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Traumatic vs. Pathological Achilles Tendon Ruptures: A Look into the Importance of the Collagen Matrix and the Preferred Method of Repair

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By

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ABSTRACTS

McLEAN, KATHLEEN Part I: A Comparative Literature Review: Traumatic vs. Pathologic Achilles tendon Ruptures and a Foresight into the Preferred Repair Method. Department of Bioengineering, June 2011.

ADVISOR: Professor Jennifer Currey

The Achilles tendon is the largest, strongest, and thickest tendon in the human body. While it may be the strongest, the Achilles tendon is also among one of the most frequently ruptured. These lower limb injuries arise as a result of athletically induced trauma or pathologically and steroidal induced trauma. While the manner in which athletic ruptures occurs can be determined on a case to case basis through the use of clinical examination, ultrasonography, and magnetic resonance imaging (MRI), the method in which the injury should be treated remains controversial. The two main methods of repair include a conservative approach and a surgical approach. Such requirements including lower re-rupture rates, earlier return to activity, lower rates of complications, restoration of normal length and tension to the tendon, and maximization of the strength and function of the calf muscle produce a debated standpoint between the two methods. Pathologically induced ruptures, on the other hand, are not well-understood. As a matter of fact, the actual effects of corticosteroids and fluoroquinolones on the tendon remain controversial. The aim of the present study, therefore, was to gather data from various researchers on Achilles tendon ruptures, induced both athletically and pathologically, and determine both the preferred repair method for athletically induced ruptures and the impact of steroids on pathologically induced ruptures. Studies reporting data regarding the surgical versus conservative methods were analyzed on a broad scale while the effect of specific surgical techniques was excluded.

McLEAN, KATHLEEN Part II: Analysis of the Achilles tendon and collagen matrix using Finite Element Analysis.
Department of Bioengineering, June 2011

ADVISOR: Professor Jennifer Currey

After completing a comparative literature review regarding the differences between athletically and pathologically induced Achilles tendon ruptures, it became evident that a computer model of the tendon was not a common entity. Advantages of using interactive graphics to model the musculoskeletal system were first described in 1977, and since then, an explosion in the advancement of computer technology has allowed many to interact with models of this system. Models of the musculoskeletal system can not only provide insight into treatment options, but can also aid in the understanding of the biomechanical consequences of certain surgical procedures. Subsequently, doctors acquire the ability to alter difficult surgeries and design more effective procedures in order to increase the likelihood that outcomes are positive.

Whether it is due to overuse or steroidal effects, the collagen matrix within the Achilles tendon plays a major role in the tendon's degradation. This study analyzed the effects of type 1 and type 3 collagen on the tendon using Finite Element Analysis, a simulation program in SolidWorks. During degradation, the percentage of type 1 collagen has shown to decrease, eventually becoming replaced by type 3 collagen. Understanding that type 3 collagen is much weaker, especially in tension, than type 1 collagen, it is hoped that the type 3 collagen matrices will depict a weaker tendon at increasingly higher loads. With the production of a working Achilles tendon model, it is possible that further research studies can be done to analyze the effects of surgical and conservative treatment upon the tendon.

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Part I.

A Comparative Literature Review: Traumatic vs. Pathologic Achilles tendon Ruptures and a Foresight into the Preferred Repair Method.

Introduction

The Achilles tendon is the largest, strongest and thickest tendon in the human body [21, 32]. The gastrocnemius and soleus muscles, which together form the calf muscle, unite to form an aponeurosis which descends and forms the Achilles tendon on the back of each leg [1, 26]. An aponeurosis simply signifies a sheet of tendon-like material that anchors the gastrocnemius and soleus muscles to the Achilles [16]. At just about 15cm in length, the Achilles inserts into the middle third of the posterior surface of the calcaneus (heel bone). The gastrocnemius fibers converge and rotate toward the lateral aspect of the calcaneus while the soleus fibers converge and descend, ultimately making up the medial aspect of the tendon's insertion. This crossing of the tendon, rotating clockwise in the left limb and counterclockwise in the right limb, significantly acts on the knee, ankle and subtalar joints. Ultimately, the Achilles is capable of managing the smooth transition to knee extension, giving stability to the foot, and plantar flexing the ankle [26].As one can see, the role of the Achilles in everyday movements is exceptionally important, and rupture to the tendon can be detrimental. Therefore, understanding the manner in which the Achilles can rupture and the best way to heal it can be very important.

While it may be the strongest, the Achilles tendon is also one of the most frequently ruptured. Approximately twenty percent of all large tendon injuries are accounted for by the Achilles [21]. More often than not, ruptures occur 2-6cm proximal to the calcaneal insertion where there is a reduction in both the number and mean relative area of blood vessels [22]. These lower limb injuries result from a variety of causes ranging from athletically induced trauma to pathologically and steroidal induced trauma.

More specifically, Achilles tendon ruptures tend to exhibit a bimodal age distribution; the first peak of injuries appearing between the ages of 30 and 40 years and the second peak occurring between the ages of 60 and 80 years [24]. The first peak of ruptures is mostly sport related while the second peak of ruptures may be a result of steroidal therapies.

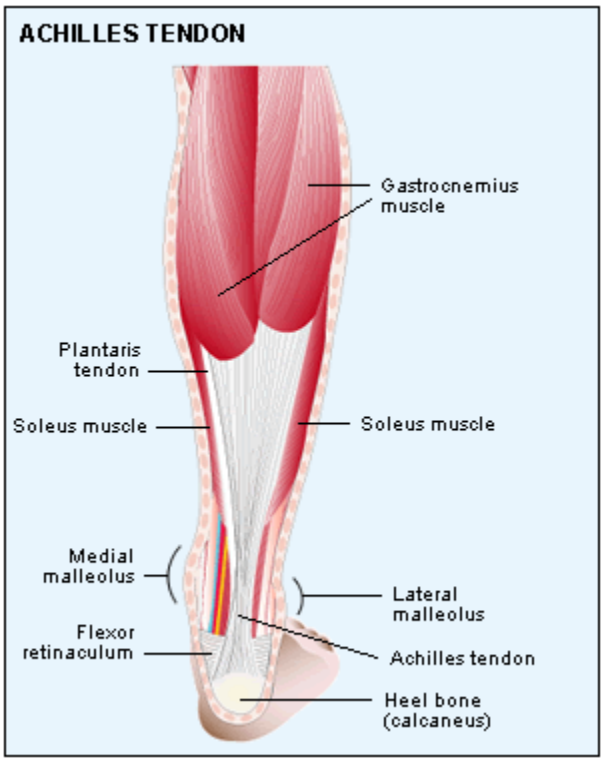
Eighty-three percent of athletically induced Achilles tendon ruptures occur between 2cm and 6cm above the calcaneus. In this area, the tendon has the poorest blood supply, causing degenerative changes of the tendon which accumulate over time and lead to rupture [23]. Sports involving sudden acceleration, jumping and instantaneous changes in speed cause micro-tears in the tendon, eventually leading to an overall weakening of the area and a higher probability of rupture [21, 23]. Through various inspection techniques including clinical examination, ultrasonography, and magnetic resonance imaging (MRI), the manner in which an athletically induced Achilles tendon rupture occurred can be determined [29].

Despite the fact that the manner in which the first peak of Achilles tendon ruptures occurs can be determined on a case to case basis, the manner in which such an injury should be treated remains controversial. In terms of repair methods, the majority of research today deals with the overuse of the Achilles tendon as a result of sports-related activities. The two main methods of repair revolve around a conservative approach and a surgical approach. Such requirements including lower re-rupture rates, earlier return to activity, lower rates of complications (wound healing, sural nerve injuries, etc.), restoration of normal length and tension to the tendon, and maximization of the strength and function of the calf muscle produce a debated standpoint between the two methods in terms of mending the tendon in the best way possible [21, 23].

When it comes to determining the cause of the second peak of Achilles tendon ruptures, the impact of corticosteroids and fluoroquinolones comes into play. While the actual role of such steroids in Achilles tendon ruptures is not fully known, a review of a series of journal articles may suggest noteworthy side-effects. The aim of the present study was to gather data from various researchers on Achilles tendon rupture, induced both athletically and pathologically. Studies reporting data regarding the surgical versus conservative repair methods were analyzed on a broad scale while the effect of specific surgical techniques was excluded. Journals detailing the use of steroids were explored in order to provide an insight to their impact on Achilles tendon rupture.

In other terms, the article attempts to answer the following questions: How do athletically induced Achilles tendon ruptures occur in comparison to pathologically induced ruptures? What is the effect of fluoroquinolones and corticosteroids on the Achilles tendon? Based on the differences in the manner of rupture, whether it be athletic trauma or steroidal trauma, is it possible to determine whether one repair method may be more useful than another?

A. Inner Mechanisms of the Achilles tendon/Sport-Related Ruptures



[http://scienceblogs.com/afarensis/achilles.gif]

Tendons are fibrous cords that attach muscle to bone, and with a very limited blood supply, they are more susceptible to repeat traumas and tiny tears that can eventually result in complete or partial rupture [1]. A combination of eccentric loads from a contracted gastrocnemius muscle and a ground force combine to initiate the rupture of Achilles in most cases. Due to this combination of forces, which can accumulate to a transmitted load of up to 5x the body weight of an individual, the weakest part of the Achilles tendon succumbs, and rupture occurs [16, 21]. In general, the majority of the tendons within the human body experience peak stresses below 30MPa, however the Achilles tendon individually experiences peak forces around 67MPa, more than double that of other tendons. During exertion of plantar flexor moments when the majority of force is transmitted through the Achilles, the peak stresses acting on the Achilles increase

dramatically, resulting in forces from 59MPa during walking to 111MPa during running [10, 31]. In normal movement, the Achilles tendon is stretched during stance, storing elastic energy, and then rapidly recoils during push-off, returning the elastic energy and producing an efficient motion [7].

In order to investigate the manner in which the Achilles ruptures, it is first important to understand the two main muscles that make up the tendon. Recalling that the Achilles is capable of managing the smooth transition to knee extension, giving stability to the foot, and plantar flexing the ankle [26], the gastrocnemius muscle plays a large role in assisting the smooth transition to knee extension. By acting as an antagonist to the quadriceps, this muscle stabilizes and prevents the knee from hyperextending [26]. The soleus muscle on the other hand, being thicker than the gastrocnemius, is a strong plantar flexor, acts as a supinator of subtalar joints, gives stability to the foot, and assists in slowing down subtalar joint pronation and internal rotation of the tibia. Since it is clear that the soleus is involved in gait, it is also important to note that this muscle assists in decelerating the flexion of the tibia in order to assist the momentum of the body to move forward in preparation for another step [26]. Achilles tendinopathy arises when there is poor gastrocnemius-soleus flexibility and when muscle fatigue resulting in elongation becomes evident [12].

While the strength of both the gastrocnemius and the soleus muscles are imperative to a healthy functioning Achilles, it is also important to understand the mechanical properties of the tendon itself. Most material properties of tendons depend on the rate at which the tendon is loaded; most tests showing higher failure stresses and failure strains at faster strain rates. In a study done depicting the properties of the Achilles

tendon, however, failure stresses were lower and failure strains were higher than properties reported for other tendons [31]. In conjunction, the high *in vivo* loading relative to the tendons failure properties may ultimately contribute to the dramatically higher incidence of Achilles tendon ruptures in comparison to all other tendons [31]. As the Achilles fails to adapt to these high *in vivo* stresses, the tendon is left in an unstable position and at a higher risk for injury.

In cases in which loading is suddenly increased, more specifically, in cases where people pick up a new hobby or sport after not participating in such events for a while, the Achilles becomes exceptionally vulnerable and ceases to maintain the loads [31]. Sport-related Achilles tendon ruptures make up 70-80% of all ruptures [14]. A dramatic increase in muscle characteristics of the triceps surae components (gastrocnemius and soleus muscles and calcaneus bone) as a result of an increase in physical activity causes non-uniform influences upon the force production and muscle activation patterns. Consequently, the loading of the tendon becomes varied and can lead to localized fiber damage, inflammation, partial ruptures and even total ruptures [3].

B. Athletic Injuries in Literature

Hard running surfaces, training errors, poor ankle flexibility and overall poorly strengthened ankles account for some of the risk factors related to the most common site of overuse injury, the Achilles tendon [32]. Subsequently, Wren et. al [31] explained that overuse injuries, such as that of the Achilles tendon, can be attributed to changes in activity such as abrupt increase in the duration of intensity of one's training. Sports that require jumping, running, sudden acceleration and quick turns also have the ability to

produce degenerative changes within the tendon, subsequently leading to mechanical micro-trauma as a result of an imbalance between muscle power and tendon elasticity [13, 16, 23, 32]. According to the study by Ying et. al, due to these risk factors and degenerative changes, the tendon is forced to continuously remodel itself through a process of minor injury and repair. Inner mechanisms, such as poor gastrocnemius-soleus flexibility and muscle fatigue resulting in tendon elongation and micro tearing, intensify the effect of normal every day loads to peak levels which the tendon can no longer endure [12]. According to the study by Kim et. al, during activities that require vigorous training, such as those in most athletic sporting events, these inner mechanisms further proliferate the effect of the fast, repetitive loads, and overuse of the Achilles results. In due course, when the tendon's ability to withstand the mechanical loading associated with increased physical activity becomes compromised, tendinopathy arises and the risk of rupture escalates.

While there is an indefinite amount of risk factors pertaining to athletically induced Achilles tendon ruptures, the overarching theme that persists through all of them is the intensity of the sporting event. According to the study by Wren et. al [31], between 1992 and 2000, the incidence of ruptures was six times higher than it was between 1986 and 1994. The reason for such an increase is due to the rise in participation of both males and females in recreational sports [30]. Although females have entered the sporting realm, a disparity in the amount of ruptures occurring between males and females occurs due to the different intensity levels and the corresponding differences in mechanical loading imposed on the tendon [18]. According to results in the study by Ufberg et. al, 2/3 of all ruptures occur in men with the mean age of rupture being 35 years. For all other

tendons, the average age of rupture is 51. Due to the fact that females also tend to enter the sporting scene later than males, the age of women with Achilles ruptures is generally older than men; Nyyssönen et. al reported an average difference of 2-3 years.

Progressive changes in the cellular and fibrous components of the tendon are closely related to the intensity and duration of mechanical stress [26]. In response to increases in the intensity of physical activity, collagen fibrils grow and thickening of the Achilles tendon ensues. According to the study by West et. al, non-inflammatory, degenerative lesions arise as a result of insufficient cellular maintenance of the tendon matrix and collagen fibers. As the amount of lesions increases, the tendon is placed under repeated strains exceeding the limit where the tendon can withstand additional tension, further denaturing the collagen fibers through breakages in the cross-linked structure [5]. As a result, inflammation to the tendon occurs and the Achilles tendon thickens [13]. After a series of repeated micro-trauma, the thickened tendon ruptures. Therefore, according to the study by Cetti et. al, it is evident that tendon ruptures are not an independent entity, but rather a complication of tendinosis, or in other words, the increased inflammation of the tendon due to tissue damage.

