

**International Symposium
on
Ligaments and Tendons
(ISL&T)**

***SAVIO WOO YOUNG RESEARCHER
AWARDS***

SAVIO WOO YOUNG RESEARCHER AWARDS

Be it resolved that as Professor Savio L-Y. Woo initiated the International Symposium on Ligaments and Tendons (ISL&T) to promote awareness of the field, the exchange of information, and collaboration nationally and internationally, and as Professor Woo has been an outstanding scientist in this area as well as an internationally recognized intellectual ambassador for training, mentoring and recognition of aspiring students in the fields of biomedical engineering and orthopaedic research on all levels, his lifelong contribution and accomplishments well deserve special recognition.

Therefore, the Woo Awards Committee is honored to present the first Professor Savio Woo Young Researcher Awards to the individuals who have performed the best research studies in three major areas, biomechanical, biological and clinical, to provide financial support to attend the ISL&T to present their work.

Dr. Albert Banes
Chair, Woo Awards Committee
President, Flexcell International Corp.



Savio Woo Young Researcher Awards Winners International Symposium on Ligaments and Tendons – X Hong Kong, SAR, China



Ms. Saira Chaudhry, Dr. Woo and Mr. Xiao Chen



International Symposium for Ligaments and Tendons (ISL&T)

Savio L-Y. Woo Young Researcher Awards

US\$ 1,000 cash prize and Certificate for each Award

[http://www.pitt.edu/%7Emsrc/islt10/Savio Woo Award Announcement.doc](http://www.pitt.edu/%7Emsrc/islt10/Savio%20Woo%20Award%20Announcement.doc)

Purpose: Professor Savio L-Y. Woo founded the International Symposium on Ligaments and Tendons (ISL&T) to promote awareness of the field, the exchange of information and collaboration nationally and internationally. The ISL&T has been a venue for lively discussion of current topics in connective tissue research and clinical applications. In addition to his leadership and significant scientific contributions to our field, Professor Woo has been an internationally recognized intellectual ambassador for training, mentoring and for aspiring students in the field of biomedical engineering and orthopaedic surgery. We are honored to present the Savio L-Y. Woo Young Researcher Awards to individuals who perform the best research studies in three major areas, biomechanical, biological and clinical and have submitted their work to the ISL&T-X meeting.

The Awards are intended to provide partial support (up to US\$ 1,000) towards the winners' research or for travel expenses to attend the ISL&T-X meeting. Up to **four awards** will be given this year.

Eligibility: Open to all graduate students and postdoctoral fellows. Applicant must be the first author of the abstract and be present at the ISL&T-X meeting to accept the award. Advisor's verification of eligibility is required.

Application: Upon submission of the abstract by the regular submission deadline, applicant must indicate his/her intention to be considered for the award. Those selected will be invited to submit extended abstracts.

Award Categories: Awards will be given in three main categories, namely:

- Biomechanical: Experimental studies involving biomechanics of ligaments and tendons, new methods for measurement of biomechanical properties, or computational analyses
- Biological: Basic science studies to characterize the cellular behavior of ligaments and tendons, as well as the extracellular matrix
- Clinical: Studies which compare existing surgical procedures or propose novel alternatives

Selection Criteria: Applicant's abstract submitted for the ISL&T-X will be reviewed by the program committee through the regular evaluation process based on scientific merit and quality of the work.

Selection Process:

1. A number of highly meritorious abstracts that have a high impact with a clear motivation and relevance as well as quality experimental methods and scientific reasoning in the field of ligament and tendon research will be invited by the Woo Awards Committee to submit a 3-page extended abstract on **November 30, 2009**. The deadline on receiving the extended abstracts will be on **December 15, 2009**.
2. The Woo Awards Committee will conduct a thorough review and select the winners. The winners will be notified on **January 1, 2010**.

Acknowledgements: Sponsored by Flexcell International Corp. and the Asian•American Institute for Research and Education (**ASIAM**).

