surface model of the bones as Stereolithography (STL) files for export. Each bone was then imported into SolidWorks (Trimech Solutions, LLC, Columbia, MD) as mesh files where they were further constructed and the bone surfaces were simplified using the ScanTo3D package. SolidWorks Motion, a dynamic rigid-body motion simulation software was used to assemble the bones, obtain proper positioning, add ligaments (modeled as springs), and add 3D surface contacts between bones (Fig. 5). The tibia was fixed in space while the fibula, calcaneus, and talus were free to move and the rest of the bones were fused together and moved as a rigid body. This ankle model includes 21 ligaments formulated as linear elastic springs with properties adapted from the literature [23],[24]. An initial strain of 2% (implemented by inserting a spring element with a length that was 2% shorter than the distance between insertion points) was assigned to most ankle ligaments (0.5% was used for the interosseous ligaments), thereby inducing *in situ* preloads in the ligaments [23]. A model sensitivity analysis has been conducted [24] to ensure that moderate ligament stiffness variations do not significantly alter model conclusions. Talus motion data were used to drive the computational ankle model to predict ligament strains, defined in percentage as the relative elongations of ligaments that occurred during each of the experiments listed in the previous section.
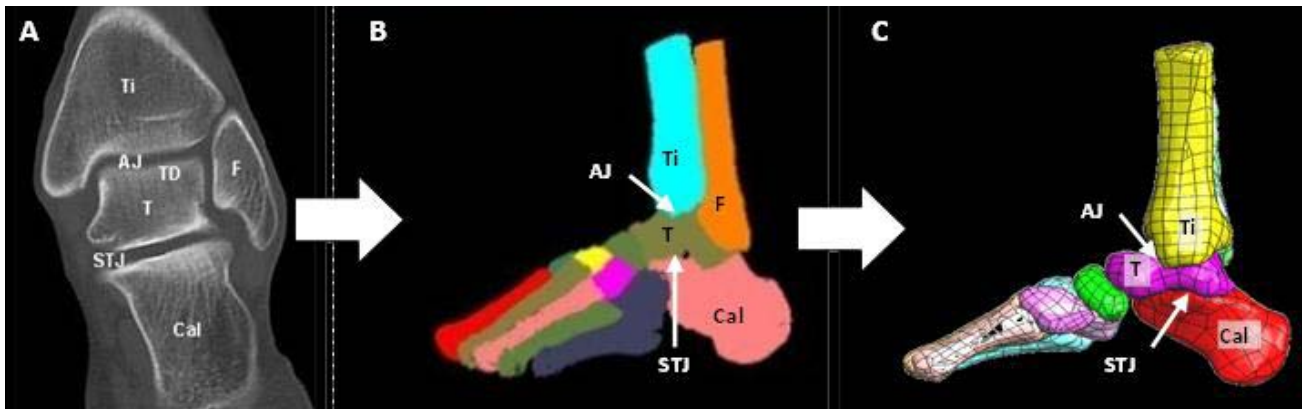
Fig. 5. CT scan of an ankle (A), selected bone masks in MIMICS (B), and meshed ankle assembly in SolidWorks (C). Key: Tibia (Ti), Ankle joint (AJ), Talar dome (TD), Talus (T), Sub-talar joint (STJ), Fibula (F), Calcaneus (Cal).