Failure of the Achilles in athletic situations generally occurs in one of two ways: rupture of the tendon or avulsion of the posterior tuberosity of the calcaneus where the tendon inserts. Ruptures occur when the tendon experiences complete torsion, generally taking place in the distal half of the tendon, where the tendon experiences the smallest width [31]. Avulsion failures are considered “premature” as they occur at loads much lower than the tendon and bone can normally handle, occurring as a result of the Achilles being pulled off of the calcaneal bone. Complete torsion is found only in fully developed

limbs, and is thus the reason why the majority of ruptures occur to patients in which full bone growth has occurred [26]. As indicated in the study by Wren et. al [30], patients less than twenty years of age typically experience avulsion failures since the tendon strength exceeds bone strength. Between twenty and fifty five years of age, ruptures predominate. And finally, in patients exceeding fifty five years of age, predominant failure changes from tendon rupture to bony avulsion due to pronounced loss of bone strength and better maintenance of tendon strength.

Rupturing of the tendon through calcaneal avulsion is, more often than not, due to a strong contraction of the triceps surae combined with external loading of the foot [30]. In the study by Wren et. al [30], 23 Achilles tendons were tested at two separate strain rates (1% and 10%), thereby inducing a strong contraction, in order to compare the difference in percentage of avulsion type failure versus complete ruptures. Out of the twelve specimens tested at the 1% strain rate, 25% failed by avulsion while the remaining 75% failed by rupture. At the 10% strain rate, approximately, 30% failed by avulsion while the remaining 70% failed by rupture. While no dramatic differences were presented, it is clear that at a higher strain rate, the number of failures by avulsion increases, even though the main type of failure remains the complete rupture.

It is apparent that ruptures associated with avulsions are determined by bone strength, and as a result, they depend on body mass density (BMD). The lower one's BMD, the higher the risk of an avulsion type failure occurring prior to a tendon rupture [30]. Because BMD tends to gradually increase with age and subsequently decreases during the onset of old age, the prevalence of avulsion type failure differs throughout the different stages of life. While the prevalence of avulsion failures differs with age, it also

tends to have a greater effect on women due to the fact that women, on average, have lower BMD rates when compared to men.

1. Introduction of Repair Methods

Considering the fact that the Achilles is loaded with about 5x the body weight simply during walking, and knowing that the tendon is incapable of adapting to high in vivo stresses by developing correspondingly high material properties as other tendons do [31], it is very clear that the risk of injury in this area is high. In the case of extreme athletics, the loads applied to this tendon are greatly increased, and failure is often a result. Tendon healing is a very complex process involving angiogenesis, cell proliferation and deposit of the extracellular matrix. Following these processes, remodeling and maturation of the tendon is carried out, allowing the tendon to ultimately regain its mechanical strength [8]. In order to repair the tendon, however, a variety of methods exist, yet the most preferred method remains controversial. In any repair situation, reconstruction of the tendon can be very challenging as the blood supply at its insertion to the calcaneal bone is extremely poor [19]. Accordingly, it is imperative that no matter the chosen repair method, long-term external fixation, avoidance of loading, and/or postoperative rehabilitation are accounted for in order to prevent loading on the tendon until a fully functional tissue can form [16].

It is evident that different treatment approaches are needed for each type of injury [30]. The two main treatment types are a surgical repair method and a conservative repair method, however treatments including eccentric exercises and the application of autologous platelet growth factors do exist as well [6, 10, 21]. For the most part, surgeons

have shown to prefer the surgical repair in situations with higher levels of pre-injury activity, largely due to the low re-rupture rates [15, 29]. Typically, however, the incidence rate of surgical complications is high and this is one of the main reasons why the two methods remain debated. Chalmers et. al show a 20x higher rate of minor and moderate complications in surgically treated patients than in those treated non-operatively. In the review paper by Khan et. al pooling data from twelve articles, the rate of re-rupture in operatively treated patients was 3.5% (six of 173) while the risk increased to 12.6% (twenty-three of 183) in the non-operatively treated group. Nonetheless, the rate of re-rupture is largely decreased in surgically treated patients [6].

Through an examination of a variety of studies in literature today, it is possible to examine the benefits of both the surgical and conservative method, and possibly suggest circumstances in which one is more preferable than the other. It is evident that a variety of factors including pre-injury activity, amount of time elapsed before presenting for treatment, recovery time, and the patients stance on risk of complications all play a major role in this determination [20].

2. Surgical Repair

Surgical repair is represented by early functional therapy in which the tendon is repaired surgically and a period of immobilization follows. Surgical repair is required in 25% of Achilles tendinopathy patients and is more often than not the chosen method for younger, middle-aged and very active older adults [1, 8]. When repaired surgically, the tendon is placed under greater tension than in conservative repair, and this has proven to help quicken the healing process. Surgical repair as a whole appears both better and

stronger than conservative repair, ultimately leading to a lower re-rupture rate and a quicker rate of resumption of sports activities [6, 21]. At the same time, however, surgical repair is well-known for an increase in complications, specifically infection of the surgical wound and sural nerve injuries. A gradual thickening of the tendon following surgical repair has also been noted. As a result, surgery may not be advantageous for patients who have significant surgical risk factors [1].

Worth et. al sent a questionnaire to 244 orthopedic consultants regarding surgical versus conservative repair. Most consultants preferred surgical repair, specifically because the mean duration of immobilization was shorter (2-12 weeks) than that of conservative repair (19 weeks). With higher levels of pre-injury, surgeons also found it imperative that surgical repair was the chosen method as the tendon required a quick fix followed by immobilization in order to heal properly. While the higher the level of pre-activity is directly correlated with the longer the length of immobilization, it is imperative to understand the importance of the immobilization step in the healing process. Separation of the tendon occurs following any surgical repair due to the high loads that run through the Achilles, and this period of immobilization is crucial in allowing the new tissue to fully form and a full recovery to be achieved [6].

In a study by Maffuli et. al, 41 females and 45 males underwent surgery after already undergoing a failed conservative treatment. Gradual progression to full sports activity at 16-24 weeks from the operation was accepted; however resumption of competition was not recommended before six months. It is important to note, however, that while many of the male patients experienced positive recovery, only 24 of the female patients achieved satisfactory recovery. It is possible that the higher body fat content in

females influences an increased post-operative complication rate. Were these females to have opted for the surgical repair method initially, it is still not understood if a different outcome would have resulted. In general, however, it must be noted that when conservative treatment fails to revive the tendon, surgical repair becomes the next step. This provides strong evidence that surgical repair is more beneficial overall, and that it is simply one's interpretation of the risks that may stray them from choosing this method in the first place.

In the study by Cetti et. al, 60 patients with ruptured Achilles tendons were all treated surgically by the same surgeon. All tendons were diffusely thickened in comparison with the contra-lateral healthy tendon, and degeneration, tenocyte necrosis, and acute inflammation were found in all sites of rupture. The degenerated collagen had irregular, thin, wavy and angulated fibers which formed structure-less, basophilic areas. Other small vessels in the tendon showed swelling of the endothelium. Considering this study involved 50 men and only 10 women, it can be suggested that the return to sporting activity was one of the major requirements for these rupture patients, and thus the surgical approach was opted for. Again, as we can see through Cetti et. al's study, the levels of pre-injury and the desire to return to normal activity more rapidly directly influence the selection of repair method.

All in all, it seems very clear that surgical repair is the best method for patients with high pre-injury activity and a necessity to return to their sporting events as soon as possible. While the risk of complications is greater in these repairs, as can be seen through the comparison of studies in Table 1, for young, active, healthy patients, these risks are minimal. Consequently, for a patient with a high risk for surgical complications

and a lower necessity to return to sporting events early, the conservative option may be superior.

3. Conservative Repair

In conservative repair, collagen fibers within the scar, which grow between tendon ends of post-rupture, become organized and oriented to resemble the structure of a normal, healthy tendon. If these ends are held in close apposition, natural repair will occur without lengthening and virtually normal function of the tendon can be restored [6]. Conservative treatment can include anything from rest, anti-inflammatory drugs, steroid injections, orthotic devices, attainment of adequate gastrocnemius/soleus strength and flexibility, proper training methods, and respect for the post-symptomatic phase [8]. Such processes involving heat treatment, ultrasound, and electrical stimulation are also commonly used. Once treated, immobilization is required and the use of a raised heel or cast is necessary. While one of the main advantages noted for this nonsurgical treatment method is the elimination of wound complications and the eradication of intra-operative sural nerve damage, a variety of disadvantages exist [15].

Metz et. al show that conservative treatment with cast immobilization not only increases the re-rupture rate, but also induces delayed recovery as the strength of the calf muscle is largely compromised as a result of long term immobilization of the ankle joint. Majewski et. al confirm the delayed recovery through a comparison of early functional therapy (surgery) and cast immobilization. On average, patients required 67 days of healing before returning to work after cast immobilization and only 37 days of healing after early functional therapy. While all patients were capable of returning to their

sporting events within one year after the injury in both methods, the lengthier immobilization has serious consequences for the entire musculoskeletal system. Consequently, several reports indicated that orientation of collagen fibers and the strength of the calf muscle are improved with surgical repair largely due to the fact that the tendon is under adequate tension for healing [14]. In addition, when re-rupture occurs in a conservatively treated tendon, more often than not, surgery is chosen as the repair method the second time around [20].

Whereas the majority of studies simply compare one repair method to the other, Chalmers et. al introduced a separate comparison involving the time lapse between rupture and treatment. In the case of 105 patients randomly assigned to one treatment or the other, 52 were treated conservatively and the remaining 53 were treated surgically. If treated within 48 hours, the results in terms of strength of plantar flexion, range of movement, and rate of re-rupture (0) were comparable between the two methods. If treated after 48 hours, however, the strength of plantar flexion was greatly reduced in the conservative group and the incidence of re-rupture increased.

While the majority of papers comparing surgical and conservative repair all contain different percentage rates of re-rupture, different immobilization periods, and different strength characterizations of both plantar flexion and calf strength (shown in Table 1), it is clear that nonsurgical, conservative treatment is not always successful and has proven to be relatively unreliable, not impressive and inadequate [6, 8]. More specifically, significant partial ruptures respond very poorly to conservative treatment and do not improve over time [8]. The requirement of physical therapy and multiple cast changes instigates a larger dependency on healthcare resources [14].

While these negative aspects may be prevalent in the majority of conservative repairs, nonsurgical repair remains a perfectly fine method for older, inactive patients as well as in patients at high risks for surgical complications [1]. Wound healing complications may pose a greater threat in some patients, and as a result, conservative treatment is advantageous. According to the study by Metz et. al, there is a 3% reduction in complications in conservative treatment when compared to the surgical treatment.

As one can see through the literature today depicting the comparison between conservative and surgical treatment, the major factor in determining what treatment option is best is determined on a case by case basis. For elderly patients, conservative treatment is the best option as complication risks due to surgery could be detrimental to their health. Surgical repair, however, is most beneficial for young and middle-aged patients who are interested in returning to work and to their sports activities more quickly. The shorter recovery period following surgical repair allows the calf to be put back into use sooner and reduces the risk of re-rupture [1]. While many of the journals in literature today point out the surgical complications as the largest complaint to this repair method, it must be noted that these complications are not deathly and arise in any surgical situation. Simply because conservative repair is an option for Achilles tendon repair that does not involve a high rate of complications does not mean it is the most favorable. All in all, the surgical repair method is the best method for athletically induced Achilles tendon ruptures for young and middle-aged patients while conservative repair is favorable in elderly patients.

Study	Mean Age at Rupture	Surgically Repaired	Conservatively Repaired	Re-rupture rate (Surgical)	Re-rupture rate (Conservative)	Compli-cations (Surgical)	Compli-cations (Conservative)
Chalmers, J. [6]		53/104	51/104	4%	8%	29 in 44 patients (20x higher than conser-vative treat-ments)	0
Ng, ES et. al [16]	36 in open repair, 34 in needle	68 (43:open, 25:needle)		1 in open repair group		1 sural nerve injury in open	
Cetti et. al (2003) [5]	37 (20-60)	60	0				
Maffuli et. al [12]			86 (41 female, 45 male)			12: suffered from super-ficial infection of the surgical wound, 8: hyper-sensitivity of surgical wounds, 4:devel-oped hyper-trophic scar, 8:under-went further surgery	

Worth et. al [28]						less frequent re-rupture rate	fewer soft tissue compli-cations, faster return to work
Khan et. al (review article):							
Nistor (1981)		45	60	2 out of 45	5 out of 60	31/45	0/60
Cetti(1993)		56	66	3 out of 56	7 out of 55	16/56	3 out of 55
Schroeder (1997)		13	15	0 out of 13	0 out of 15	2 out of 13	0 out of 15
Moller (2001)		59	53	1 out of 59	11 out of 53	10 out of 59	2 out of 53
Totals		173	194	6 out of 173	23 out of 183	59/173	5 out of 183

C. Metabolic Achilles tendon Ruptures

Because the majority of Achilles tendon ruptures in literature are generally the result of repetitive injury, it is essential to understand the unusual causes such as drug-induced and pathologically induced tendinosis and tear [11]. In so many cases, these types of ruptures are overlooked simply because they occur in the second peak of Achilles ruptures, those that occur to people who fall within 70 and 80 years of age, when a rapid return to health is not as essential. These types of ruptures do not occur in the same manner as do athletically-induced ruptures, and in many cases, patients who experience steroidal-induced rupture are not completely certain that they have ruptured the tendon. The snapping sensation that can be felt in athletic ruptures is not present in pathologically induced ones. Instead, the constant weakening of the tendon through degeneration of the collagen matrix and the influence of various quinolones simply forces the tendon to give out. Thus, this type of rupture is more of a gradual weakening as opposed to a sudden dislocation of the tendon from the calcaneal insert.

Pathological rupture can be attributed to a variety of factors including medial hypertrophy during corticosteroid administration, ischemic changes due to vascular narrowing, reduction of fibroblast activity, and protein catabolism [15]. The lack of severe symptoms prevents the early diagnosis of these ruptures, and the rupture of the tendon is, as a result, secondary to other trauma [15]. Inflammatory diseases such as Rheumatoid Arthritis (RA) and aging are among the most important factors leading to this second peak of Achilles tendon ruptures. For example, as shown in the study by Mikashima et. al, blood circulation through the Achilles tendon in RA patients is

dramatically reduced in patients over the age of 30, placing the tendon under ischemic conditions which may eventually lead to rupture.

Patients with chronic diseases who are treated with long-term corticosteroid therapy are extremely prone to Achilles tendon ruptures as these steroids cause a weakening of the collagen structure of the tendon [20, 21]. The collagen structure becomes altered due to the presence of multiple micro-traumas. The combination of a decreased rate of blood flow in older patients and the avascular properties of the tendon accelerates the production of these micro-tears and initial weakening of the tendon begins [5, 9]. As these changes occur, an acute inflammatory response is triggered which can further weaken the tendon until it is no longer capable of resisting mechanical forces even during walking, and a rupture results. In healthy patients, minute tears in a degenerated Achilles would be expected to heal, however in older patients induced with corticosteroids and fluoroquinolones this is not the case [20]. Kim et. al explain that corticosteroid use does not allow the tendon to repair adequately, and as a result, irreversible matrix alterations lead to rupture.