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SAVIO WOO YOUNG RESEARCHER AWARDS WINNER

Clinical Research

Ms. Saira Chaudhry



Dr. Woo, Ms. Saira Chaudhry, and Dr. K.M. Chan

SAVIO WOO YOUNG RESEARCHER AWARDS WINNER

Clinical Research

ECCENTRIC & CONCENTRIC CALF MUSCLE LOADING: AN IN VIVO STUDY OF FORCE & EMG

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INTRODUCTION

Achilles tendinopathy is a painful condition occurring in and around the Achilles tendon (AT), thought to be a failed healing response. It is often characterized by disruption of collagen fibres, an increase in non-collagenous matrix and random proliferation of tenocytes as shown by tendon thickening, disordered tendon fibrils and neo-vascularisation on ultrasound imaging. Excessive repetitive overloading of the Achilles is one of the main stimuli leading to tendinopathy [1], hence it is common in athletes with 11-24% of runners suffering with this disease [2]. However, it also occurs in non-athletes, with around 33% of tendinopathic presentations being in sedentary individuals [3].

Achilles tendinopathy is prone to recurrence, as current treatments are only partially successful. The main conservative treatment is heavy load eccentric training (ET) of triceps surae, shown to be effective in various controlled clinical trials and systemic reviews, with a success rate of 60-82% [4, 5]. The ET protocol was proposed by Stanish et al back in 1986 [6], and the first controlled trial of this method carried out by Alfredson in 1998 [4] confirmed the superior outcomes of EC training over concentric training (CT). Since this time, a number of studies have evaluated the efficacy of ET providing a sufficient level of evidence to support this treatment method [4, 5, 7, 8].

Eccentric training involves the lengthening of the muscle-tendon unit during the application of load. This is the opposite to concentric loading where the muscle-tendon unit gets shorter and differs from isometric exercises, in which the muscle tendon unit length remains unchanged. ET has been shown to result in positive clinical outcomes for Achilles and other tendinopathies, improving pain, patient satisfaction and function when compared with concentric exercise. It has also been shown to increase the total strength and mass of the muscle as compared to CT [9]. However, it is unclear why ET results in better outcomes in Achilles Tendinopathy than CT and the mechanism behind the superior muscle strength and mass gain during EC loading remains unclear. Based on the specificity principle of strength training, it has been postulated that eccentric and concentric actions provide different stimuli to the triceps surae, and therefore produce different muscle and tendon adaptations [10]. It is likely that the whole triceps surae unit will be involved in mechanical remodelling and not just the Achilles tendon, hence the aim of our study was to compare eccentric and concentric loading modalities to investigate differences in muscle activity and tendon force (TF) across the muscles of the triceps surae. Thus, this study hypothesized that CT and ET of the triceps surae would lead to different force production and muscle activation patterns.

METHODS

Twelve healthy volunteers (6 male and 6 female, mean age = 27.8 ± 1.9 years (SD)) performed eccentric and concentric loading exercises for the triceps surae. For ET, subjects were asked to stand on the toes of one foot with the heel raised, before lowering the heel in a controlled manner. The exercise was performed off the edge of a step to allow full dorsi-flexion to be reached (Fig.1A). For CT, subjects started with the heel below the toes and raised the heel during the exercise in the same controlled manner (Fig.1B). In order to keep speed consistent, subjects were taught to complete the heel raise or heel drop during a count of four beats on a metronome. After completing either exercise, the subjects used the other leg to assist in returning to the starting position, before repeating the exercise. A single data collection consisted of 2 cycles of either CT or ET performed consecutively. Three sets of such data were recorded for each loading paradigm for each subject, in a randomized order. Tendon force and extension were measured during each exercise.

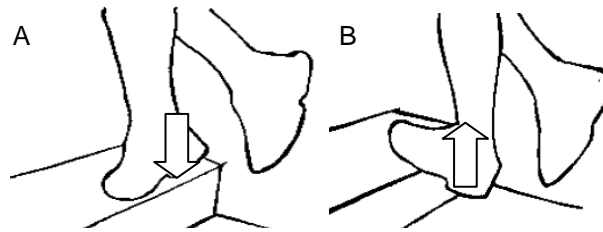


Fig.1 A) Eccentric loading B) Concentric loading

An active infra red motion analysis system (CODA, CX1, Charnwood Dynamics, Rothley, UK) was used to determine gross movements of the leg, with simultaneous ultrasound tracking of the medial gastrocnemius muscle-tendon junction (MTJ). The ultrasound probe was placed on the leg such that the medial gastrocnemius muscle-tendon junction (MTJ) could be imaged (Voluson e, GE Healthcare, UK), and three motion tracking markers placed on the probe, so its location could be embedded into the laboratory coordinate system in order to spatially synchronise the data. In addition, electromyography (EMG) recordings were used to determine muscle activity using dual electrodes with a 20mm inter-electrode distance placed on the belly of the soleus, lateral gastrocnemius, medial gastrocnemius and tibialis anterior muscles following the EU guidelines [11]. EMG data was then rectified and smoothed using MATLAB in order to compare the activation patterns during ET and CT.