III. RESULTS

In the foot constraint experiments, the mean failure torque of the ankles with a potted foot was 69.5±11.7 Nm at an angle of 40.7±7.3° of external rotation. The 78.5±18.1 Nm failure torque of the taped ankles was slightly higher and the external rotation angle of 65.5±10.7° was significantly increased over the potted ankles. Talar motions relative to the fixed tibia for the two groups were compared at a pre-injury level of 35° external foot rotation. Similar external rotation of the talus occurred between the potted and taped ankles (33.7±0.5° and 32.7±1.8°, respectively), but there was 4.7±2.5° of talar dorsiflexion and 0.5±0.8° of talar inversion for the potted foot and 0.7±3.1° of talar plantar flexion and 2.5±4.1° of talar eversion in the taped ankles. The computational models predicted the highest ligament strain in the PTaFL for the potted ankles, while the strain was higher in the ATiTL for the taped ankles (Fig. 6). These results paralleled the cadaver injuries, where damage to the PTaFL was the primary injury in potted ankles and ATiTL damage occurred in taped ankles. The ATiFL strain was less than 10% in both models and did not show any gross injury experimentally.
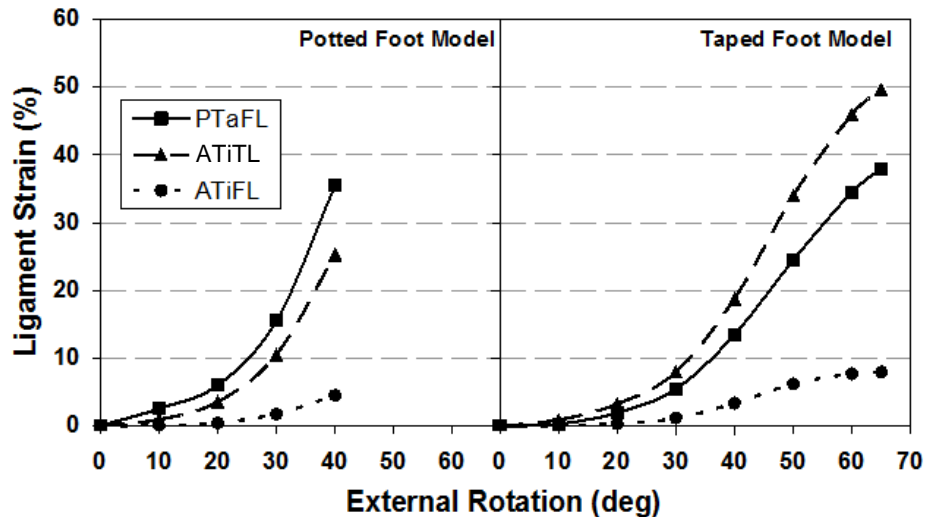

Fig. 6. Ligament strain during external foot rotation of the potted and taped models [24].

In the shoe stiffness experiments, a pre-injury level of 30° external foot rotation applied to either a flexible shoe (Nike Air; rotational stiffness of 21.9±2.8 Nm/deg) or a rigid shoe (Nike Flyposite; rotational stiffness of 50±1.7 Nm/deg) produced maximum torques of 35.4±5.7 Nm and, 46.2±9.3 Nm, respectively. The talus motions were also significantly different between shoe designs, with 12.1±1.0° talar external rotation in the flexible shoe and 15.9±1.6° in the rigid shoe. There was also 5.6±1.5° of eversion in the flexible shoe, but only 1.2±0.8° in the rigid shoe. The ADL strain (average of the ATiTL and TiNL strains) was similar between the flexible (4.9±0.4%) and rigid (5.2±0.7%) shoes, but the ATiFL strain was significantly higher for the flexible (4.5±0.4%) versus rigid (2.3±0.3%) shoes due to the increased talar eversion observed during external rotation of the foot.

In the *in vivo* study the hind foot of subjects had average peak motions of 27±7° dorsiflexion, 12±2° eversion and 29±1° external rotation (Fig. 7). The motion data was used to drive a computational model simulation for

each subject. The ankle joint moments calculated from the Oxford Foot Model and from the computational simulation compared well in all three directions (Fig. 7). Strain in the ATiFL was the highest (9.2±1.1%), followed by the ADL (7.8±0.8%). Those strains were both significantly higher than all other ligaments, including the PTaFL (4.2±0.5%) and the PTiFL (3.6±0.7%). Strain in the ATiFL started earlier than in the ADL as the ankle joint everted and dorsiflexed, but ATiFL strain peaked at the same time as external rotation (Fig. 8). Dorsiflexion and eversion did not influence the strain in the ADL.
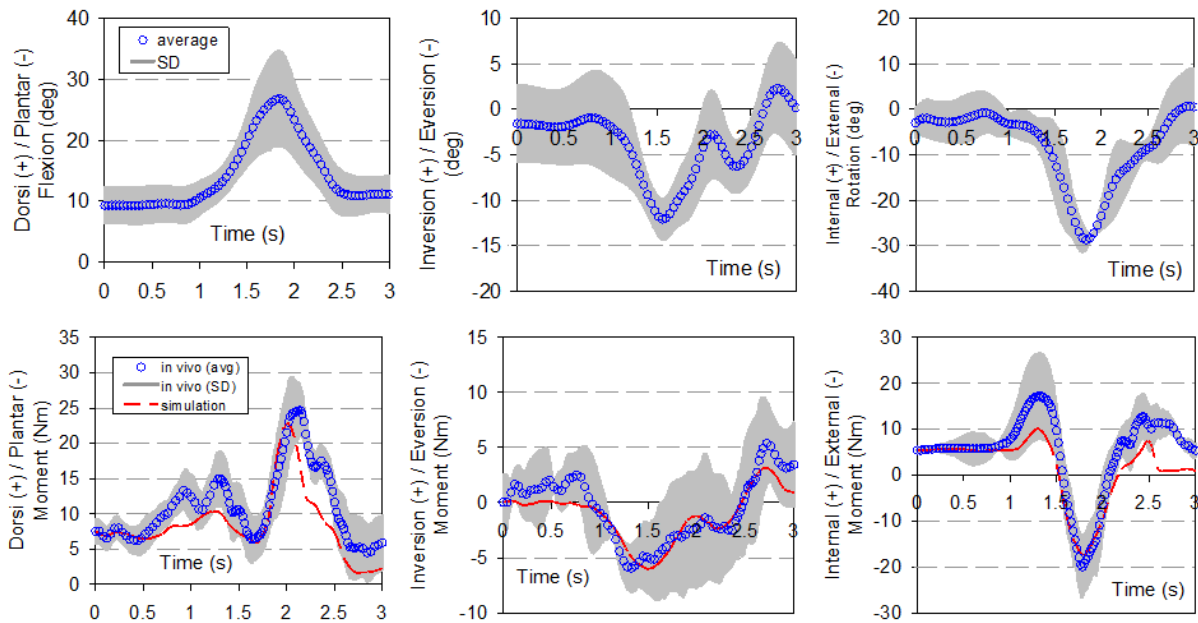