In most cases, bilateral ruptures result. While in the majority of athletic injuries spontaneous ruptures result, these bilateral ruptures occur due to a sudden dorsiflexion of a plantar-flexed foot containing a degenerated tendon [9]. As a result, patients with a type O blood group, gout, hyperthyroidism, and rheumatoid arthritis are all at a higher risk of obtaining a damaged Achilles tendon due to the impact on the tendon of the steroids used to treat these illnesses [20, 22]. Patients with inflammatory and autoimmune conditions, genetically determined collagen abnormalities, infectious diseases, tumors and neurological conditions are also at a higher risk as these conditions are generally not of

primary surgical nature and must instead be treated using steroids, or more specifically, using corticosteroids [2, 9].

Hyperthyroidism causes decreased synthesis and degeneration of the collagen structure by inhibiting epimerase. The inhibition of this enzyme reduces the amount of chondroitin sulfate and elevates the levels of hyaluronic acid, together weakening the collagen matrix. Hypercalcaemia arises due to hyperthyroidism and causes calcification of the tendon and small vessels, ultimately decreasing the vascularity and reducing the strength of the tendon. This eventually leads to delayed healing, and consequently, the minute tears build up overtime and become too much for the tendon to handle [20].

In patients with Rheumatoid Arthritis (RA), Ames et. al noted that Achilles tendonitis has been reported in up to 64% of the cases, however only a few full ruptures can be considered directly attributable to the disease. Stafford et. al subsequently concluded that while Achilles tendonitis is commonly associated with inflammatory arthritic diseases, including RA, very few ruptures have actually been recorded. Another study confirmed that symptoms referable to the lower limb were four times more frequent than elsewhere in the body in these patients[26].

While many studies suggest that full ruptures are not as common in RA patients as previously suggested, it is still apparent that corticosteroid treatment has a profound effect on inflammation of the tendon. Vidifal et. al examined patients with RA who reported having chronic heel problems and these researchers found that in a study of 43 patients, many suffered from pain in two well defined regions: at the insertion of the Achilles tendon into the os calcis, and at the insertion of the plantar fascia into the os calcis. Through further examination, it was observed that the area of insertion of the

Achilles into the calcaneum had been eroded in ten patients. In the study conducted by Mikashima et. al, histologic examination of ruptured tendons in RA patients revealed rheumatoid granulation comprising lymphocytes, histiocytes, and small vascular proliferation within the tendon tissue. These results ultimately suggest that degenerative changes are indeed present in the tendon prior to rupture, and a more thorough examination may be beneficial in order to prevent such ruptures from occurring. It is the lack of severe symptoms that really allows the degeneration of these tendons to go unnoticed. Looking into the future, it is clear that many of these ruptures can be avoided if better examinations are carried out earlier in patients carrying the disease [15].

Corticosteroids have also shown to increase the rate of onset of diabetes mellitus which, in due time, leads to problems with the Achilles tendon. Diabetes mellitus is a diffuse endocrine disease characterized by metabolic abnormalities and long-term complications. Non-enzymatic glycation of proteins results in accumulation of glycosylated end-products in most human tissues, specifically tendons, ultimately altering the biomechanical properties of these tendons. In study by Akturk et. al, electron microscope investigation of the Achilles tendon demonstrated increased density of collagen fibrils, abnormal fibrin morphology and decreased fibrillar diameter. A significant thickening of the tendon was also noted along with increased vascularization.

This increase in the density of collagen fibrils causes a dramatic increase in stiffness within the tendon, ultimately affecting the stretch distribution between the muscle and tendinous tissues during simple motions as walking. When the distribution becomes altered, rupture results [7]. During normal walking, the muscle-tendon units of the lower limb are naturally stretched; however, this implication of increased tendon

stiffness forces an increase in the stretch transferred to the muscle fibers and a decrease in the movement efficiency as the elastic energy storage within the tendon depletes [7]. While diabetes mellitus results in complications in the foot-ankle complex due to this depletion of elastic energy, the presence of complete rupture is very minimal. While in this disease scenario the use of corticosteroids cannot be directly attributed to rupture, it is still evident that these steroids negatively associate with the tendon and subsequently have an adverse effect on movement efficiency.

While it is clear that corticosteroids have a particularly strong influence on Achilles tendon ruptures through their instigation of tendon atrophy and weakening [11], the presence of fluoroquinolones may be even more dramatic [19]. Fluoroquinolones are antibacterial agents that act by inhibiting bacterial DNA gyrase [24]. DNA gyrase is directly involved in DNA replication and cell division [11]. Due to their broad spectrum, their relatively few serious adverse reactions and their good oral absorption, these steroids are among one of the most frequently prescribed antibacterial agents [23]. They can be used to treat infections involving the respiratory, urogenital, and gastrointestinal tracts.

While they may be beneficial in the treatment of certain infections, fluoroquinolones exhibit a pronounced affinity for connective tissue and have the ability to disturb the physiological interaction between cells and the collagen matrix by chelating divalent ions [23]. In doing so, these quinolones become ideal for joint and bone infections as their chelating properties cause direct toxicity to type 1 collagen synthesis and ultimately promote collagen degradation. Fluoroquinolones then further increase tendon risks as they induce chronic inflammation and overproduction of the parathyroid

hormone [1]. While many types of fluoroquinolones exist, ciprofloxacin and ofloaxin remain among the most popularly involved in tendon rupture. In the study by Kim et. al and van der Linden, both ciprofloxacin and ofloaxin exhibited strong associations with tendon disorders, however ciprofloxacin was found to be the causal agent in 90% of the cases of Achilles tendon problems. Subsequently, the risk of tendinopathy appeared to be dose independent.

The average age of fluoroquinolone induced tendinopathy is approximately 64 years, with a male to female ratio of 2:1. While these steroids can be used to treat a variety of inflammatory diseases within the body, more than 95% of the cases of resulting tendinopathy involve the Achilles tendon. Kim et. al suggested that the rapid and immense loading during weight-bearing activities places the Achilles tendon at a greater risk than other tendon sites, and in due course, allow fluoroquinolones to demonstrate a 3.8 fold greater risk for development of Achilles tendinitis and subsequent rupture [11].

In patients using fluoroquinolone treatments, the Achilles tendon can become affected with symptoms compatible with painful tendinitis or rupture within the first two weeks of treatment [24]. In the study by van der Linden [24], 97 out of 1841 users of fluoroquinolones registered side effects dealing with tendinitis or tendon rupture. In another study by van der Linden [23], the majority of quinolone-associated Achilles tendon ruptures occurred within one month after beginning a 7-to-10 day course of treatment. In the study by Kim et. al, 50% of tendon ruptures occurred within one week of fluoroquinolone administration. Out of a total of 1367 cases, the odds ratio of rupture in patients ages 60-79 was 6.4% while in patients 80 years and older, the odds ratio of rupture was 20.4%. This study showed that the risk of Achilles tendon rupture is strongly

dose dependent and does not depend on the duration of use of quinolones. The effect of the quinolones was not suggested to be modified by sex, but instead showed evidence that age was an important modifier [11, 23].

While both fluoroquinolones and corticosteroids each display their own individual effect on Achilles tendon inflammation and ruptures, van der Linden et. al [23] showed that the risk of Achilles tendon rupture was highest among elderly patients (older than 60yrs) who were concomitantly treated with corticosteroids and fluoroquinolones. While it is important to note that the absolute overall risk of Achilles tendon ruptures is low, even in patients 80 years and older, still, approximately 2%-6% of all ruptures in people older than 60 years old can be attributed to the use of fluoroquinolones [23]. In van der Linden et. al's study [23], it also became apparent that while patients with obesity, hyperparathyroidism, musculoskeletal disorders and diabetes are all at risk of experiencing Achilles tendinitis and/or rupture, their risk potential is exacerbated by concomitant fluoroquinolone and corticosteroid exposure.

Although much is understood regarding how these steroids work and it is agreed that they have a profound effect specifically on Achilles tendon tendinitis and resulting ruptures, Read et. al show that no comparisons have yet to be made between a 'normal' spontaneous rupture and a pathologically induced rupture [19]. A pathophysiological mechanism linking tendinitis to fluoroquinolones still remains unknown [9]. In a study pertaining to Achilles tendon ruptures in 83 patients treated with steroid injections, histological changes of the Achilles tendinopathy showed both fibrinoid degeneration and macroscopic changes of fibrillation, cleavage, tissue paper tears and focal degeneration [19]. Similar findings have been found in 'normal' ruptures, and thus the direct

consequence of these steroids remains unclear [11]. Read et. al propose that there quite frankly may not be a difference. Instead, what became clear through their research, in which 67% of the patients treated with steroids acquired severe injuries in comparison to 37% in the non-steroidal group and 43% of the steroid group had experienced a recurrence of injury while only 16% did in the non-steroid group, is simply that local steroid injections are not beneficial. Steroids may be used to prevent or reduce inflammation; however, in terms of healing degenerative changes within the tendon, they are useless [19].

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Part II.

Analysis of the Achilles tendon and collagen matrix using Finite Element Analysis

Introduction

The structure of the foot is a very vital connection between the human body and the ground and is involved in almost every movement during locomotion [6]. Being able to understand the stresses and strains within all the different structural components not only shines light on a better understanding of certain disorders [6], but also allows for both the prevention of injury and the development of advanced shoe orthotics. Knowledge of the effect of soft tissue compliance or other structural characteristics on the stress distribution of the plantar foot surface and bony structures is absolutely essential for achieving individualized treatment when necessary [3]. Techniques for plantar pressure measurements and orthopedic shoe implants are very well established using computer aided design mechanisms, however a direct measurement of internal stresses and strains within the foot has proved trying [1, 3].

After completing a comparative literature review regarding the differences between athletically induced and pathologically induced Achilles tendon ruptures, it became evident that a computer model of the Achilles tendon was not a common entity. Because a majority of the factors regarding Achilles tendon ruptures were understood through comparisons of multiple research studies analyzed in the literature review, it appeared worthwhile to put these values into a CAD software program. Upon doing so, it would be possible to analyze whether the same results occurred through computerized testing as had occurred through the use of cadavers and healthy human subjects. Advantages of using interactive graphics to model the musculoskeletal system were first described in 1977, and since then, an explosion in the advancement of computer technology has allowed many to interact with models of this system [4]. Models of the musculoskeletal system can not only provide insight into treatment options, but can also

aid in the understanding of the biomechanical consequences of certain surgical procedures. Subsequently, surgeons and other doctors acquire the ability to alter difficult surgeries and design more effective procedures in order to increase the likelihood that the outcomes are positive [4].

Finite element analysis, a simulation aspect of the SolidWorks program, has been used increasingly in many biomechanical studies with great success. Due to its capability of modeling structures with irregular geometry and complex material properties, many more in depth analyses have been carried out that previously were not available. Finite element analysis also allows for easy simulation of complicated boundary and loading conditions under both static and dynamic settings [1]. As a result, an attempt to construct the Achilles tendon and the calcaneal support was conducted using SolidWorks and a simulation was run using Finite Element Analysis (FEA).

Whether it is due to overuse or steroidal effects, the collagen matrix within the Achilles tendon is understood to play a major role in the tendon's degradation. Therefore, in this study, while the Achilles tendon and calcaneus model remains quite linear, finite element was used to depict the effect of differing collagen matrices. Using the material properties of type 1 and type 3 collagen, along with a third intermediary collagen matrix value, an analysis of the tendon under common force loads was carried out, and a comparison between the collagen models was explored. During degradation, the percentage of type 1 collagen has shown to decrease, eventually becoming replaced by type 3 collagen [16, 19]. Understanding that type 3 collagen is much weaker, especially in tension, than type 1 collagen, it is hoped that the type 3 collagen matrices will depict a weaker tendon at increasingly higher loads. It is also hoped that the main locations of

stress on the tendon itself occur in similar locations as noted in previous studies; between 2 and 6cm above the tendon insert. With the production of a working Achilles tendon model, it is possible that further research studies can be done to analyze the effects of surgical and conservative treatment upon the tendon. Ultimately, due to the creation of a tendon-calcaneal model, advances in the controversy between the two most common surgical methods may arise.

Methods

Type 1 collagen has the highest mechanical properties out of all types of collagen, while type 3 collagen appears to exhibit the weakest strength. Type 3 collagen generally appears during the construction of new tendons and ligaments, and is therefore more of a foundation material than a material of high tensile strength [13, 16]. Gradually, type 1 collagen takes over, giving the tendon its lifelong strength. As understood during previous research, it is evident that as an Achilles tendon begins to weaken, the amount of type 3 collagen increases. Subsequently, the overall strength of the tendon is compromised and the probability of a rupture increases. This takeover of type 3 collagen occurs because as the tendon weakens, it experiences many small microtears. These microtears do not work as normal fractures do, in which the build-up of small fractures converge and eventually cause a tear in the material. Instead, the build-up of microtears promotes the work of type 3 collagen to lay down new foundations in order to rebuild the tendon. As a result, the amount of type 1 collagen decreases in comparison to the amount of type 3 collagen, and the tendon's strength is compromised. Consequently, the mechanical properties for a high strength collagen (type 1), a low strength collagen (type 3), and an intermediary strength collagen (average between type 1 and type 3) were used

in this analysis. The intermediary strength collagen was used to represent the period in which the tendon is made up of a combination of both type 1 and type 3 collagen.

In order to create these three collagen materials within the SolidWorks program, it was necessary to input values of Young's Modulus, density, Poisson's ratio, Yield Strength, and Tensile Strength. The corresponding values of the three different strengths of collagen are shown in Table 1: Collagen Matrix Values in Achilles Tendon and Calcaneus. The values for the calcaneus were also found through previous studies and are shown in Table 1 as well.

Table 1: Collagen Matrix Values in Achilles Tendon and Calcaneus

	High Strength Collagen (Type 1)	Intermediate Strength Collagen (avg. between type 1 and type 3)	Low Strength Collagen (Type 3)	Calcaneus
Young's Modulus(GPa)	2.75	7	11.5	6
Density (kg/m^3)	1400	1400	1400	94
Poisson's Ratio	0.4	0.2	0	0.3
Yield Strength (MPa)	100	80	40	32
Tensile Strength (MPa)	220	120	50	20

This project represents one of the first attempts to depict the Achilles tendon and calcaneus through a computer aided design program. As a result, a linear model was used in order to study the effects of these various collagen matrices on the strength of the tendon under a variety of forces which represent a range of common everyday athletic activities. Due to limited resources and the lack of cadavers or MRI x-rays, a simple

cylinder was used for the calcaneus. It is clear that limitations exist in this assumption, including the fact that the ground force applied will not be distributed as it would in a normal, bony calcaneus. The widely accepted and most important concept of a rigid connection between the calcaneus and the Achilles tendon, however, is present. The tendon itself is modeled as a thin rectangular piece with varying widths, as depicted in previous studies [12]. The depth of the tendon remains consistent at 6.60mm throughout the entire 150mm tendon. Normal Achilles tendons range from 120 to 150mm, and therefore, this model depicts the larger scale end of tendon lengths. The remainder of the Achilles was constructed according to measurements shown in the diagram below (Figure 1: Achilles tendon measurements (mm)).