RESULTS

While some inter-subject variability was apparent, CT and ET resulted in distinctly different loading patterns and muscle activation patterns across all subjects. ET resulted in greater values for both the rate of change of tendon force, and the maximal force ($1604 \pm 89.5\text{N}$) compared with CT ($1410 \pm 79.7\text{N}$) $p < 0.01$. In contrast, when looking at EMG data both the rate of change of muscle activation and the maximal activation values were significantly higher ($p < 0.01$) in CT than ET. Pairing eccentric and concentric EMG data for each subject highlighted that, whilst the mean CT muscle activity levels were higher, the ratio of mean concentric to mean eccentric activity for each subject varied between muscles, with values of 1.08-2.00 for soleus, 1.25-3.03 for lateral gastrocnemius, 1.08-1.48 for medial gastrocnemius and 0.09-1.83 for tibialis anterior. Figure 2 shows the variation of the muscles activity and force with respect to heel height (HHt). As the heel position varies between its highest and lowest positions, there is high rate of change of force during ET while high rate of change of EMG activity across four muscles during CT.

Figure 3 shows the variation of force with tendon length during ET for one subject. Mean data for all subjects will be available before the February meeting. The significant drop in force is explained by the bilateral weight bearing during the return phase of ET; however, a typical hysteresis curve is demonstrated.

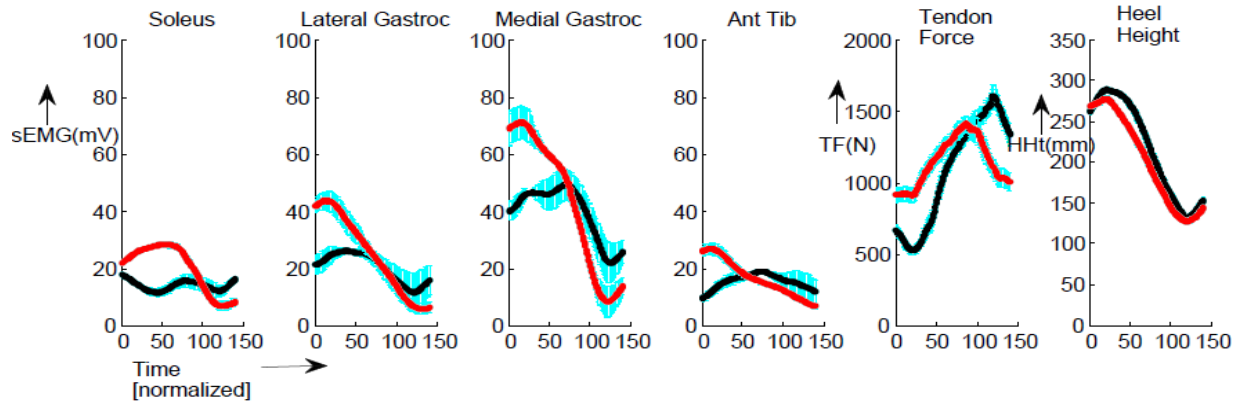


Fig.2 Graphs of mean (SEM in blue) soleus / lateral gastrocnemius / medial gastrocnemius / anterior tibialis EMG and tendon force are shown against normalised time. In addition heel height is shown in order that the previous graphs can be interpreted with respect to position. In all graphs the eccentric action is in black, and the concentric in red.

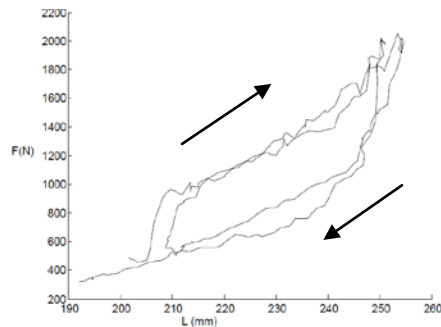


Fig.3 Achilles tendon length and force measurements against time for two consecutive ET cycles in one subject. Arrows indicate the general trend for rise and fall of the force against length.