Fig. 7. Hind foot kinematics determined from *in vivo* tests and comparisons of ankle moments between *in vivo* tests and model simulation [30].

In the cadaver eversion experiments, the mean failure torque of ankles in a neutral position was 84.4±16.9 Nm at 37.7±5.4° of external foot rotation, while the failure torque for ankles with 20° of eversion was 90.3±8.6 Nm at an external rotation angle of 46.8±6.1°. The failure torques were similar, but there was significantly more external rotation in the everted ankles than in the neutral ankles at the point of injury. At failure, the estimated strains in the neutral ankle were 31.5% strain in the ADL and 20% strain in the ATiFL. When the ankle was everted, the strain in the ATiFL was the highest at 32%, followed by the ADL with 24.5% strain. These results paralleled the cadaver injuries where neutral limbs demonstrated deltoid ligament ruptures with one ankle having combined damage with the ATiFL. The everted limbs showed only ATiFL injuries. Subsequent parametric analyses of increasing talus eversion for 30° of external rotation showed a negative correlation with ADL strain, while ATiFL strain increased with higher levels of talus eversion. Interestingly, the ligament having the maximum strain changed at 8.5° of eversion, suggesting a possible threshold of talus eversion that predicts the location of ligament injury (Fig. 9).
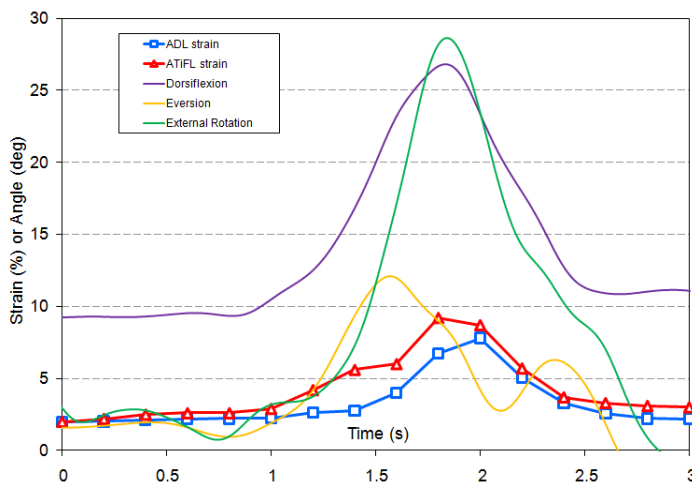

Fig 8. Average kinematics and model predicted ligament strains during external foot rotation studies on human subjects [30].
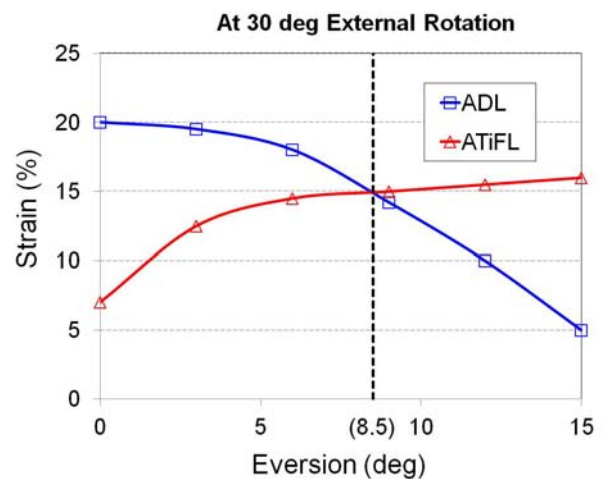
Fig. 9. Predicted ligament strains from a parametric study at 30° of external rotation with increasing degrees of talus eversion [32].