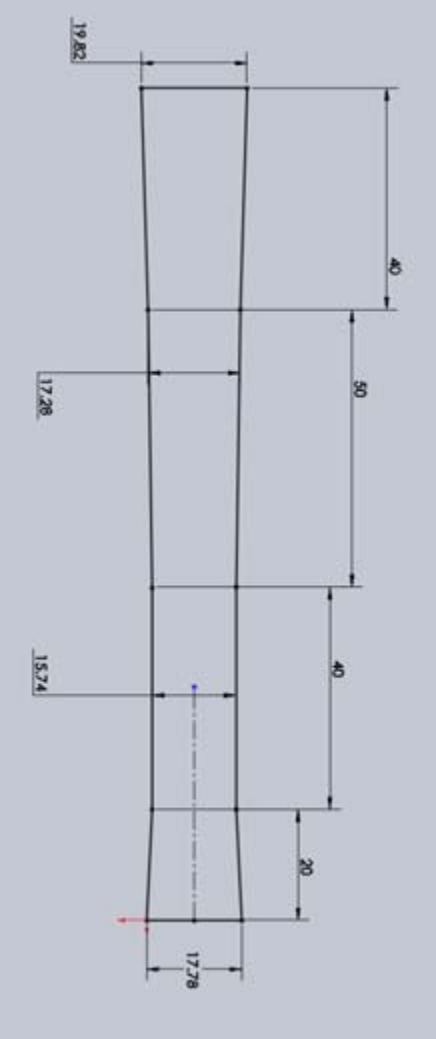

Figure 1: Achilles tendon measurements (mm)

Understanding that the Achilles tendon has been known to rupture frequently between 2 and 6 cm proximal to the calcaneus, it is hoped that as the magnitude of force increases, the highest stress will be seen within this range. As the tensile value of the collagen decreases from type 1 to type 3, the tendon should exhibit a weakening and the overall strength of the tendon should decrease. This decrease in strength should be well represented through both factor of safety plots and strain plots within the FEA analysis. Although the stress plots are expected to remain consistent among the three materials and

only change when the force is adjusted (due to the fact that the force is applied over a constant area), the factor of safety plots and strain plots will depict whether or not the differing collagen matrices actually play a role in the tendons degradation.

In order to analyze the model, two forces and a variety of connections were input into the computer aided design program. A ground reaction force of 350N was applied to the sides of the calcaneus through six force vectors [1, 3], and the Achilles tendon and the calcaneus were rigidly connected. A contact set was applied in which no penetration between the tendon base and its insert into the calcaneus could occur. A global contact was applied in order to bond the calcaneus and the tendon. Lastly, the calcaneus ends were fixed to create a restraint for the study.

Furthermore, an increasing force from 700N to 9000N was applied at the top of the tendon through four force vectors [1]. The forces applied included 700N, 1500N, 5300N, and 9000N. These forces were chosen since they most resemble the forces applied to the tendon during standing (700N), walking (1500N), light running (5300N), and sprinting (9000N), and they mimic values that have been used in previous studies [1, 2, 3, 5, **7**, **8**, 9, 10, 14, 17, 18, 20, 22]. Because several muscles within the foot are responsible for movement and subsequently act on the tendon and calcaneus, these values represent rough averages for a typical 70kg male. In any subject, the parameters involved in gait will differ significantly depending on body structure characteristics and gender [6].

Finally, the assembly was meshed using a medium mesh and the studies were run. Displacement, von Mises stress, Principle Stress, Factor of Safety, and Strain plots were

acquired for the tendon under all loading conditions and all variations of collagen matrices.

Results

The displacement plot for each tendon remained unvarying throughout the entire study. This simply confirms that the force was applied constantly to the tendon. Were the displacement graph to have changed according to the force applied, it would suggest that the Achilles tendon was disproportionally distributing the force, and thus not acting as a linear model. Figure 1 shows the depiction of the displacement graph seen in all studies. As is evident through Figure 1, the greatest displacement occurs at the tip of the tendon, thereby representing the reasoning behind the stretching of the tendon *in vivo*. No matter the force applied, the greatest displacement will always occur at the top of the tendon, as this is farthest from the rigid connection with the calcaneal bone.

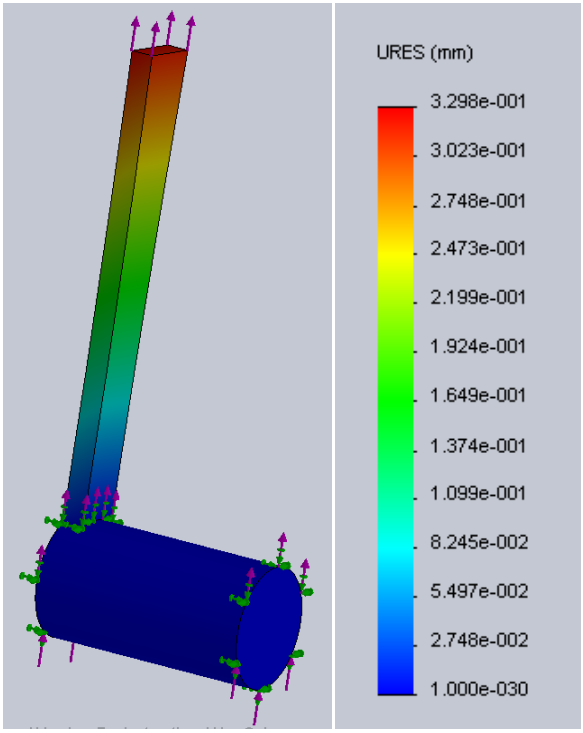

Figure 2: Displacement of Force through the Achilles Tendon

As far as the stress characterization goes, Figures 3, 4, 5, and 6 depict what was initially hypothesized. Because stress is the representation of force over area, and because the area of the tendon remained the same for each model under differing collagen matrices, the von Mises stress plots only vary depending on the force. These plots were taken from the type 1 collagen matrix tendon-calcaneal models; however the von Mises stress plots for the intermediary collagen matrix and the type 3 collagen matrix were exactly the same. The highest stress was found under the greatest load, 9000N, as would be hypothesized due to the fact that stress and force are proportionally related according to: $\sigma=F/A$.

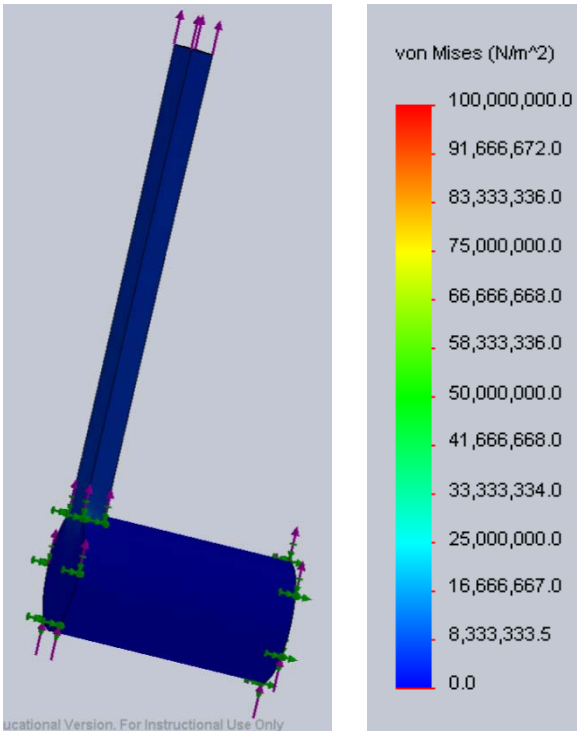

Figure 3: von Mises Stress Plot of Achilles Tendon (700N Force Applied)

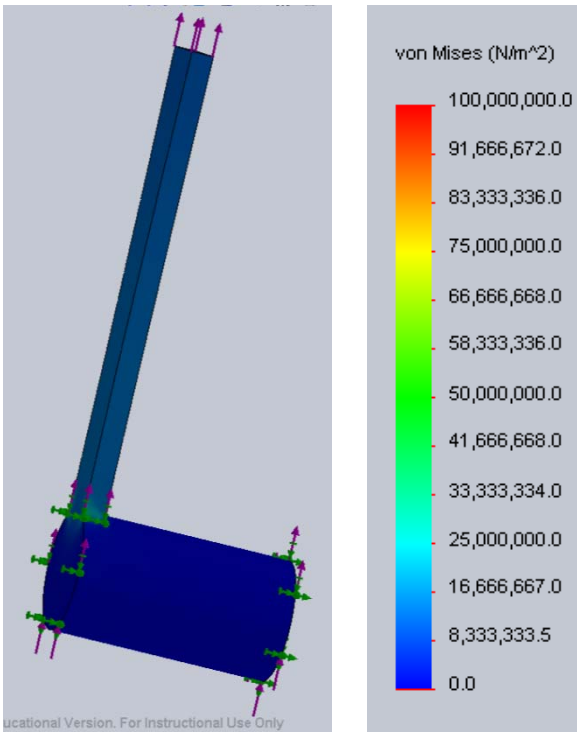

Figure 4: von Mises Stress Plot of Achilles Tendon (1500N Force Applied)

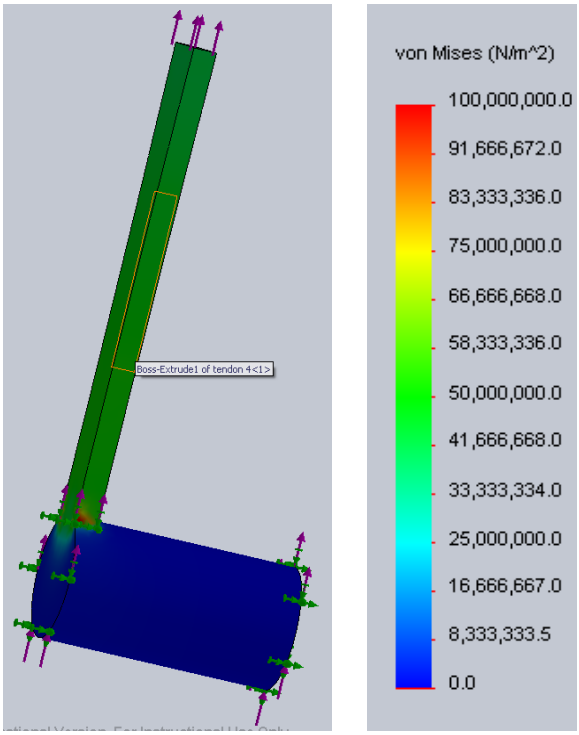

Figure 5: von Mises Stress Plot of Achilles Tendon (5300N Force Applied)

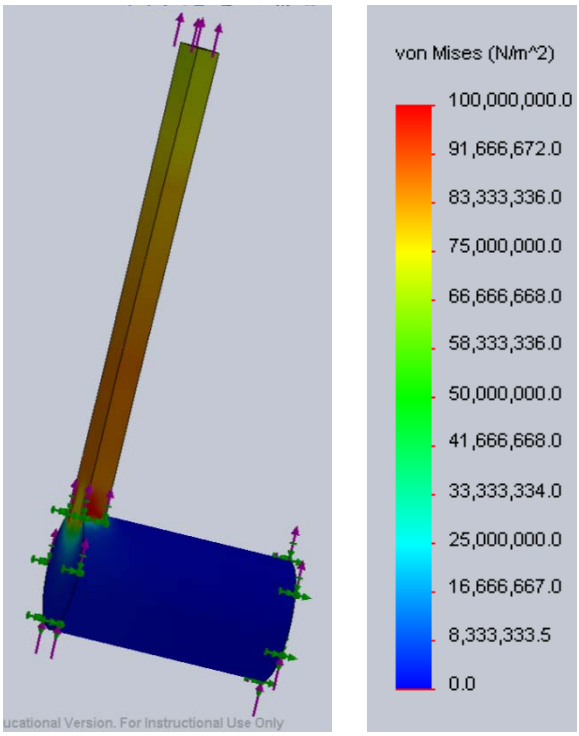

Figure 6: von Mises Stress Plot of Achilles Tendon (9000N Force Applied)

On the basis of *in vitro* measurements, the Achilles tendon has been reported to have a mean breaking stress of 111MPa [11]. As a result, the scales to the right of Figures 3-6 range from 0 to 100 MPa in order to show both where the tendon experiences its highest stresses, and subsequently, where the tendon may fail first under the given loading situations. Based on this understanding, the tendon shows aspects of failure under both the 5300N and 9000N loading situations. The areas of highest stress are right above the calcaneus, or roughly 2-6cm above the insert of the tendon into the calcaneus. The results of the stress plots are consistent with results from previous studies, with the highest stresses of 111MPa (breaking/peak stresses) occurring right above the calcaneal insert. It is important to note as well that stresses exceeding the breaking point only become evident when the tendon is exposed to extremely high forces. This result correlates nicely with situations of extreme physical activity (i.e. excessive running,

sprinting, sharp cutting). It is under these situations that most Achilles ruptures occurs, and the stress plots of the linear model only provide further support to these phenomenon.

While Figures 3-6 represented the von Mises stress, or the theoretical measure of stress used to estimate yield failure criteria and fatigue strength, Figure 7-10 depict the principle stress on the model [21]. Principle stress, on the other hand, is a direct representation of force over area, generally considered the more "real" measurement of stress [21]. Again, the principle stresses remained the same for each collagen matrix and only differed with a change in force. In comparison to the von Mises stress plots, the highest stress points lie relatively in the same areas, and again, the highest stress occurs with the highest force. The only real differences between these plots were the areas where failure would occur. Again, a scale of 0 to 111 MPa was used as a reference since the Achilles tendon has been noted to experience a peak stress of 111MPa. The areas where failure would occur in the principle stress plots fell slightly more toward the middle of the tendon as opposed to right on the edges, as seen in the von Mises plots. The difference in placement is very minimal however, and is still located between 2 and 6cm from the insert to the calcaneus bone. As a result, Figures 3-10 support previous studies which identify both the location of rupture (2-6cm above the insert to the calcaneus) and the forces under which rupture is most likely to occur (5300N-9000N).

