Patellar Tendon Strain Is Increased at the Site of the Jumper’s Knee Lesion During Knee Flexion and Tendon Loading

Results and Cadaveric Testing of a Computational Model

Michael Lavagnino, PhD, Steven P. Arnoczky,* DVM, Niell Elvin, PhD, and Julie Dodds, MD

From the Laboratory for Comparative Orthopaedic Research, College of Veterinary Medicine, Michigan State University, East Lansing, Michigan

Background: Patellar tendinopathy (jumper’s knee) is characterized by localized tenderness of the patellar tendon at its origin on the inferior pole of the patella and a characteristic increase in signal intensity on magnetic resonance imaging at this location. However, it is unclear why the lesion typically occurs in this area of the patellar tendon as surface strain gauge studies of the patellar tendon through the range of motion have produced conflicting results.

Hypothesis: The predicted patellar tendon strains that occur as a result of the tendon loads and patella–patellar tendon angles (PPTAs) experienced during a jump landing will be significantly increased in the area of the patellar tendon associated with patellar tendinopathy.

Study Design: Descriptive laboratory study.

Methods: A 2-dimensional, computational, finite element model of the patella–patellar tendon complex was developed using anatomic measurements taken from lateral radiographs of a normal knee. The patella was modeled with plane strain rigid elements, and the patellar tendon was modeled with 8-node plane strain elements with neo-Hookean material properties. A tie constraint was used to join the patellar tendon and patella. Patella–patellar tendon angles corresponding to knee flexion angles between 0° and 60° and patellar tendon strains ranging from 5% to 15% were used as input variables into the computational model. To determine if the location of increased strain predicted by the computational model could produce isolated tendon fascicle damage in that same area, 5 human cadaveric patella–patellar tendon–tibia specimens were loaded under conditions predicted by the model to significantly increase localized tendon strain. Pre- and posttesting ultrasound images of the patella–patellar tendon specimens were obtained to document the location of any injured fascicles.

Results: Localized tendon strain at the classic location of the jumper’s knee lesion was found to increase in association with an increase in the magnitude of applied patellar tendon strain and a decrease in the PPTA. The principal stresses and strains predicted by the model for this localized area were tensile and not compressive in nature. Applying the tendon strain conditions and PPTA predicted by the model to significantly increase localized strain resulted in disruption of tendon fascicles in 3 of the 5 cadaveric specimens at the classic location of the patellar tendinopathy lesion.

Conclusion: The localized increase in patellar tendon strain that occurs in response to the application of tendon loads and decreased PPTA could induce microdamage at the classic location of the jumper’s knee lesion.

Clinical Relevance: The association of decreasing PPTA with increasing localized tendon strain would implicate the role of knee-joint angle as well as tendon force in the etiopathogenesis of jumper’s knee.

Keywords: patellar tendinopathy; computational model; strain; patella–patellar tendon angle; jump landing

*Address correspondence to Steven P. Arnoczky, DVM, Director, Laboratory for Comparative Orthopaedic Research, College of Veterinary Medicine, G-387, Michigan State University, East Lansing, Michigan 48824 (e-mail: arnoczky@cvm.msu.edu).

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Jumper's knee (patellar tendinopathy) is a condition usually associated with activities that require repetitive, forceful quadriceps muscle contraction. Clinically, jumper's knee is characterized by a localized tenderness of the patellar tendon at its origin on the inferior pole of the patella and a characteristic increase in signal intensity at this location on MRI. Although the cause of this condition has yet to be clearly defined, tendon overloading during jump takeoff and/or landing, and the resultant creation of microtears within the tendon, has been suggested to be the cause of patellar tendinopathy. Clinical studies have indicated that athletes who subject their patellar tendons to higher loads and increased knee flexion angles are at a higher risk for tendinopathy.