IV. DISCUSSION

The results from the two cadaver failure experiments and corresponding computer simulations supported the hypothesis that for various foot constraints and ankle positions, the ligament with the maximum strain would predict ankle ligament injuries. This study also demonstrated that dynamic external foot rotation can produce two distinct injuries in the form of either a high ankle sprain (ATiFL injuries) or a medial ankle sprain (ADL injuries) depending on the amount of talar pre-eversion. Additionally, shoes with different rotational stiffness designs were shown to affect the amount of talar eversion that occurs during external foot rotation, as well as increasing ATiFL strain when increased talar eversion occurs. Although the ankles in these experiments were initially positioned in a neutral position, the flexible shoe had more coupled eversion of the talus during external rotation of the foot. The coupled rotations produced a more compliant response of the ankle and significantly less ankle joint torque with the flexible shoe than a rigid shoe at 30° external foot rotation. In the failure experiments there were significant differences between the maximum external rotations when injury occurred for different foot constraints and ankle positions. This also supported the notion that increased talar eversion decreases the rotational stiffness of the ankle joint in external rotation and may act as a torsion dissipating mechanism [33]. Human subjects performed a movement that was similar to the cadaver experiments and also demonstrated coupled external rotation and eversion of the ankle. Although the shoe stiffness and *in vivo* experiments were at subfailure levels of ankle joint torque, they provided additional and perhaps a more realistic understanding of joint biomechanics and ligament strains than failure cadaver experiments, which were more precisely controlled. The current study may suggest some method of parallel restraint to the foot, such as with a rigid football shoe design or ankle taping [34] would affect the ankle joint mechanics and injury response under excessive levels of external foot rotation.

Numerous biomechanical studies have been conducted to help understand mechanisms of high ankle sprain in controlled laboratory experiments. Many of these, however, have been ligament sectioning studies that show increased lateral motion of the fibula relative to the tibia during external rotation of the foot as syndesmotic structures are sequentially cut [35]-[39]. Other biomechanical studies have largely been designed to investigate ankle fractures rather than sprains [19],[40]. For example, a study by Haraguchi and Armiger [41], using a relatively rigid experimental restraint for the foot, has shown external rotation of the foot with the ankle in pronation produces damage to the ATiFL with fibular fracture, followed by medial injury that involves the deltoid ligament. Another early study by Stiehl et al. [42], using a more compliant fiberglass cast tape restraint of the foot onto a rigid plate, documents ATiFL injury in combination with fibular fracture. Although patients with a high ankle sprain typically report tenderness over the anterolateral tibiofibular joint where the fibers of the ATiFL are often disrupted [43], a review of the literature by the authors have found no previous *in vitro* studies producing isolated ligament injuries to the ankle that represent a clinical high ankle sprain.

The mechanism of injury in high ankle sprains can be confusing because of the different anatomic structures involved and the manner in which these structures develop excessive stress in the three planes of motion [44]. Talus motion plays an important role in developing ankle ligament strains, especially under rotational loading [9], and therefore its motion is crucial in the study of ankle ligament injury mechanisms [45]. A more detailed motion analysis of the relative motion between the talus and tibia shows that in the everted foot, axial loading causes lateral translation of the talus and likely pre-straining of the ATiFL [31]. During external foot rotation the talus is forced to rotate externally and translate laterally, resulting in further separation of the fibula from the tibia. In the neutral foot, however, lateral translation of the talus is minimal during both axial loading and external rotation to result in ADL rupture [31]. A clinical review indicates that external rotation of the neutral foot will "first rupture the deltoid ligament, with subsequent injury to the ATiFL" [46]. This notion was supported in our computer simulations where the ADL experienced the highest strain under external rotation of the neutral foot, while ATiFL strain was also elevated compared with other ankle ligaments. In addition to the mechanism of medial ankle sprains being due to excessive external foot rotation, other mechanisms potentially causing deltoid ligament damage could be excessive eversion, excessive dorsiflexion, or a combination of both [19].