DISCUSSION

This study demonstrates that eccentric calf muscle loading produces higher forces within the tendon than concentric loading with lower muscle activation levels. Results from our study show that at the start of concentric movement the calf muscles are activated, the AT force increases and the subject accelerates upwards. The peak AT force occurs in the middle of the movement, possibly as the moment arm of the triceps surae is furthest from the ankle joint axis for sagittal plane movement during this phase. In the eccentric movement, the subject starts to drop ‘under control’. The movement is controlled (resisted) by lengthening of the activated calf muscle and by rate-dependent stretching of the AT (figure 3). The maximal derived AT force occurs at the end of the eccentric movement when maximum force is required to decelerate the subject against gravity. It was observed that there was

no significant differences in the forces midway between the two loading cycles which is consistent with the previous findings reported by Rees et al. [12] where they compared similar ET and CT loading approaches. Our study has a greater subject number and additionally shows significantly greater forces occurring at the start and end of loading. If the mechanism of action of ET is via higher force fluctuation, then these differences may have therapeutic relevance [6].

The higher progressive force on tendon during eccentric loading may partially explain the difference in therapeutic effect, as tendon cells respond to mechanical forces by adapting their production of neo-matrix. These higher tendon forces may lead to improved matrix anabolism, thus improving the structural and mechanical properties of the tendon. The differences in the peak load and the rate of change of tendon force are consistent with previous findings in other muscle groups. For example, Westing and Serge found significantly higher mean eccentric compared to concentric torques in quadriceps and hamstring muscles [13].

One of the aims of this study was to observe calf muscle activity during the two loading protocols. The relatively low EMG activity during eccentric action was expected. The combination of the EMG and force data indicates that eccentric contractions required lower levels of voluntary activation by the nervous system to achieve a given muscle force, applied to the tendon. The reduced EMG observed during eccentric loading suggests an incomplete activation of the motoneurons that innervate the muscle, which may take the form of a lower level of activation distributed across the entire population of motoneurons or be the activation of only a subset of the entire population. It may be that the associated energy preservation during ET, via ATP sparing, allows a greater volume of exercise to be carried out under eccentric conditions than concentric.

In conclusion, significant differences in EMG and tendon force were demonstrated between eccentric and concentric loading protocols, which may underlie some of the differences in therapeutic effect. Future, imminent, work will clarify group stress – strain relationships.

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SAVIO WOO YOUNG RESEARCHER AWARDS WINNER

Biological Research

Mr. Xiao Chen



Dr. Woo, Mr. Xiao Chen and Dr. K.M. Chan

SAVIO WOO YOUNG RESEARCHER AWARDS WINNER

Biological Research

TENDON-LINEAGE DIFFERENTIATION OF HUMAN EMBRYONIC STEM CELLS BY OVEREXPRESSION OF SCLERAXIS AND DYNAMIC MECHANICAL STRESS

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INTRODUCTION

Tendons are frequently damaged during sports and other strenuous activities [1, 2] and have quite poor self-repair capacity. Their healing occurs via the formation of a fibrotic scar and results in significant dysfunction and disability.

Currently, stem cells and tissue engineering techniques show great potential for tendon regeneration. Adult tendons lack regeneration capability; however, fetal tendons have great regeneration potential. More interestingly, this capability is intrinsic to the fetal tendon itself [6]. Thus, fetal cells seem to play a crucial role in fetal tendon regeneration. Embryonic stem cells (ESCs), which are primary embryonic cells and hold tremendous potential for cell-based therapies, may have the potential for tendon regeneration. As shown in our previous work, ESCs can be used for tendon regeneration by stepwise induction. However, the maturation of tendon-like tissues and regeneration of repaired tendon are yet to be achieved.

No optimal method to induce ESC into tenocyte differentiation for tendon regeneration has been found yet. The signals that are specific to tendon development may be used to induce tendon-lineage differentiation. In embryonic development, TGF and FGF signals, ectoderm signals and mechanical stress [3, 4] are critical for tendon development and associated with tenocyte recruitment and differentiation. Scleraxis, a bHLH transcription factor, is a highly specific marker for tendons, and scleraxis knockout causes severe defects in force-transmitting tendon [5]. This suggests that mechanical stress and scleraxis may play a synergic role in the tendon development. However, ectopic expression of scleraxis is not enough to induce ectopic tendon formation. Dynamic mechanical stress could also induce tendon-lineage differentiation of bMSC. The signaling mechanisms that mediate force-induced tenocyte differentiation and collagen expression are currently not defined. In this study, we tested the hypothesis that mechanical stress interacts with the transfer growth factor-beta (TGF-beta) pathway and scleraxis transcription factor to stimulate tendon-lineage differentiation and tendon regeneration.