Although excessive tendon loading appears to be a contributing factor in patellar tendinopathy, the precise link between the mechanical loading conditions and the pathologic response remains obscure. For example, it is still unclear why the jumper's knee lesion typically occurs in the proximal portion of the patellar tendon. While excessive strain in the posterior fibers of the patellar tendon during knee flexion has been postulated as an inciting factor in patellar tendinopathy, recent cadaveric strain gauge studies examining the surface strains experienced by the patellar tendon during the range of motion have produced conflicting results. Others have suggested that the lesion of jumper's knee represents an adaptational change of the tendon in response to a compressive force acting at the proximal, posterior aspect of the patellar tendon. In this theoretical scenario, the inferior pole of the patella is thought to impinge against the proximal portion of the patellar tendon during knee flexion. It is theorized that an alteration in the type of load (compression vs tension) experienced by the tendon will result in an adaptational change in the extracellular matrix of the tendon, making it more susceptible to injury. However, this theory has never been rigorously examined.

It is possible that a finite element (FE) model of the patella–patellar tendon interface could provide some insight as to the strain environment at this location during tendon loading. Finite element models have been used to predict the strain distribution in the supraspinatus and Achilles tendons. Therefore, the purpose of this study is to create a computational model of the human patella–patellar tendon complex in an effort to characterize the strain patterns developed within the tendon in response to the loading forces and knee joint flexion angles predicted to occur during a jump landing. The conditions generated by the computational model are then tested in a cadaveric specimen to determine if the location of microdamage to the patellar tendon can be accurately predicted. We hypothesize that during knee flexion and patellar tendon loading, tendon strain will be markedly increased in the area of the patellar tendon commonly associated with patellar tendinopathy. We also hypothesize that this localized area of increased strain will coincide with the area of the patellar tendon most at risk for fascicle damage during a simulated jump landing.

**MATERIALS AND METHODS**

**Creation of the Computational Model**

To determine the location of highest principal strain within a loaded patella–patellar tendon complex, a 2-dimensional computational FE model composed of a patella and patellar tendon was developed. The patella was modeled with 2-node plane strain rigid elements, and the patellar tendon was modeled with 8-node plane strain elements with neo-Hookean (ie, nonlinear elastic) material properties of tensile modulus (E = 1000 MPa) and Poisson ratio (ν = 0.49). Plane strain rigid elements only examine strain within the 2-dimensional plane. Tie constraints were used to join the patellar tendon and patella. The anatomic relationships of the patella and patellar tendon, as well as the boundaries of the insertion of the patellar tendon onto the inferior pole of the patella, were determined from lateral radiographs of a human knee. Sequential lateral radiographs of a normal, human knee taken at 0°, 15°, 30°, 45°, and 60° of flexion were used to confirm the patella–patellar tendon angles (PPTAs) at each degree of knee flexion and to calculate the instant center of rotation of the patella during knee joint flexion using a previously described method.

In the model, a uniform initial displacement (0.553 mm) was applied to the distal portion of the tendon to approximate a 1% tensile strain and engage the collagen fibers of the model. Thereafter, a second displacement step corresponding to either 5%, 8%, 10%, 12%, or 15% total tensile strain (ε_t) was applied to the tendon with or without a rotation (0°, 4°, 8°, 13°, or 17°) of the patella at the calculated center of rotation in 0.005-second intervals for 0.2 seconds. The second step approximates the change in PPTA (162° to 145°) that occurs during knee flexion between 0° and 60° at the same loading rate as if landing from a 60-cm height. The maximum force generated in the patellar tendon from a jump landing has been shown to occur at the end position of this second step with the PPTA at 145° and the corresponding knee flexion angle at 60°. The in vivo forces that occur within the patellar tendon from a jump landing have been predicted to range between 4000 and 5000 N. These forces, when compared with cadaveric patellar tendon studies with an average cross-sectional area of 150 mm², correspond to a range of stress from 26.7 MPa to 33.3 MPa and strains from 8.5% to 15%.