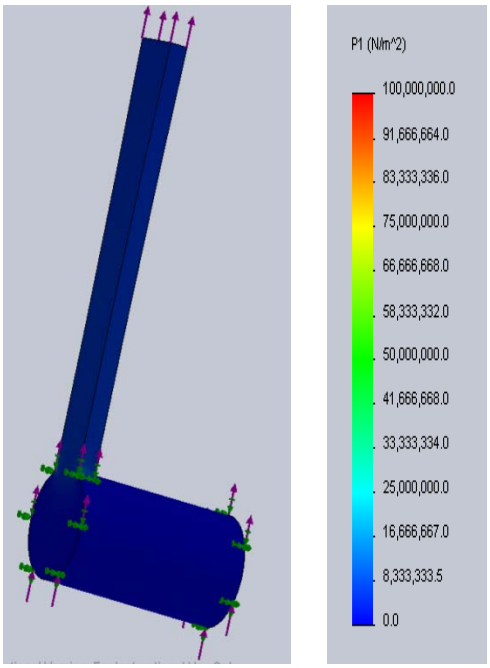

Figure 7: Principle Stress Plot of Achilles Tendon (700N)

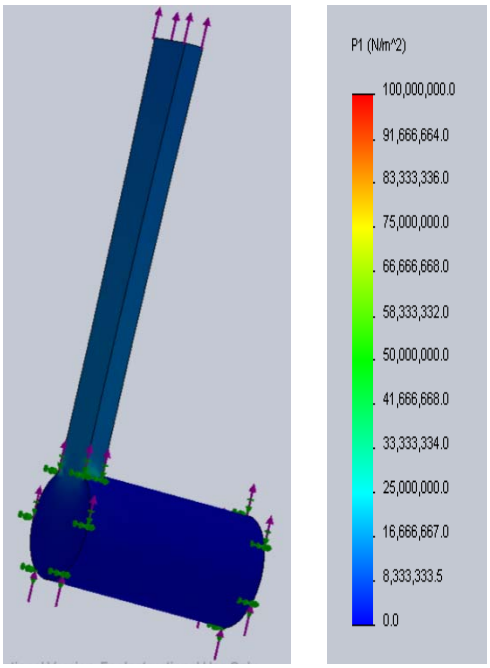

Figure 8: Principle Stress Plot of Achilles Tendon (1500N)

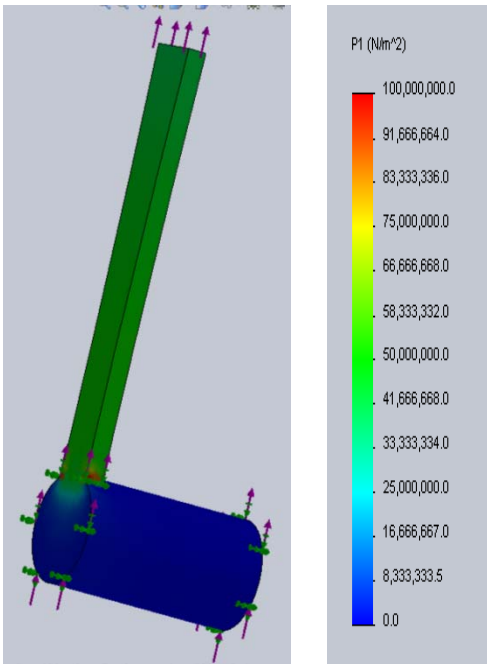

Figure 9: Principle Stress Plot of Achilles Tendon (5300N)

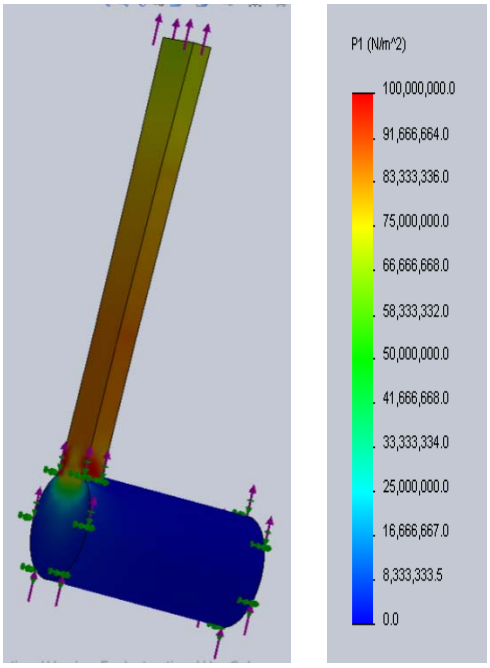

Figure 10: Principle Stress Plot of Achilles Tendon (9000N)

Figures 11 through 14 provide a comparison of factor of safety plots between the low strength (type 3), medium strength, and high strength (type 1) collagen matrices. Three plots were created per load, one for each collagen matrix, and the range of factor of

safety values was chosen to be 0 to 3. These values reflect those used in previous studies [11]. While many tendons in the human body have factors of safety reaching 4 and above, the Achilles tendon can undergo stresses as high as 70MPa during standing, and therefore has shown to only have a factor of safety, on average, of 1.5.

As can be noted in each figure, the tendon begins failing gradually as the collagen mechanical properties weaken in transition from type 1 to type 3 (left to right). It is noted that the factor of safety gets surpassed, or in other words, a rupture occurs, at the higher forces. This typically occurs right near the calcaneal-tendon insertion point. While type 1 collagen, or high strength collagen, exhibits gradual weakening from 700N to 9000N, it is evident that the extent of the weakening is not as great as it is under these loading conditions in the type 3 collagen matrix. As can be seen in Figures 13c and 14c, the tendon really begins to experience failure, as noted by the red coloring. This result helps portray what previous studies have alluded to- the fact that the tendon cannot handle as extreme forces when degrading as it can when it is healthy. At the same time, this model helps provide further evidence that an exchange in collagen matrices from high strength to low strength during degradation may very well be the major player in tendon ruptures.

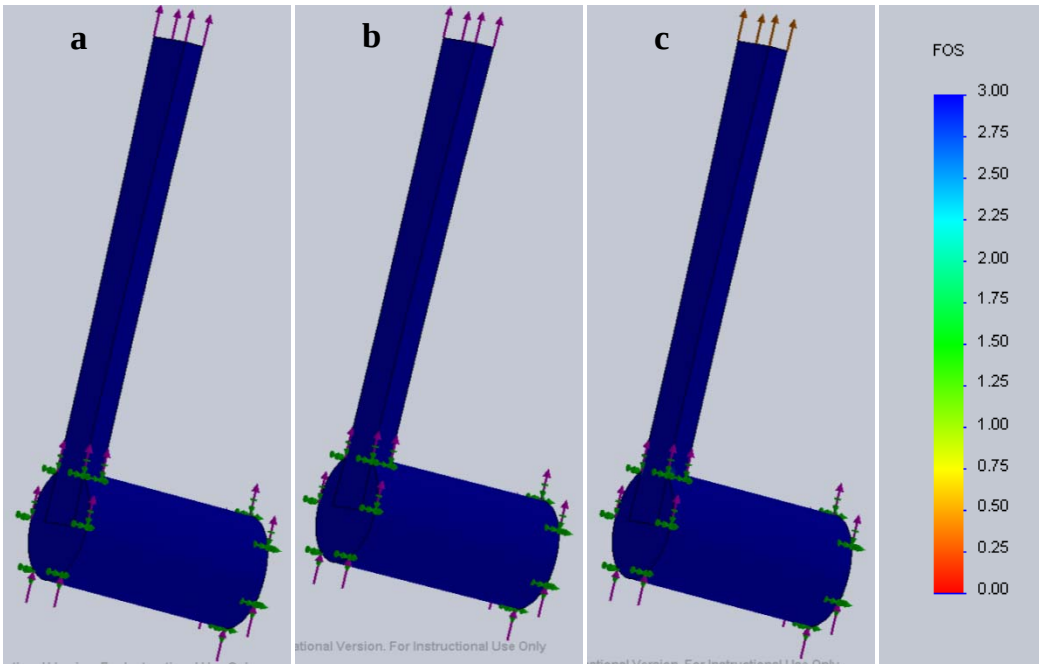

Figure 11: Comparison of Factor of Safety Plots (700N Force Applied) between a. High Strength Collagen; b. Medium Strength Collagen; and c. Low Strength Collagen

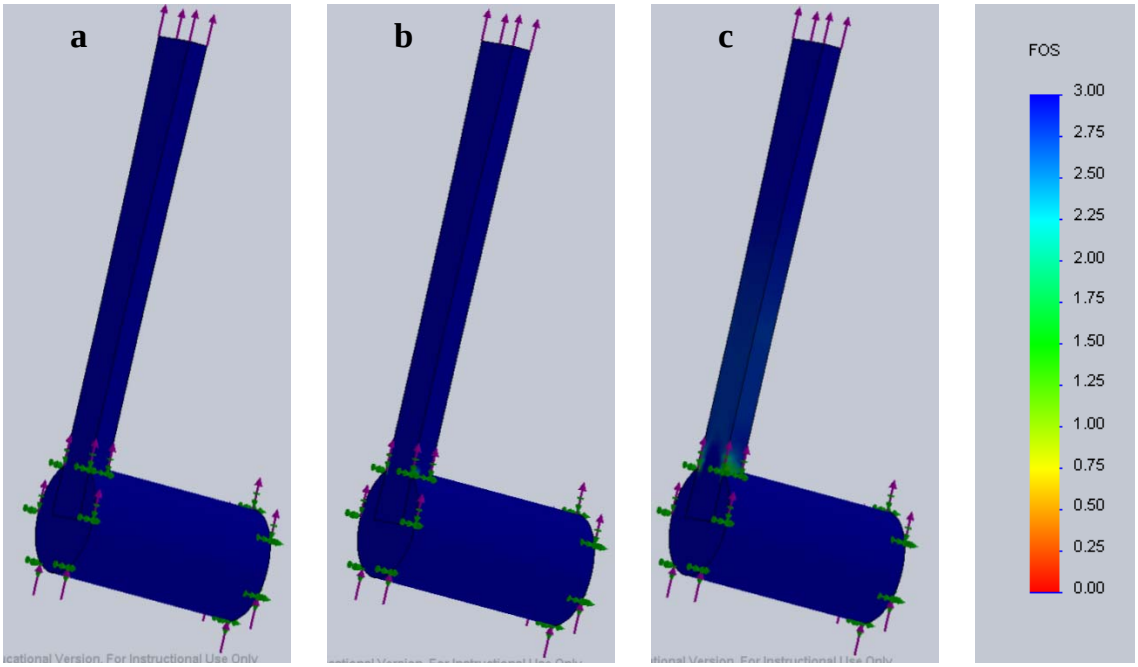

Figure 12: Comparison of Factor of Safety Plots (1500N Force Applied) between a. High Strength Collagen; b. Medium Strength Collagen; and c. Low Strength Collagen

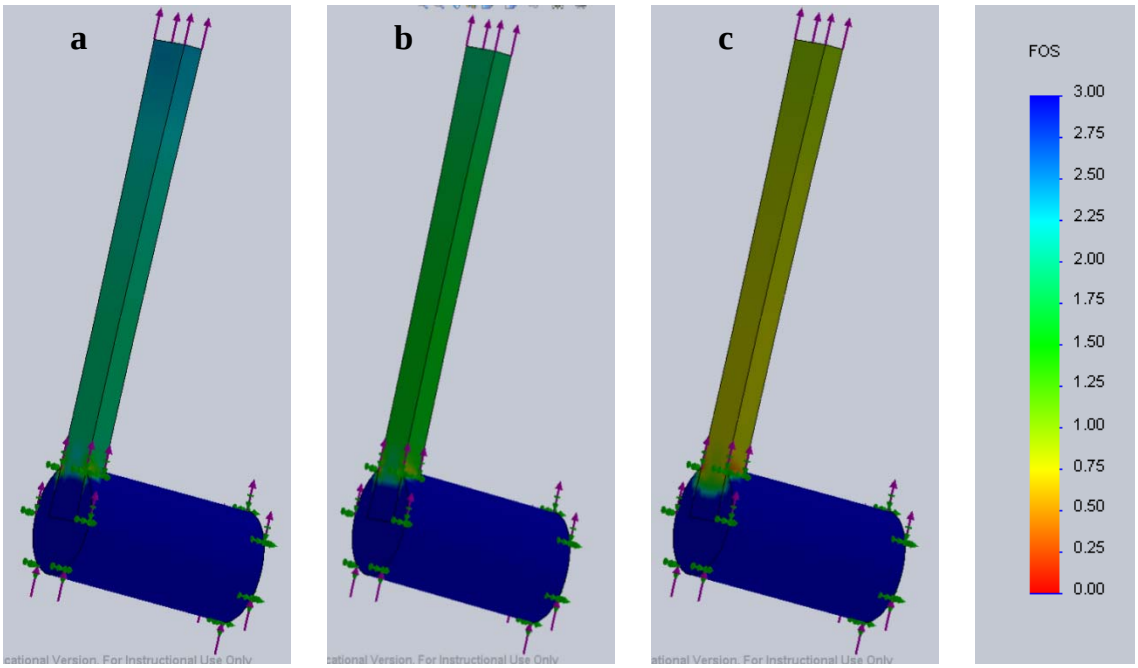

Figure 13: Comparison of Factor of Safety Plots (5300N Force Applied) between a. High Strength Collagen; b. Medium Strength Collagen; and c. Low Strength Collagen

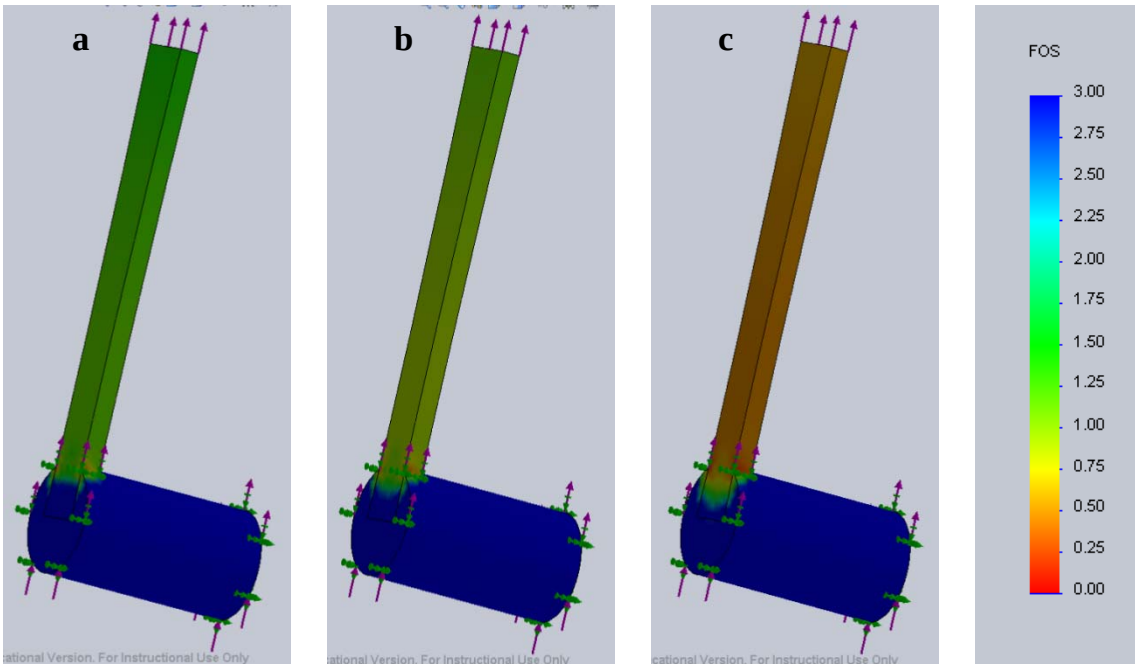

Figure 14: Comparison of Factor of Safety Plots (9000N Force Applied) between a. High Strength Collagen; b. Medium Strength Collagen; and c. Low Strength Collagen

Figures 15 through 18 represent the strain plots for the Achilles tendon model under the same loadings as previous stages. In this study, only the high strength collagen and low strength collagen matrices were used. It is expected that the intermediary collagen would produce an in-between result in comparison to the low and high strength collagen models. As is evident through the progression from Figure 15 through to Figure 18, with an increase in force applied, the strain on the tendon increases dramatically. Because the low strength collagen (the figures represented in 15b, 16b, 17b, and 18b) has a young's modulus (3.75GPa) that is much lower than that of the high strength collagen (11.5GPa), it experiences dramatically higher strains at higher loads. While the two models do not dramatically differ at the lower loads of 700N and 1500N, it is apparent beginning at 5300N and rather obvious at 9000N that the type 3 collagen matrix is much weaker. Again, the main area of failure falls a few centimeters above the calcaneus, subseqently suggesting that rupture is most likely to occur there during times of extreme activity. Ultimately, the strain plots illustrate best the negative effect that type 3 collagen can have on an Achilles tendon during continuous repair and regeneration, thereby supporting previous studies.