METHODS

Cell Culture Human ESCs were first induced to differentiate into mesenchymal stem cells (MSCs) as previously MSCs (hESC-MSCs) was analyzed by flow cytometry. described [6]. The immuno-phenotype of hESC-derived

Scleraxis Transfection and Mechanical Stress Scleraxis-hESC-MSCs were obtained by transfection with scleraxis-lentivirus followed by selection with blasticin (2 µg/ml). LacZ or GFP genes were used for control. To fabricate engineered tendons, the cells were then embedded in collagen gel at 10⁷ cells/ml, seeded on knitted silk scaffolds, and cultured for 2days. The engineered tendon then was subjected to a dynamic mechanical stress of 1HZ for 2h/day to evaluate the combination effect of scleraxis and dynamic mechanical stress.

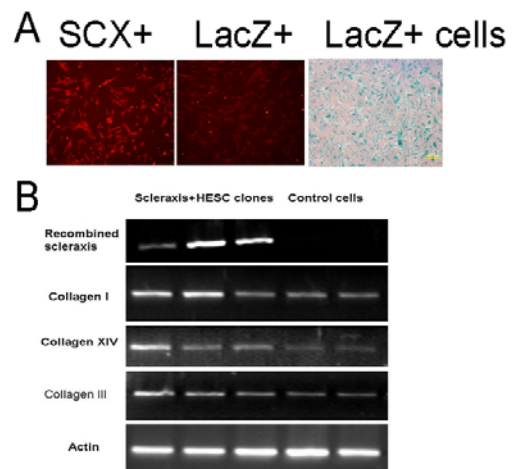


Fig.1 Scleraxis overexpression induce tendon ECM expression. A. Immunofluorescence of scleraxis and virus transfection efficiency. B. scleraxis positive clones showed high scleraxis expression and higher expression of collagen I, III

Fabrication of scaffold-free Engineered Tendon hESC-MSCs formed cell sheets after 14 days of culture, and engineered tendons were formed in vitro [6]. The engineered tendons were subjected to a dynamic mechanical stress of 1HZ at 10% strain for 2h/day for 7 days. Then the regeneration potential of the engineered tendon tissues was evaluated in an in-situ rat patellar tendon window repair model.

Cell labeling To identify genetically engineered hESC-MSCs within the site of implantation in vivo, the cells were infected either with DII [6].

RESULTS

hESC differentiation into MSCs hESC-MSCs had the potential to differentiate into the three mesenchymal lineages, including osteogenesis, adipogenesis and chondrogenesis, and were positive for MSC surface markers (data not shown).

Scleraxis initiate mesodermal differentiation After selection with blasticidin, 100 percent of cells were transfected by the recombinant genes. Scleraxis expression in the transfected cells was significantly higher than the control (Fig 1A). Scleraxis overexpression increased the collagen I, III, XIV expression in clones, reduced collagen II promoter activation (Fig 2A) and bmp induced smad activation(Fig 2B). However, ALP activity and alizarin red staining showed scleraxis also increased the bone induction.

Scleraxis and mechanical stress synergistically induce tendon differentiation The expression of tendon-specific genes, such as collagen I $\alpha 1$, collagen I $\alpha 2$, collagen XIV was higher and osteo-/chondro-specific genes, such as osteocalcin, PTC, aggrecan were lower in scleraxis-hESC-MSCs.

However, osteo-specific gene runx2 was higher and several tendon-specific genes, such as tenomodulin, nafatc4 were lower in scleraxis-hESC-MSCs. In the

in-vitro tissue engineering tendon model, 4 hour cyclic mechanical stress significantly reduced runx2 and increased tenomodulin and nafatc4 expression in scleraxis-hESC-MSCs, while mechanical stimulus had no such effect in control group. (Fig.3). The findings implicated that scleraxis initiate both teno- and osteo-lineage early differentiation, while mechanical stress synergistically promote the teno-differentiation effect of scleraxis and inhibits its osteo-differentiation effect.