The FE program ABAQUS 6.4 (ABAQUS, Inc, Pawtucket, Rhode Island) was used for all analyses. The maximum principal strain value at each node in a predetermined set of nodes, which corresponded to the site of the jumper's knee lesion on MRI, was averaged to give the mean localized strain value (ε_l) (Figures 1 and 2). In addition, the maximum principal strain was determined at a unique node in the location of the highest maximum principal strain (Figure 2). However, because the degree of material inhomogeneity of the patellar tendon is unknown, both the maximum point strain (at a single node) and mean localized strain are reported. A series of comparisons of the FE analysis results were based on the distribution pattern of the principal strains and the mean localized strain value. To determine the role of applied strain on the
location and magnitude of principal strains, various applied tensile strain magnitudes were compared at a single PPTA. To determine the role of PPTA on the location and magnitude of principal strains, various PPTAs ranging from 162° to 145° were compared at a single applied tensile strain. In addition, the distribution of principal stresses was also analyzed for the condition with the greatest principal strains (15% applied strain with a 145° PPTA).

The computational robustness of the model (the ability of the FE model to work with a range of physiologic parameters) was tested under varying material properties and model assumptions to ensure consistency. The tendon’s principal strains were calculated over a wide range of previously reported tendon tensile moduli (213 MPa, 584 MPa, 1232 MPa)\textsuperscript{14,15} and Poisson ratios (0.25, 0.375, 0.49).\textsuperscript{4} The model convergence was checked by analyzing the differences in recoverable strain energy for a range of node numbers (between 4938 and 19 752 nodes). Convergence of the model is achieved when doubling of the number of FE nodes does not significantly (<1%) alter the recoverable strain energy results of the model.

Cadaver Testing of the Computational Model

To test the ability of the computational model to predict the area of highest principal strain within the patellar tendon, and thus the area most susceptible to tensile damage, 5 human patella–patellar tendon–tibia cadaveric specimens were evaluated (4 males ages 21 years [2 males], 26 years, and 44 years; 1 female, age 26 years). All the tendons appeared grossly normal. Each specimen was mounted in a custom-made jig that allowed the patella to be rotated and fixed at a predetermined PPTA (Figure 3). Using the PPTA value determined by the computational model to produce the highest localized principal strains (145°) the specimen was preloaded to 100 N on an Instron 8501 materials testing system (Instron Corp, Canton, Massachusetts). The length of the patellar tendon was measured from the patella to the insertion on the tibia along the posterior surface of the tendon.\textsuperscript{13} The measurement was repeated 3 times and the average calculated to determine the applied displacement and rate. The patellar tendon was then loaded to 15% tensile strain at a rate of 100% strain per second. This strain level represented the high end of the calculated strain level estimated to occur in a patellar tendon during a jump landing.\textsuperscript{30,32} The specimens were kept moist with physiologic saline spray throughout the testing.

To determine the mechanical properties of the patellar tendon, the load and displacement values were recorded using a computer data acquisition system. The rectangular cross-sectional area of the patellar tendon was determined at the patella–patellar tendon junction by measuring both the thickness and the width 3 times using a digital caliper and taking the average of each dimension. The standard deviation of the calculated cross-sectional area based on these 3 measurements was ± 6.4 mm\textsuperscript{2}, which is in agreement with a previous study that used this measurement technique.\textsuperscript{15} Load at the maximum (15%) strain, stiffness, stress at 15% strain, and tensile modulus were computed from the load–deformation data.

The tendons were evaluated before and after mechanical testing for damage using a Shimadzu SDU-12000 X-plus ultrasound system (Shimadzu Corp, Kyoto, Japan) with a 9- to 12-MHz linear probe and stand-off attachment.

RESULTS

Computational Model

The computational model predicted that the mean localized strain ($\varepsilon_L$) (Figure 4) and maximum point strain
(Figure 5) increased in response to a decrease in the PPTA and an increase in the applied patellar tendon strain. The mean localized strain at a 162° PPTA (corresponding to a knee flexion angle of 0°) was within 10% of the applied strain on the tendon. However, when the PPTA was reduced to 145° (corresponding to a knee flexion angle of 60°), the mean localized strain increased to nearly twice the applied strain (Figure 4). For example, the results of the computational model predict a mean localized tendon strain of ~17.4% at the inferior pole of the patella when the knee is flexed to 60° (145° PPTA) and 10% global strain is applied to the tendon (Figure 6). This region of high strain is only present when the knee is undergoing flexion (and intensifies as the knee approaches 60° of flexion) and the PPTA decreases (Figure 6). This region of high strain is also only present when applied strains are greater than 5% (Figure 7).