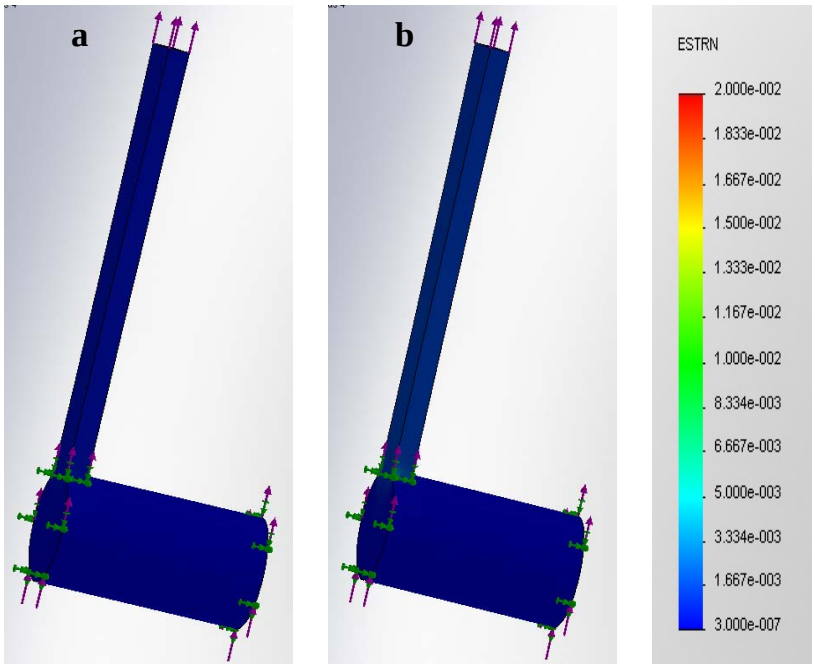

Figure 15: Comparison of Strain plots of Achilles Tendon (700N Force Applied) between High Strength Collagen (a) and Low Strength Collagen (b)

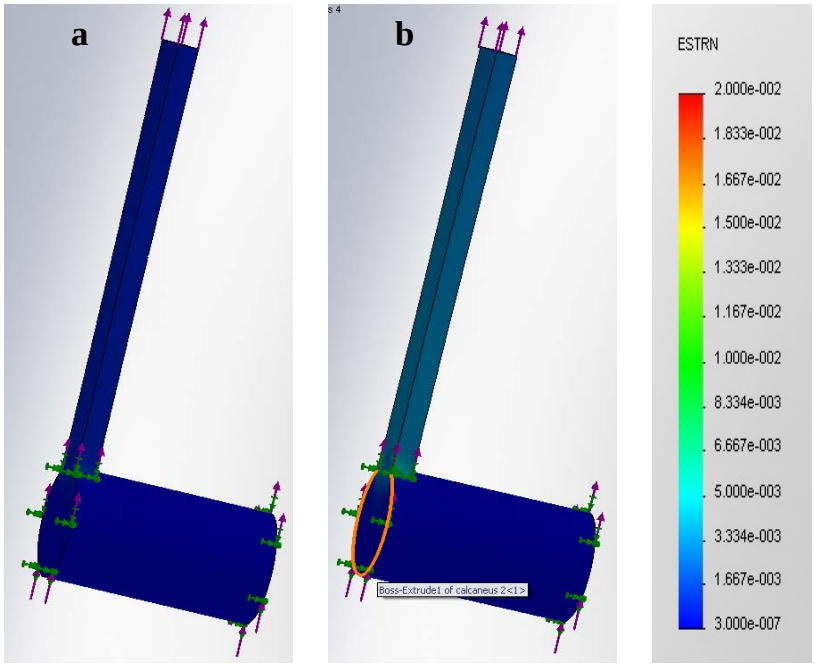

Figure 16: Comparison of Strain plots of Achilles Tendon (1500N Force Applied) between High Strength Collagen (a) and Low Strength Collagen (b)

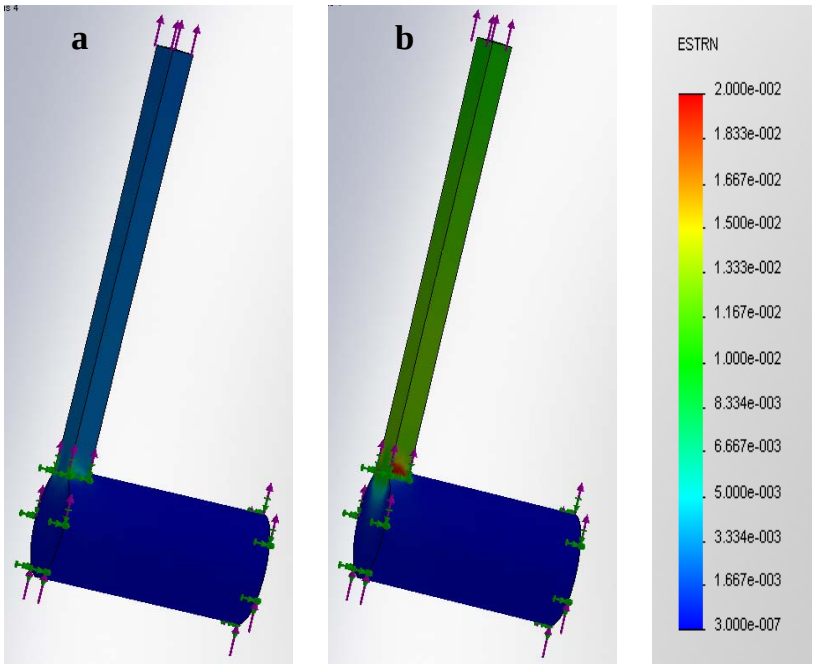

Figure 17: Comparison of Strain plots of Achilles Tendon (5300N Force Applied) between High Strength Collagen (a) and Low Strength Collagen (b)

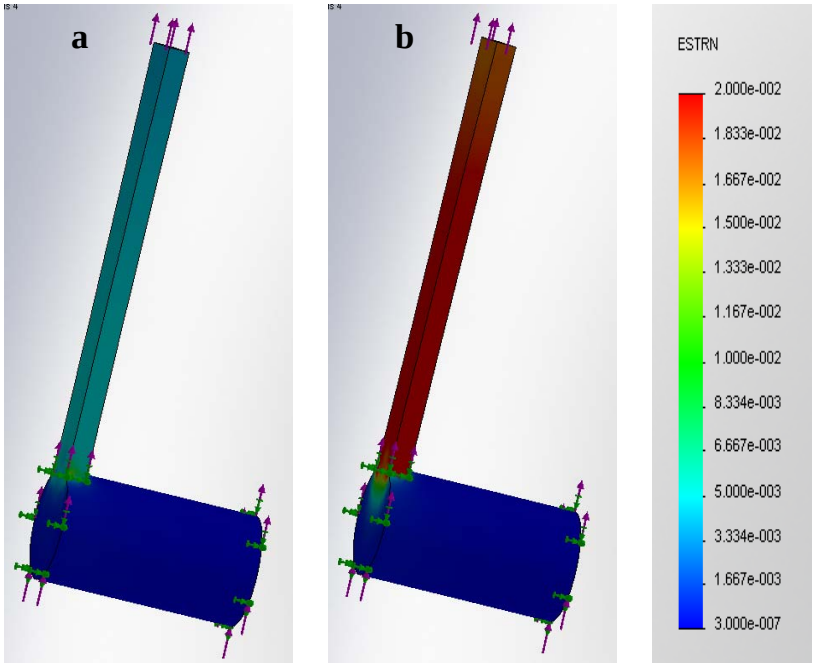

Figure 18: Comparison of Strain plots of Achilles Tendon (9000N Force Applied) between High Strength Collagen (a) and Low Strength Collagen (b)

Discussion

All in all, the finite element analysis of the Achilles tendon-calcaneus model does suggest that the mechanical properties of type 1 and type 3 collagen do play a major role

in the strength (or weakness) of the tendon. While the placement and magnitude of von Mises and principle stresses along the tendon were the same for each model, the factor of safety plots and the strain plots show the effect of the weaker material properties on the tendon complex. As the force applied to the tendon increases, as would occur in a progression from standing to sprinting, the tendon succumbs to the higher load and acquires the ability to rupture. Were the tendon to be constructed mainly of type 1 collagen, the model suggests that this load could be withstood and the person would have no problems continuing their run. As the tendon experiences slight mini tears however, the collagen matrix must be rebuilt and remodeled. As a result, type 3 collagen invades the area in order to build more foundation for type 1 collagen. During the rebuilding process, however, the tendon becomes weaker; and if that same person were to start running, their probability of rupture would increase dramatically. According to Figures 6, 10, 13c, 14c, 17c and 18c, this rupture would occur right above the tendon-calcaneal insert.

At the same time that the majority of the stress occurs along the length of the tendon, it is evident in Figures 6, 9, 10, 14, 17 and 18 that the calcaneus experiences some stress as well. The result of this stress placement could lead to an avulsion in which the tendon fully tears away from the calcaneal bone instead of snapping into two pieces. While more research is necessary to determine why avulsions occur and under which loading situations they occur, this model shows that under typical Achilles tendon loadings, a calcaneal aversion is most likely to occur at higher stresses (i.e. between 5300N and 9000N).

While this study suggests that the change in the strength of the collagen matrix can alter the overall strength of the tendon and lead to a rupture, the linearity of the model provides some drawbacks. The Achilles tendon *in vivo* is a nonlinear musculoskeletal component. As higher forces are applied, the tendon can stretch, thus dispersing the force applied and reducing the overall stress. Under these circumstances, the tendon should be able to withstand much higher stresses than other areas of the body, hence why it remains the strongest tendon in the body. The next step in the process of producing a reliable Achilles tendon model in orthopedic research would be to create a nonlinear model. In this model, it would be suggested that even higher forces than 9000N could be withstood by the tendon as the tendon could stretch like a spring and disperse the load. However, if type 1 and type 3 collagen matrices are tested on a nonlinear model, similar results to those found in this study should result. Type 3 collagen is simply weaker than type 1 collagen, and thus in any model, the material with the lowest mechanical properties will typical induce failure first.

Similarly, while this model is only linear, the tendon is also represented by a long rectangle. *In vivo*, it is understood that the tendon is not simply a rigid rectangle, but rather more circular or oval shaped, and the insertion into the calcaneal bone appears to be crescent shaped [14]. Therefore, at the same time that a nonlinear model would be ideal, certain alterations to the current model can be easily changed in order to make it slightly more complex. Within the future work section of this paper, the tendon was altered to a circular model with similar measurements as the rectangular model and the same forces, constraints, and contacts were applied. The results obtained in this model appear to more closely match the results found in literature and will be discussed later.

In conclusion, the linear model of the Achilles tendon represents a step in the right direction in terms of furthering the construction of a model that can be used in orthopedic research. Having a better understanding of just how the tendon works can only lead to more improved repair methods. Similarly, being able to manipulate a model according to proposed surgical or conservative repair methods can only lead to better overall healing, and may even help alleviate the current controversy between these two methods.

Future Work

As noted, a nonlinear model would be the ideal representation of the Achilles tendon in terms of conducting *in vitro* studies that would produce reliable and consistent measurements according to previous *in vivo* tests. Before such a model can be constructed, however, a few alterations can be made to the linear model to improve the results. In this section, a second model was constructed using a circular tendon implanted into the back of the same calcaneal piece. The diameter of the tendon is equivalent to the widths of the previous rectangular model. Similarly, the same forces, contact points, restraints and material properties were applied. The mesh constructed was slightly coarser due to large displacement errors. The following results represent the displacement plots, von Mises stress plots, principle stress plots, and strain plots. In each figure, the rectangular model is present so as to more easily compare the differences in results.

In Figure 19, the displacement plots for each model are compared. It is evident that the distribution of force is alike for both models, again due to the linearity of the model and the fact that stress is distributed evenly up the model.

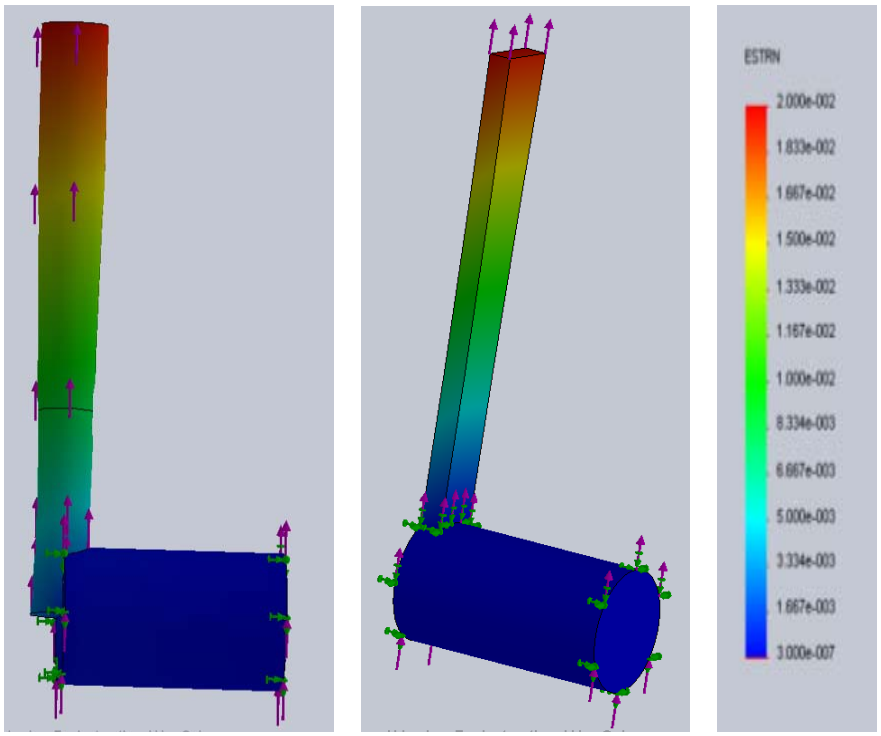

Figure 19: Displacement of Force through the circular and rectangular Achilles tendon models

Figures 20 through 23 depict the von Mises stress plots of both models. The stress plots were the same between the two different materials, and therefore only the pictures of the high collagen models are shown. What is interesting in the circular model is that the highest location of stress, although still located between 2 and 6cm from the base, is most apparent on the inner portion of the tendon. While Figures 20 and 21 show a great deal of similarity, the main differences arise at the higher forces (Figures 22 and 23). When a force of 5300N is applied to the circular model, the tendon shows much higher stresses both along the calcaneal edge and along the inner face of the tendon. Finally, at 9000N, the distribution of stress is quite high, roughly up half of the tendon, in the circular model, whereas the rectangular model still shows high stress only right around the calcaneus. It is clear that the circular model demonstrates the aspect of rupture

occurring anywhere between 2 and 6cm above the calcaneal better than the rectangular model did.