The ligament strains predicted by our computer simulations supported the experimental observations by producing failure level strains in the ATiFL for the everted foot and in the ADL for the neutral foot experiments. The literature shows that ligament failure typically occurs at 30-35% strain [47], which was consistent with the model outputs for both the neutral and everted cases (31.5% in the ADL and 32% in the ATiFL, respectively). A

limitation of the study was that it assumed simple, linear-elastic behavior of the ankle ligaments. Funk et al. [47], produced non-linear models for eight major ligaments of the foot-ankle complex, which accounted for both their elastic and viscoelastic properties. However, they concluded that the elastic response function curve-fits were only valid up to 10-20 percent strain, depending on the specific ligament and that the viscoelastic assumption could also be neglected for very slow (>1000 s) or very fast (<0.1 s) step times. In the current experiments the ligaments reached their maximum strain in 0.5 s, indicating that there could be some viscoelastic effect. Since the objective of the current study to determine ligament strains at failure, non-linear elastic functions would likely have resulted in an overestimation of the ligament forces. Yet, in order to improve upon the model in future simulations of ankle ligament failure strains from *in vitro* and *in vivo* studies, more accurate constitutive modeling would seem to be warranted.

A recent computer simulation of the neutral versus everted ankle experiments used subject-specific models from CT scans of each specimen [48], instead of the generic model used in the current study. The failure strains were similar to the current results with 30.6±4.3% in the ADL for neutral ankles and 32.4±4.1% in the ATiFL for everted ankles. Ankle sprains have been graded in the clinical literature and associated with the degree of damage to a ligament resulting from excessive strains [49]. One of the benefits of using subject-specific models to predict ligament strain is the ability to investigate the degree of damage in different specimens in more detail. Ligaments that suffered a partial tear had a failure strain of approximately 26%. This indicates that grade II ligament sprains, consisting of a partial macroscopic tear, might occur around this level, but currently there are no references that have identified the strain magnitude required for a partial ligament tear. In three of the subject-specific models, despite similar ankle kinematics, a range of damages was predicted based on the magnitude of ligament strain. One possible explanation for the injury severity inconsistency among subjects could be variability of geometry in the subtalar joint. The curvature of the tronchlea tali (top surface of the talus) determines the stability of the ankle in the anterior-posterior direction [50] and the talar facets (contacting surfaces) have a significant impact on the position of the axis of rotation between the talus and calcaneus [51]. This suggests that in cases of significant anatomical differences between individuals, a generic model may not be suitable for certain computer simulation studies.

A common limitation of cadaver studies is that the effects of active muscles are not included. However, a limitation of *in vivo* studies is that the safety of human subjects must be protected and therefore the forces and moments must be kept at subfailure levels. While the effects of active muscles on talus motion and subsequent ankle injury occurring in sports are currently unknown, kinematic data may be generated in future studies using game films of injury-producing events [11],[52]. By combining this video analysis technique with our computer simulation techniques, ligament strains at the time of injury might be estimated. Specimen age may also be a factor that affects ligament properties. While the study was not conducted on a typical young, sport-active population, contralateral limbs of each subject were used to study the effect of foot constraint and ankle position. Although the joint kinematics may not be so influenced by specimen age, failure torque data in current studies that lack active muscle are likely conservative values for the high ankle sprain injury threshold. In fact, the peak torques measured with football shoes and a one times body weight axial load were approximately 90 Nm for natural grass and 115 Nm for artificial surfaces [14]. The cadaver fixation method of transecting and potting the tibia and fibula may have also limited the normal function of the proximal tibiofibular syndesmosis to affect ankle joint mechanics in our experiments. Limitations and assumptions of the computational model include the use of linear spring elements to represent nonlinear and viscoelastic ligament behavior. While the strain rates were above the level recommended for viscoelastic constitutive models, the nonlinear stiffness of ligaments up to failure levels of strain should be included in future studies.

V. CONCLUSIONS

The major achievement of this study was to integrate *in vitro*, *in vivo* and computer simulation methods for the goal of elucidating the external rotation mechanism of injury for high ankle sprains in sports. The hypothesis that ligament strain would predict ankle ligament injuries for various foot constraints and ankle positions was supported. This study also demonstrated that external foot rotation could produce two distinct injuries; high ankle sprains (ATiFL injuries) and medial ankle sprains (ADL injuries), depending on the amount of talar pre-eversion. Shoe designs that constrain the subtalar joint were also found to affect ankle joint mechanics and

injury response during external rotation of the foot. And finally, this series of experiments was the first *to* consistently produce isolated ligament injuries to the ATiFL that represent a clinical high ankle sprain. The study outcome may provide an avenue for the development of football shoe designs or other protective gear that may help reduce the incidence and/or severity of medial and high ankle sprains on the athletic field.

VI.   ACKNOWLEDGEMENT

VII.   REFERENCES

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