The model also demonstrated that the principal mechanical stresses in the region of localized strain were parallel to the long axis of patellar tendon fascicles, signifying a tensile rather than a compressive force acting in this area.

In comparing the principal strains on the tendon surface just distal to the patella–patellar tendon insertion, the FE model also predicted that the anterior surface of the patellar tendon would consistently experience higher strains than the posterior surface at a 145° PPTA (Figure 7). The model appeared robust as the principal strain values and locations were minimally affected by varying the tensile modulus and/or Poisson ratio of the tendon. The model was also found to be convergent, as there was no difference (<1%) in recoverable strain energy when the number of nodes were halved or doubled.

**Figure 3.** The specimen mounted in a custom-made jig with the tibia on top, and the patella rotated and fixed at a predetermined patella–patellar tendon angle of 145°.

**Figure 4.** Localized mean strain (\(\varepsilon_L\) ± standard deviation) as a function of patella–patellar tendon angle (PPTA) for a range of applied strains (\(\varepsilon_A = 5\%-15\%\)).

**Figure 5.** The maximum principal strain at a point as a function of patella–patellar tendon angle (PPTA) for a range of applied strains (\(\varepsilon_A = 5\%-15\%\)).

**Figure 6.** The specimen mounted in a custom-made jig with the tibia on top, and the patella rotated and fixed at a predetermined patella–patellar tendon angle of 145°.

**Figure 7.** Maximum point strain (%) as a function of patella–patellar tendon angle (PPTA) for a range of applied strains (\(\varepsilon_A = 5\%-15\%\)).

**Figure 8.** The specimen mounted in a custom-made jig with the tibia on top, and the patella rotated and fixed at a predetermined patella–patellar tendon angle of 145°.

**Cadaver Testing**

Ultrasound examination of the strained cadaveric patella–patellar tendon–tibia complex revealed an obvious disruption of the fascicular architecture within the patellar tendon immediately adjacent to the inferior pole of the patellar in 3 of the 5 cadaver specimens (Figure 8). This fascicular disruption occurred at the precise location of highest principal strain predicted by the model for the 145° PPTA being tested.

The 2 specimens without obvious ultrasound damage had similar mechanical properties (stiffness, stress at 15% strain, and modulus) compared with the 3 damaged tendons (Table 1). However, these undamaged patellar tendons were from the 2 smallest patellar tendons based on their cross-sectional area and length (Table 1). In addition, the undamaged tendons required lower loads to produce the same strain (Table 1).

**DISCUSSION**

Patellar tendinopathy usually affects athletes whose sport involves repetitive explosive extension or eccentric flexion of the knee. While the excessive tensile loading is thought to induce microscopic tears within the tendon, ultimately leading to degeneration, the suggested
pathogenesis of stress overload and tensile failure has, to date, not been able to explain why the lesion of patellar tendinopathy is localized to the proximal portion of the patellar tendon at its insertion to the inferior pole of the patella.17

Cadaveric studies examining the surface strains of the patellar tendon through the range of motion have produced conflicting results.1,5 One group found that on flexion, the tensile strain on the patellar tendon increased on the anterior aspect of the tendon and decreased on the posterior aspect.1 This led the authors to conclude that “stress shielding” is a more important etiologic factor in patellar tendinopathy than repetitive tensile loading. Another group of investigators found that during knee flexion, the posterior fascicles of the patellar tendon experienced significantly greater strains than the anterior fascicles.5 A third study examining isolated fascicles from the patellar tendons of young men demonstrated that fascicles from the anterior portion of the tendon displayed greater peak stress, yield stress, and tangent modulus when compared with fascicles from the posterior portion of the tendon.14 The authors concluded that the posterior fascicles are weaker than the anterior fascicles and, perhaps, more prone to injury. However, none of the previous studies were able to provide a compelling rationale for the classic proximal location of the patellar tendinopathy lesion.