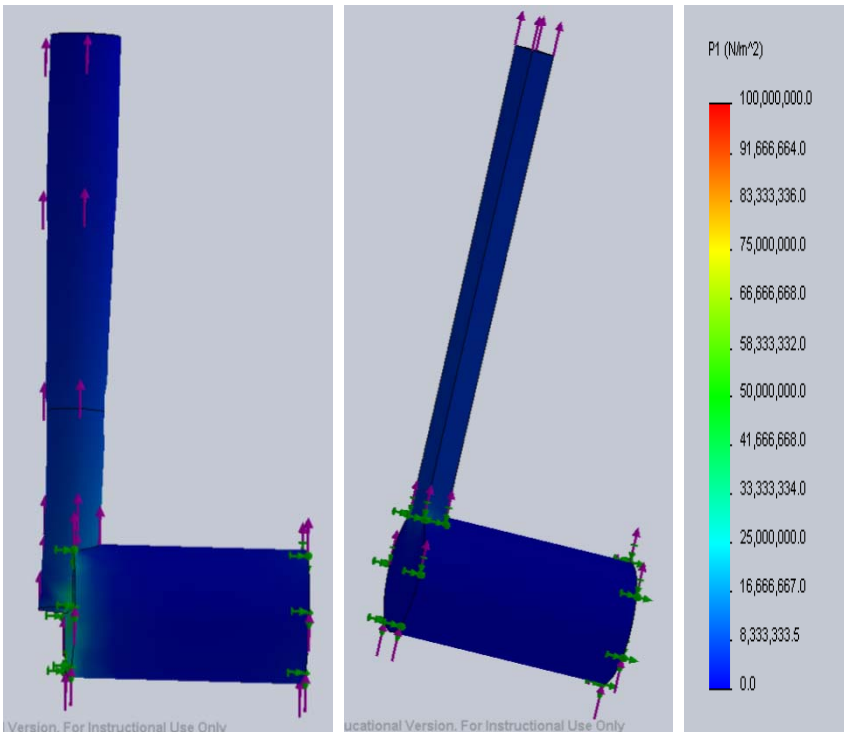

Figure 20: von Mises Stress Plot of circular and rectangular Achilles tendon models (700N Force Applied)

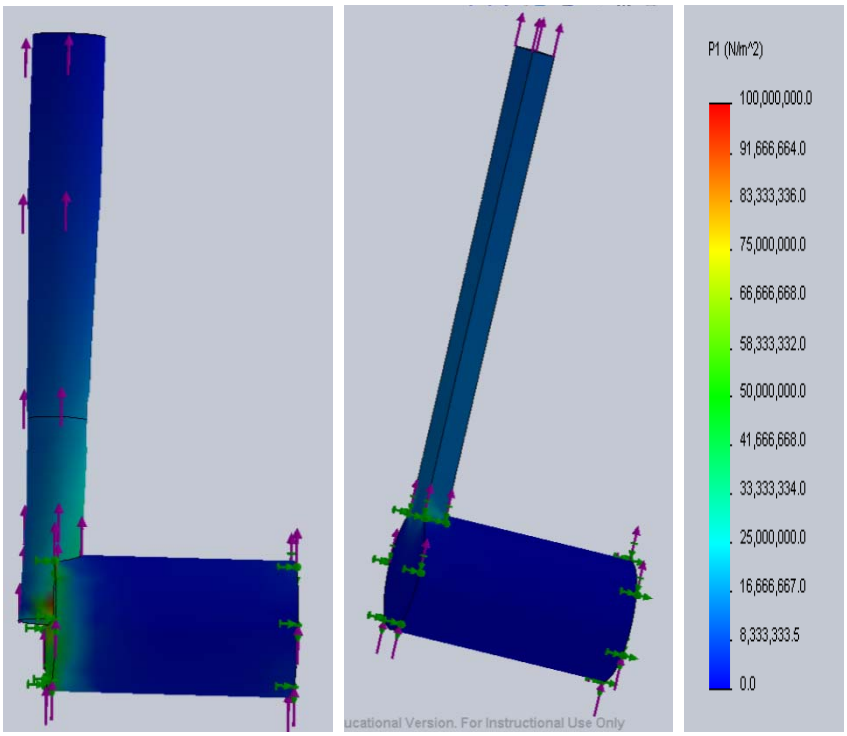

Figure 21: von Mises Stress Plot of circular and rectangular Achilles tendon models (1500N Force Applied)

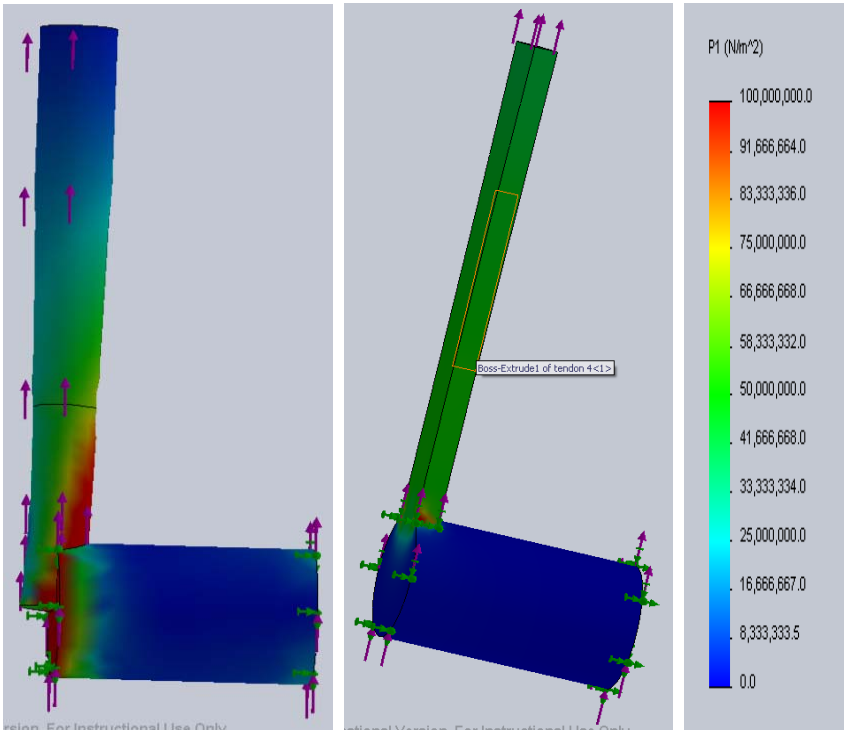

Figure 22: von Mises Stress Plot of circular and rectangular Achilles tendon models (5300N Force Applied)

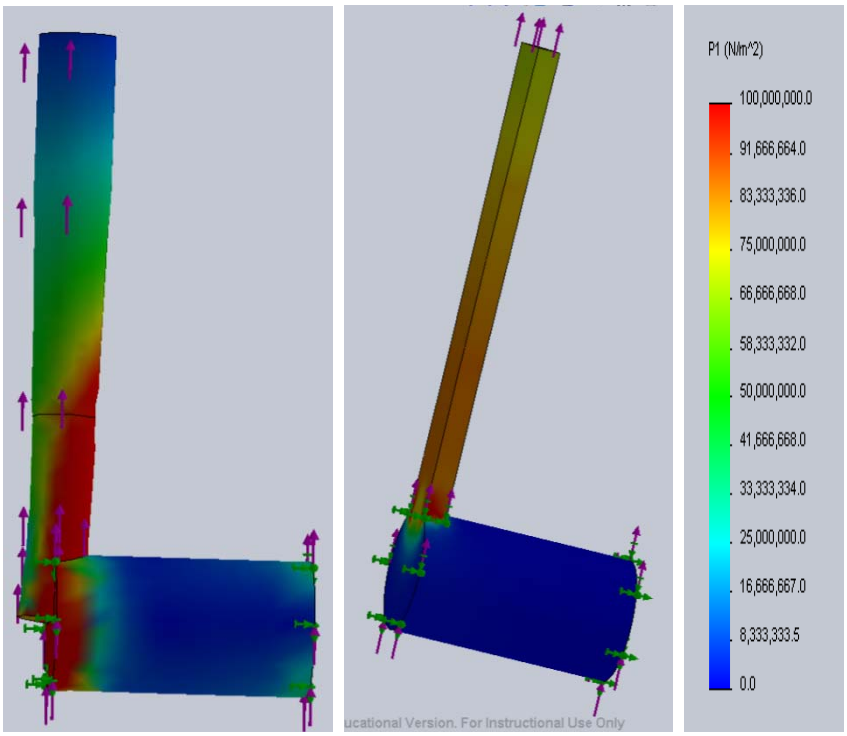

Figure 23: von Mises Stress Plot of circular and rectangular Achilles tendon models (9000N Force Applied)

Figures 24 through 27 illustrate the principle stress plots for both models. Again, the principle stress plots were equivalent for both the low and high collagen materials, and thus only the diagrams of the high collagen models are shown.While the rectangular model showed very little differences when comparing the von Mises stress plots and the principle stress plots, the circular model acts differently. Although the areas of highest stress remain on the inner face of the tendon, the principle stress plots show less stress applied to the calcaneal bone under higher loads. This distinction is most evident at 9000N. Besides this main distinction, the stresses remain highest under the higher loading conditions as would be expected according to the equation for stress ($\sigma=F/A$).

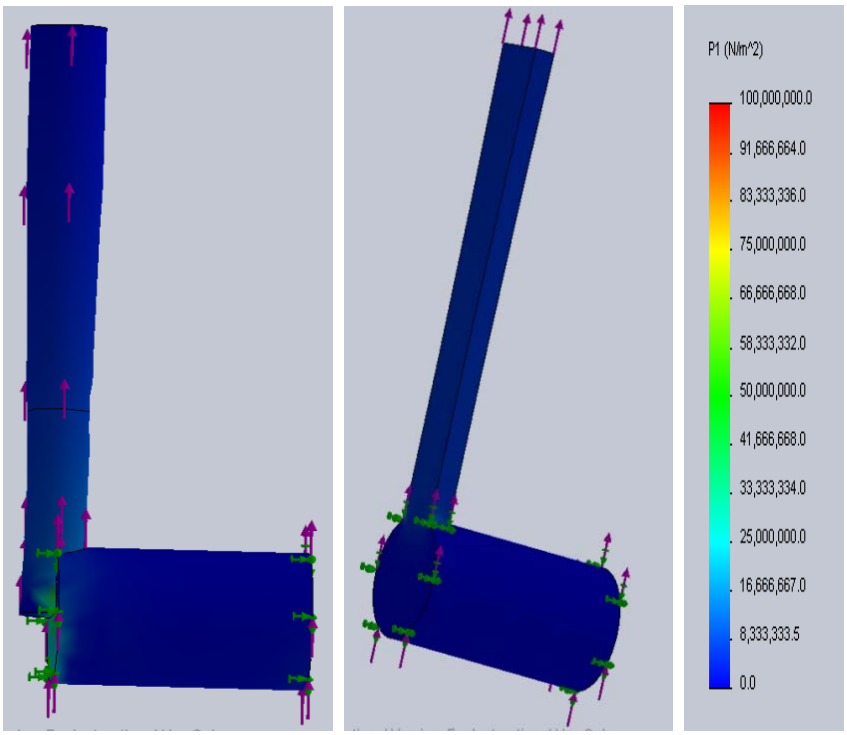

Figure 24: Principle Stress Plot of circular and rectangular Achilles tendon models (700N)

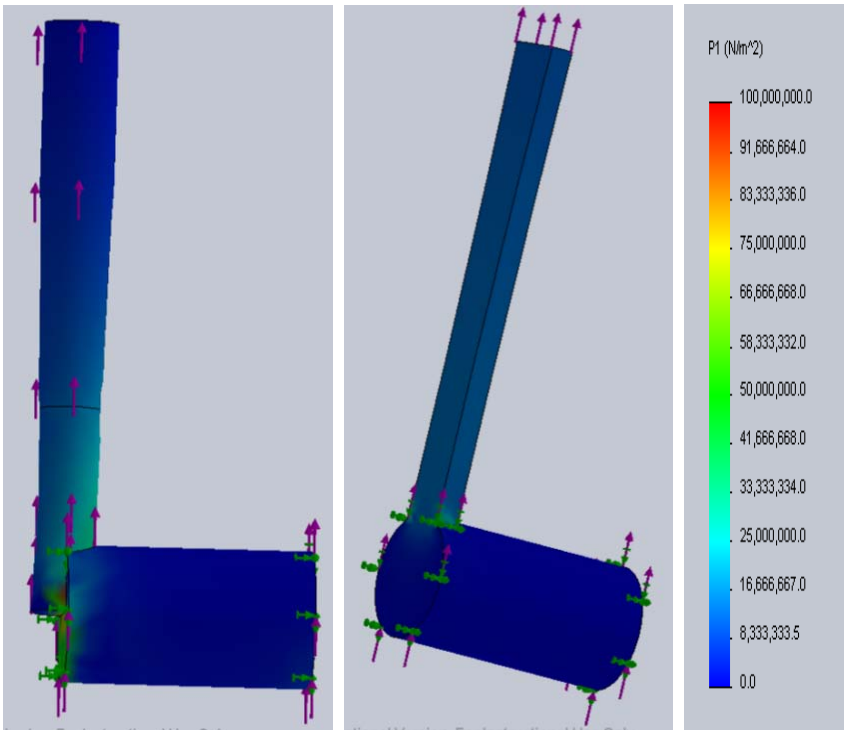

Figure 25: Principle Stress Plot of circular and rectangular Achilles tendon models (1500N)

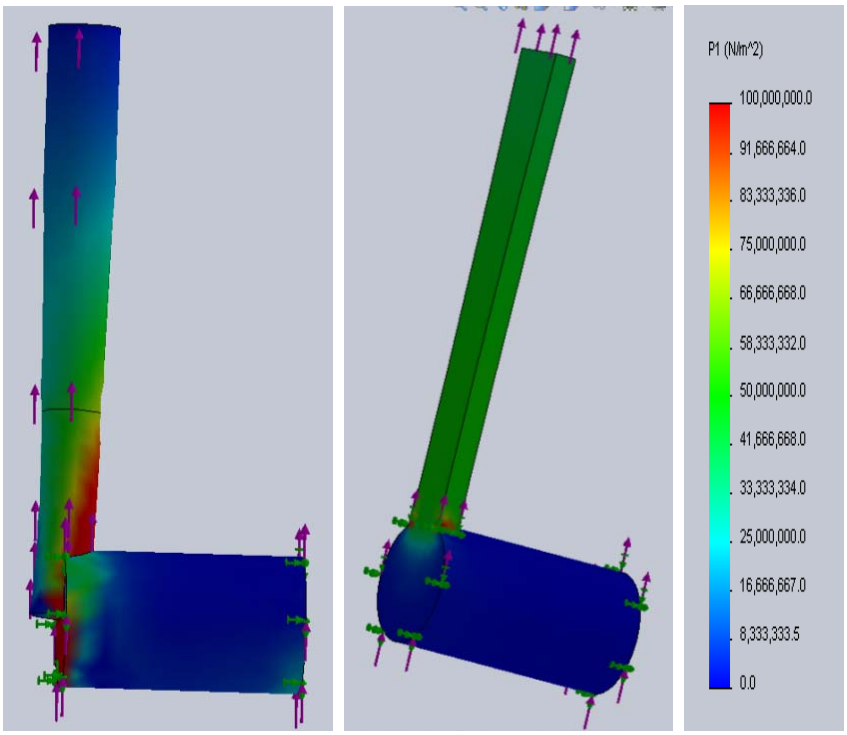

Figure 26: Principle Stress Plot of circular and rectangular Achilles tendon models (5300N)