Scleraxis and mechanical stress promote tendon differentiation and regeneration in vivo In the in-vivo ectopic implantation models, mechanical stress induce larger collagen fibers and scleraxis showed synergetic effect (Fig.4). In the in-situ tendon repair model, the engineered tendon treated with scleraxis-hESC-MSCs and mechanical stress had significantly better mechanical properties than those of the control group (Fig.5). Furthermore, hESC-MSCs remained viable at the tendon wound site for at least 4 week. Moreover, no teratoma was found in any samples.

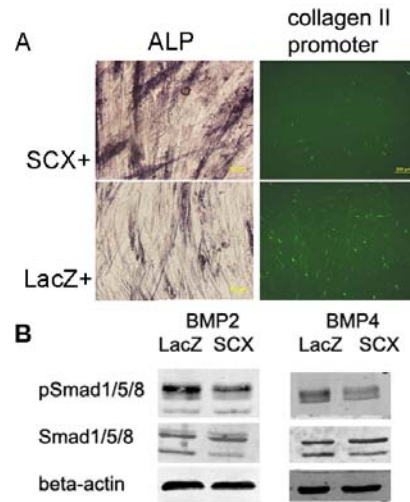


Fig.2 Scleraxis affect osteogenesis and chondrogenesis. A scleraxis increase ALP activity and reduce collagen II promoter activation. B scleraxis reduce bmp2 and bmp4 induced smad activation

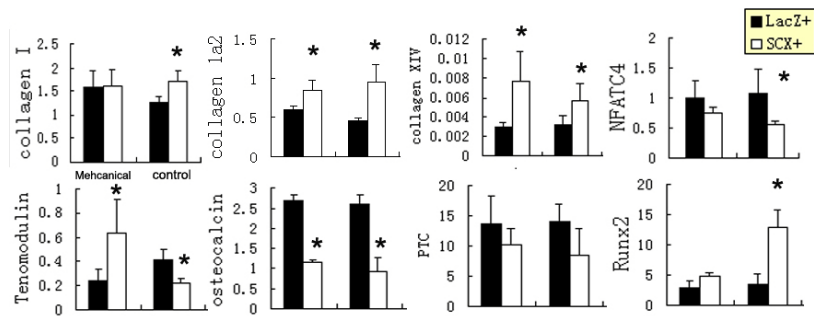
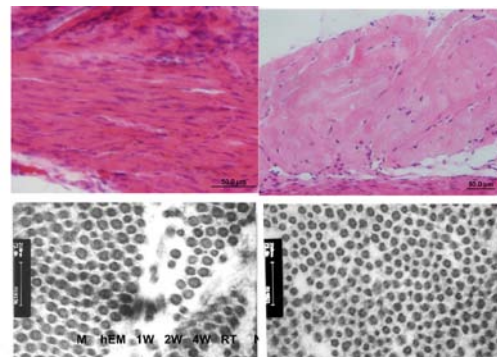


Fig.3 scleraxis and mechanical stress effect tendon-/osteo-specific gene expression *P<0.05 compared to LacZ+ cells



SCX+ mechanical LacZ +mechanical
Fig.4 scleraxis and mechanical stress increase maturation of tendon-like tissues in vivo

DISCUSSION

The present study demonstrates that mechanical stress and scleraxis have a synergetic function on tendon differentiation of hESC-MSC as well as tendon regeneration. Previous research indicated that scleraxis is also upstream genes of bone and cartilage specific genes, such as collagen I, collagen II and aggrecan, and it is involved in bone and cartilage differentiation and development [7-10]. Scleraxis alone is not enough to induce tendon lineage differentiation both in-vitro and in-vivo [7, 11]. As shown in the present study, which is consistent with previous work, scleraxis may not only be involved in tendon differentiation, but also in bone and cartilage differentiation. Scleraxis increases tendon specific ECM, such as collagen I $\alpha 1$, I $\alpha 2$ and collagen XIV. It also increases runx2 expression in hESC-MSC engineered tendon. The role of scleraxis on tendon differentiation is partially to change the activation of BMP-smad pathway.

Dynamic mechanical stress could also induce tendon-lineage differentiation of bMSCs. However, the mechanism involved and the effect on maturation of collagen fibers are not mentioned [12, 13]. This study showed that mechanical stress have synergetic function on inducing teno-lineage differentiation by reducing runx2 and increasing tenomodulin and nafatc4 expression in scleraxis-hESC-MSC engineered tendon. These results were confirmed by the in-vivo study that mechanical stress with scleraxis induces larger collagen fibers. Patellar tendons treated with the mechanical-scleraxis-hESC-MSCs engineered tendon had much better mechanical properties than did controls.