Therefore, we developed a 2-dimensional FE model of the patella–patellar tendon complex to determine if the strain patterns predicted by the model, for a given set of tendon strains and PPTAs, could help explain the site predilection of the patellar tendinopathy lesion. Recent studies using 2-dimensional FE models of the human supraspinatus tendon have identified areas of high principal strain concentration and have shed considerable light...
on the pathogenesis of rotator cuff tendinopathy.\(^\text{36,40}\) In the current computational model, a range of strain values calculated from both in vivo and in vitro determinations of patellar tendon forces experienced during jump landings\(^\text{20,32}\) and the mechanical properties of human patellar tendons\(^\text{6-8,10,15,16,18,25}\) were used as input variables. In addition, the PPTA angles as well as the patellar tendon loading rate experienced during a jump landing\(^\text{32}\) were used in this computational model.

The results of the current FE model predict that as the PPTA decreases from 162° to 145° (which approximates 0° to 60° of knee joint flexion), the maximum point strain as well as the mean localized strain increased significantly within the area of the proximal patellar tendon classically associated with the location of the patellar tendinopathy.\(^\text{17}\) Likewise, both maximum point strain and mean localized strain significantly increased with increasing applied patellar tendon strain.

The relationship between knee flexion angle and the magnitude of applied patellar tendon force in the etiopathogenesis of patellar tendinopathy has been previously suggested.\(^\text{35}\) A clinical study quantifying the lower extremity dynamics experienced by elite male volleyball players suggested that the likelihood of patellar tendinitis was significantly related to high loading forces in the knee extensor mechanism combined with deep knee flexion angles.\(^\text{35}\) The results of the current model support those findings, as a decrease in PPTA and an increase in applied patellar tendon force is predicted to significantly increase localized tensile strain in the area of the patellar tendinopathy lesion.

Although the precise level of patellar tendon force experienced during a jump landing is not known, an elegant mathematical model has predicted that during a drop landing from a height of 60 cm, peak quadriceps forces occur at approximately 60° of knee flexion.\(^\text{32}\) This degree of

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**Figure 7.** Principal strain distribution and mean localized principal strain ($\varepsilon_L$) within the patellar tendon at a patella–patellar tendon angle (PPTA) of 145° with varying applied strains ($\varepsilon_A$). Note that the region of localized high strain intensifies with increasing applied strain. The encircled x (⊗) indicates the center of rotation of the patella.
knee flexion also coincides with the most acute PPTA (145°) experienced throughout the range of motion. This knee position (PPTA) also corresponded with the highest mean localized patellar tendon strain predicted by the computational model and resulted in an almost doubling of the applied strain at the classic site of patellar tendinopathy lesion. Therefore, this orientation was used to test the model predictions in a human cadaveric specimen. In addition, the upper range of predicted patellar tendon strain (15%) that occurs during a jump landing was used in this test.

The results of the cadaveric testing produced a localized disruption in the tendon fascicle architecture at the junction of the patellar tendon and the inferior pole of the patella in 3 of the 5 specimens. This was the same site of localized increased tensile strain predicted by the model and would suggest that this area of the patellar tendon is at greatest risk for microdamage during the loading conditions associated with jump landings. The 2 specimens that did not demonstrate obvious ultrasound damage had lower maximum loads at 15% strain than did the damaged tendons. The undamaged patellar tendons were from the 2 smallest specimens and thus had smaller cross-sectional areas, lengths, and force required to produce the same strain. Therefore, it is possible that these specimens were at a lower risk for developing collagen fiber disruption at the applied strains due to their anatomic size or the individual mechanical properties of the tendon.26 A recent study has demonstrated gender-based differences in the mechanical properties of human patellar tendons.26 The study reported a significant decrease in stiffness and tensile modulus in female patellar tendons when compared with those of males.26 Such differences could explain the failure of the 1 female specimen to exhibit ultrasonographic evidence of damage after the application of 15% strain. Finally, it is also possible that fiber damage may have been present but was undetectable using the applied ultrasound technology.