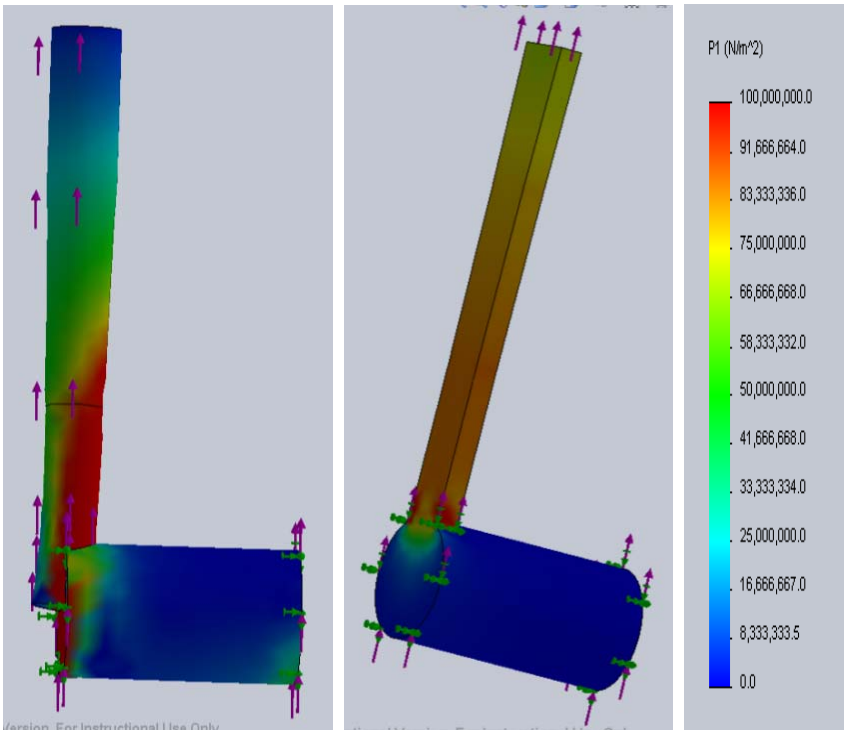

Figure 27: Principle Stress Plot of circular and rectangular Achilles tendon models (9000N)

Figures 28 through 31 display the strain diagrams of the rectangular and circular models. Due to the coarser mesh, the appearance of the strain in the circular models is slightly different. (The appearance of the distribution of strain does not alter the overall results, however.) Again, it is apparent that the low-strength collagen models experience higher stresses and strains than the high-strength collagen models. For the first two loading conditions, the strain plots for the circular model are very similar to the rectangular model. As the force increases, however, the distribution of strain around the tendon changes between the two models.

At 5300N (Figure 30), it is evident that the presence of strain is focused largely at the base of the tendon and travels roughly halfway up the tendon. The rectangular models, on the other hand, show the strain distributing entirely throughout the tendon piece. Subsequently, the circular model shows a failure point on the inner face of the tendon at 5300N, whereas the rectangular model simply shows moderate strain. As we reach 9000N, similar results occur. Again, the circular model depicts the strain largely in the base of the tendon, surrounding the calcaneus. The top of the tendon remains strain-free for both materials in the circular model, whereas the rectangular models show high stress throughout the entire length. Ultimately, it is clear that the circular model provides a better depiction of the strain along the tendon as it illustrates the exact regions where the strain is present, as opposed to simply subjecting the entire tendon to high strain, which is unreasonable and does not occur *in vivo*.

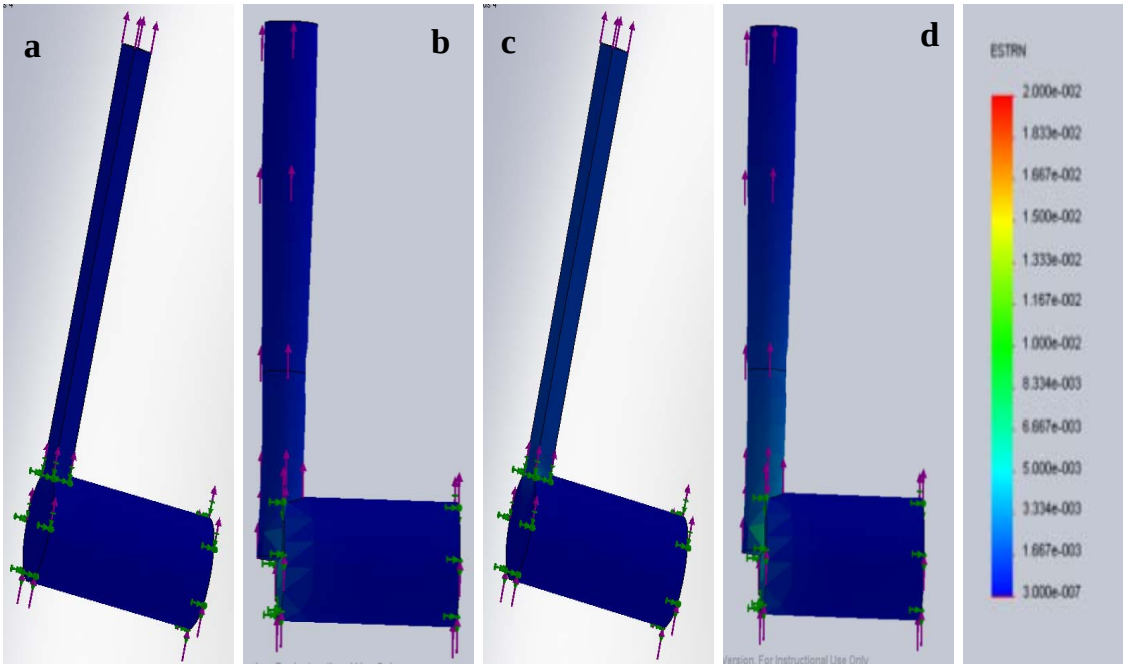

Figure 28: Comparison of Strain plots of rectangular and circular Achilles tendon models (700N Force Applied) between High Strength Collagen (a, b) and Low Strength Collagen (c, d)

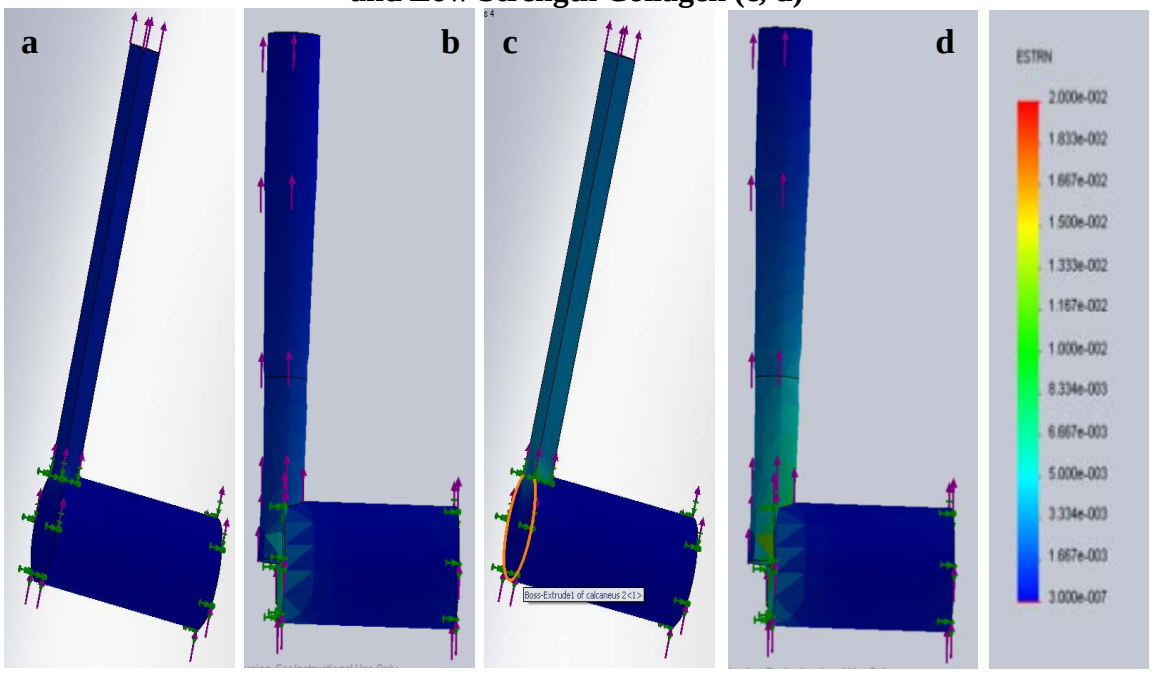

Figure 29: Comparison of Strain plots of rectangular and circular Achilles tendon models (700N Force Applied) between High Strength Collagen (a, b) and Low Strength Collagen (c, d)

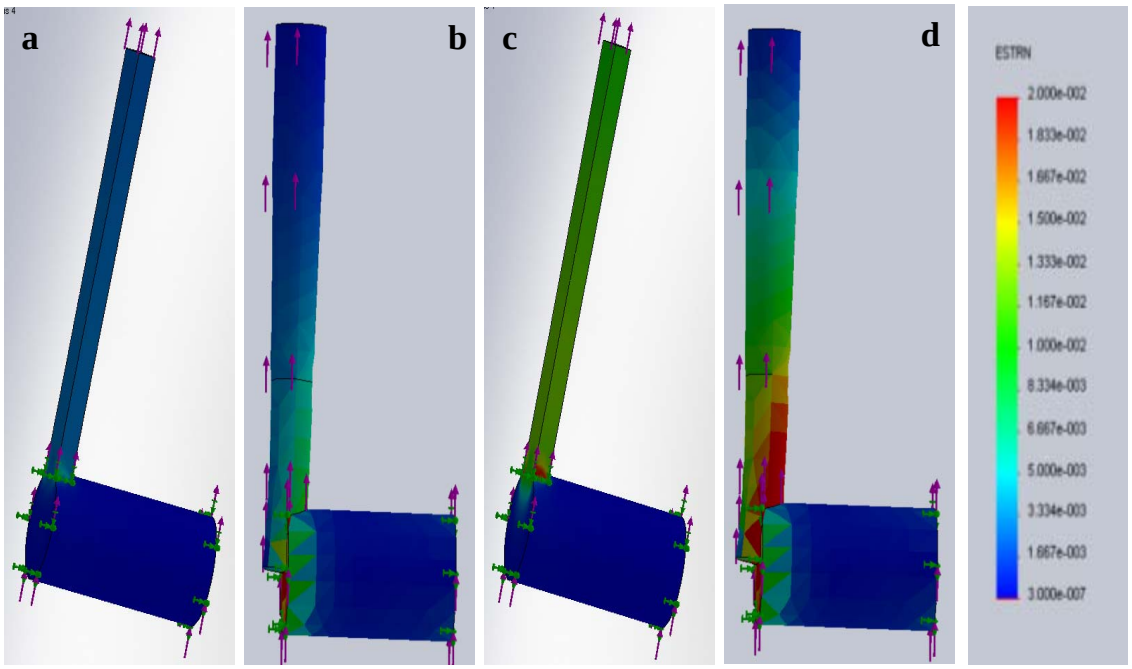

Figure 30: Comparison of Strain plots of rectangular and circular Achilles tendon models (700N Force Applied) between High Strength Collagen (a, b) and Low Strength Collagen (c, d)

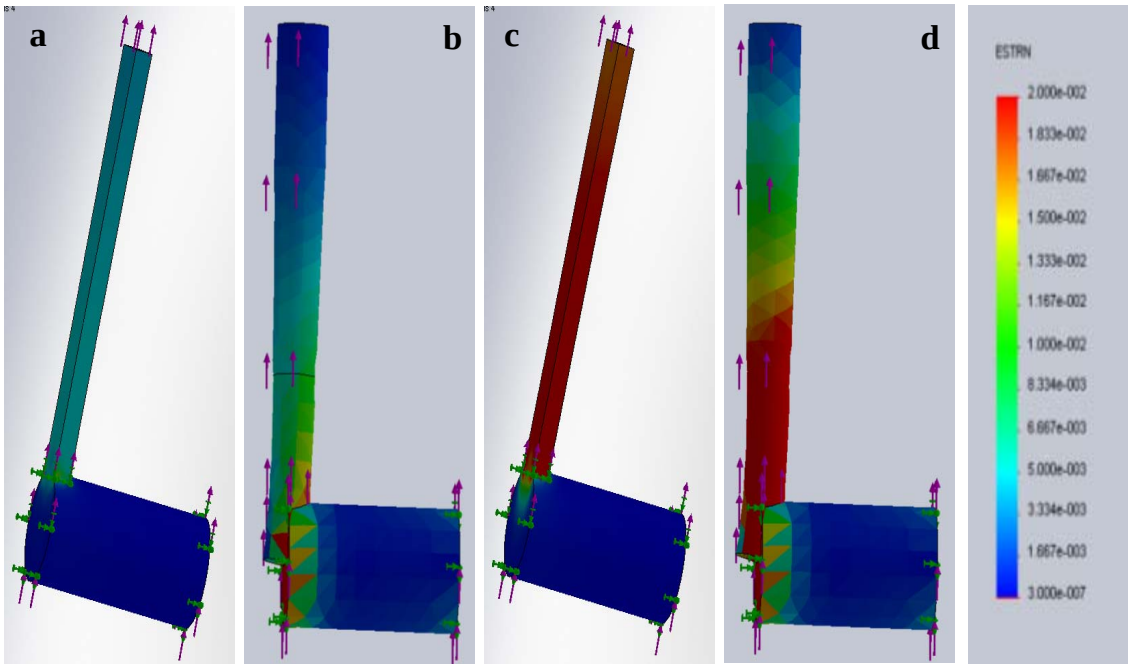

Figure 31: Comparison of Strain plots of rectangular and circular Achilles tendon models (700N Force Applied) between High Strength Collagen (a, b) and Low Strength Collagen (c, d)

All in all, the circular model appears to be yet another step in the right direction in terms of depicting the actual response of an Achilles tendon under the different loading conditions and different collagen matrices. The circular model sheds more light on the fact that most ruptures occur between 2 and 6cm above the calcaneus. While the rectangular models showed high stress and high strain just above the calcaneal insertion, the circular model shows high stresses and strains both right at the calcaneal insertion and along the bottom half of the tendon. Further research must be conducted regarding the insertion of the tendon into the calcaneus so that this can be properly depicted within the model. All in all, both further research regarding the insertion point and avulsions, and the construction of a nonlinear model are necessary before this computer aided model of the Achilles tendon can physically be used in *in vitro* studies.

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