These findings may have considerable importance in understanding the roles of mechanical stress and scleraxis on tendon differentiation as well as developing therapeutics for tendon regeneration.

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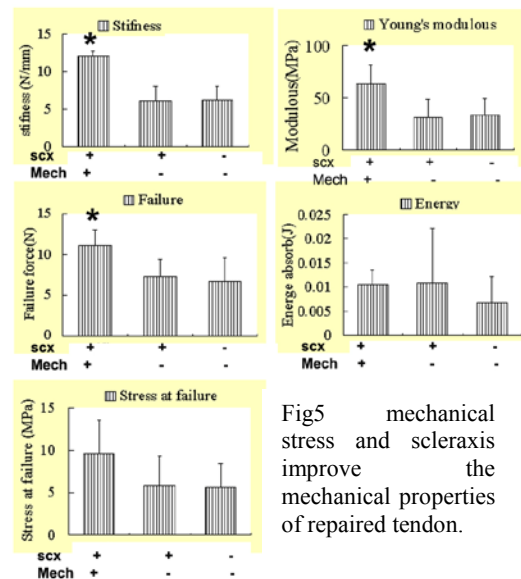


Fig5 mechanical stress and scleraxis improve the mechanical properties of repaired tendon.

Savio L-Y. Woo, Ph.D., D.Sc. (Hon), D. Eng. (Hon)

Dr. Savio L-Y. Woo is a Distinguished University Professor of Bioengineering and the Founder and Director of the Musculoskeletal Research Center (MSRC), a diverse multidisciplinary research and educational center in the Department of Bioengineering, Swanson School of Engineering at the University of Pittsburgh. He arrived at the University of Pittsburgh in 1990 after spending 20 years at the University of California, San Diego (UCSD) as a Professor of Surgery and Bioengineering.



Dr. Woo received his B.S. degree from Chico State College (1965), and M.S. and Ph.D. degrees (1966, 1971) from University of Washington. In 1999, Dr. Woo was bestowed a Doctor of Science Degree (Hon.) from the Trustees of the California State University System and in 2008, he earned a Doctor of Engineering Degree (Hon.) from The Hong Kong Polytechnic University.

Dr. Woo is a pioneer in bioengineering and is renowned for his 40 years of translational research in healing and repair of tissues. Together with his team, they have authored **304** original research papers in refereed journals as well as **137** book chapters and review articles. Their work has significantly impacted the management of ligament and tendon injuries including clinical paradigm shifts that have led to improved patient outcome.

More recently, Dr. Woo has focused on using novel functional tissue engineering to heal and to regenerate ligament and tendon at the molecular, cellular, tissue and organ levels. Also, he has pioneered the use of robotic technology to study the function of ACL and to improve ACL reconstruction procedures. When combining it with biplanar fluoroscopy, he and his team will be able to better characterize mechanisms of ACL injury and find better ways for its prevention.

Dr. Woo has educated over **460** orthopaedic surgeons, post-doctoral fellows and students from all around the globe including, Japan, Germany, Greece, Italy, Taiwan, Turkey, Korea, Canada, England, Norway, India, Thailand, Hong Kong SAR, and China. He has also mentored **37** junior faculty members.

Dr. Woo has been a leader in Bioengineering and Orthopaedics. He has served as **Chair** of ASME's Bioengineering Division, United States National Committee of Biomechanics, and the World Council for Biomechanics as well as **President** for The Orthopaedic Research Society, American Society of Biomechanics, and International Society for Fracture Repair. He has also founded the International Symposium on Ligaments and Tendons (ISL&T) and World Association for Chinese Biomedical Engineers (WACBE).

Dr. Woo has been inducted into the Institute of Medicine, the National Academy of Engineering, and the Academia Sinica, only one of four persons who have gained all three of these honors.

He has also received the highest honors from many professional societies, including the Kappa Delta Award, the Herbert R. Lissner Medal, the O'Donoghue Sports Injury Research Award, the Giovanni Borelli Award, and the Muybridge Medal, among others. Most recently, he was given the prestigious Diamond Award for Distinguish Achievement from the University of Washington. In 1998, Dr. Woo received the Olympic Prize for Sports Science from the International Olympic Committee and the first Olympic gold medal at the Nagano Games in Japan.

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