As noted previously, the precise strain magnitudes experienced by the patellar tendon during a jump landing are unknown. The 15% strain used in the cadaveric testing in the present study represented what was determined to be the upper range of predicted values for a jump landing from previous studies.

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Table 1: Biomechanical Properties of the Tested Cadaveric Specimens

Figure 8. Preinjury (A) and postinjury (B) ultrasound images of a human patella–patellar tendon interface from a 44-year-old male showing the presence of disrupted fascicles (arrow) in the precise location of the patellar tendon predicted to have the highest principal strains. The patellar tendon was strained to 15% with a patella–patellar tendon angle of 145°. PT, patellar tendon; P, patella.
the failure properties of human patellar tendons between the ages of 17 and 50 years. In that study, 19 of 20 patellar–patellar tendon specimens failed in the midsubstance of the patellar tendon (the remaining specimen failed by tibial bone avulsion). Although the investigators used the same strain rates (100% per second) used in the current study, their testing jig used a PPTA of 180°. Thus simply altering the PPTA appears to change the area of the patellar tendon most at risk during tensile loading.

Finally, the results of the current study also suggest that the principal stresses and strains at the site of the classic tendinopathy lesion are tensile in nature rather than compressive. This finding would call into the question the theory that the pathologic changes seen in patellar tendinopathy represent an adaptational change by the tendon to an increase in compressive forces secondary to an impingement of the inferior pole of the patella on the patellar tendon during flexion. A previous clinical study showed no evidence of any alteration in the PPTA between patients with and without patellar tendinopathy. This finding, coupled with the results of the current computational model, would suggest that excessive compression of the patellar tendon secondary to patellar impingement may not be a significant factor in the origin of jumper's knee.

As with any initial iteration of a computational model, there are limitations that must be acknowledged. First, the current FE model did not take into account the anisotropic, viscoelastic properties of either the tendon or the patellar bone. Although this may not cause a significant change in the results predicted by the model, these properties must be considered in future iterations. Second, the degree of material inhomogeneity of the patellar tendon is unknown. A recent study has suggested that the human patellar tendon has region-specific mechanical properties. However, the precise degree of inhomogeneity has yet to be determined. Gender differences in the mechanical properties of the patellar tendon must also be considered in future iterations of the model. In addition, the 3-dimensional motion and structure of the patella–patellar tendon complex was not taken into consideration in this analysis. A recent study has suggested that varus and valgus forces during jump landings may contribute to increased risk of knee injury. Finally, only 1 normal human knee was used to determine the anatomic and rotational properties for the current computational model. However, the change in PPTAs associated with the knee flexion angles from this 1 knee was similar to those reported in an earlier study. This same study also demonstrated that the geometry of the inferior pole of the patella had no obvious relationship to patellar tendinopathy. Additional studies are necessary to completely define the specific parameters that will allow the computational model to most accurately reflect the material and functional characteristic of the patella–patellar tendon complex during activities such as a jump landing. Additionally, further cadaveric testing is needed (and ongoing) to refine and validate the computational model.

The above-noted limitations notwithstanding, we believe the computational model presented in this study represents a significant first step in understanding the relationship between the mechanical loading conditions experienced in the patellar tendon during a jump landing and the potential stimulus for the pathologic lesion seen in patellar tendinopathy. The results of this study suggest that under specific conditions of tendon loading and knee flexion (PPTA), the localized increase in tensile patellar tendon strain could induce microtrauma to the tendon in the precise area commonly associated with the jumper's knee lesion. In addition, the significance of knee joint angle (PPTA) in concentrating this tensile strain suggests that landing style could have a significant impact on the etiopathogenesis of patellar tendinopathy. This, in turn, would imply that modifying landing techniques may be a way to lessen the localized strain in this area and thus potentially prevent patellar tendinopathy.

The ability to correlate clinical, computational, and cadaveric studies may represent the most comprehensive approach to linking loading patterns in tendons to the pathologic responses associated with patellar tendinopathy. Understanding the etiopathogenesis of patellar tendinopathy could allow the development of interventional and rehabilitative programs to prevent or lessen the debilitating pathologic changes associated with this condition.

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