



Achilles Tendon Structure in Distance Runners does not Change Following a Competitive Season

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Introduction

Achilles tendinopathy is a painful degeneration of the tendon that is ten-times more common in running athletes compared to age-matched peers.¹ Tendon loads in excess of twelve body weights are cyclically applied during running, which may be the driving factor in tendinopathy development in these athletes.² However, the progression of asymptomatic and symptomatic tendinopathies is not well understood.³ Structural changes associated with symptomatic tendinopathy such as decreased collagen alignment—or ‘organization’—and increased tendon thickness have both been reported in athletic populations. Previous work by our group has demonstrated that competitive collegiate distance runners have thicker and less organized tendons than their recreationally active peers even in the absence of signs or symptoms of tendinopathy.⁴ Similarly, hypertrophy of the Achilles tendon has been observed in elite and recreational athletes^{5,6} indicating a relationship between structural differences and the cyclic loading experienced during running.

These differences in tendon structure have been linked to decreased mechanical properties in both humans⁷ and small animals⁸ in response to tendinopathy and acute injury, respectively. In contrast, the mechanical properties of healthy endurance runners’ tendons have been shown to be similar to non-runners⁹ despite being structurally different. Our prior work has demonstrated that trained runners have structurally different tendon prior to the rigors of a competitive running season. However, it is unclear how tendon structure in highly-trained runners changes in response to prolonged bouts of training.

Therefore, the aim of this study was to prospectively quantify Achilles tendon structure of competitive distance runners at the beginning and completion of a cross country season. We hypothesized that, in the absence of injury, there would be no significant changes in tendon thickness, organization, or echogenicity for a runner with a habituated tendon. Should signs or symptoms of tendinopathy develop, there should be detectable changes in thickness, organization, and echogenicity as a result. Understanding how a tendon responds to the continued demands of

high risk activities such as running is crucial to understanding how tendon disorders progress.

Methods

Nineteen collegiate cross country runners (9 females; Age: 19 ± 1.5 years; Height: 172 ± 7 cm; Weight: 60.4 ± 8 kg) provided written consent in this IRB approved study. All participants had no signs or symptoms of Achilles tendinopathy before or after participation. Subjects were seen a week prior to and a week following competing in a Division I NCAA Cross Country season. Each study visit consisted of a self-reported assessment of tendon health and a quantitative ultrasound assessment. Subjects were asked to fill out a clinical outcome questionnaire (VISA-A)¹⁰ to determine the level of health and function. The structure of the tendon was determined by measuring the level of organization present in its collagen fascicles through ultrasonography.

Longitudinal B-mode ultrasound images of the of the mid-substance of the right Achilles tendon were acquired while subjects lay prone on a treatment table with ankles placed in the resting position off the end of the table. Images were acquired using an 18 MHz transducer (L18-10L30H-4, SmartUs, TELEMED) with a scanning width of 3 cm (scan parameters: Dynamic Range: 72dB; frequency: 18 MHz; gain: 47 dB). Collagen organization was quantified in the ultrasound images using custom-written software.¹¹ This image processing algorithm is a computational analog to crossed polarizer imaging, which assesses collagen fascicle alignment and quantifies tendon ‘organization’ as the circular standard deviation (CSD) of these collagen structures and has been shown to be reliable in Achilles tendon.¹² These images were also used to quantify the longitudinal thickness and mean echogenicity of the tendon.

Tendon organization, thickness, and echogenicity as well as VISA-A scores were compared between the two study visits using two-way paired t-tests. Additionally, effect sizes were determined for any differences found to be statistically significant ($P < 0.05$). Effect sizes were reported using Cohen’s d, calculated as the mean difference divided by the pooled standard deviation.¹³

Table 1. Mean and standard deviation values for VISA-A as well as tendon thickness, organization, and mean echogenicity for measurement sessions 1 and 2 are shown. The percent changes between these measurements is also reported as well as their statistical significance. Thickness was found to increase significantly but the effect size of this increase was small.

	Session I (Mean ± STD)	Session II (Mean ± STD)	Percent Change	Significance
VISA-A (out of 100)	93 ± 8.1	94 ± 6.9	1%	$P > 0.05$
Thickness (cm)	0.54 ± 0.1	0.56 ± 0.1	7%	$P < 0.05$
Organization (CSD)	9.4 ± 0.7	9.2 ± 0.4	2.50%	$P > 0.05$
Mean Echogenicity (%)	14 ± 3.5	15 ± 5.8	4.50%	$P > 0.05$

Results

Achilles tendon symptoms did not develop in any of the runners, which were confirmed by no change in VISA-A scores between the pre- and post-season sessions ($P > 0.1$, Table 1). Similarly, tendon organization and echogenicity did not change over the course of the competitive season between the two sessions ($P > 0.05$, Table 1). Tendon thickness increased by 7% ($P < 0.001$, Table 1) but the effect size of this change was small ($d = 0.36$).

Discussion

We confirmed our hypothesis that competitive distance runners have Achilles tendon structure that is habituated to prolonged cyclic loading and does not change over a competitive season. These findings agree with previous work that showed that collegiate distance runners do not undergo Achilles tendon hypertrophy throughout a competitive season.¹⁴ This habituated tendon appears to be a protective adaptation, allowing trained runners to cyclically load their tendons without injury. Mechanically, the thicker-habituated tendon should undergo the same amount of strain observed in a naïve tendon at lower levels of stress. This would result in similar maximal ankle torque generation potential while decreasing the impact of the rapid loading experienced during distance running.

The processes by which tendon remodels from a naïve to a habituated—and from a healthy to a pathologic state—are still not well understood. Exercise has been shown to increase levels of collagen synthesis in humans^{15,16} but the effects of this increase has not been directly linked to tendon remodeling. Additionally, different types of running demands appear to have different effects on tendon remodeling. Sprinters, for example, have stiffer Achilles tendons than distance runners and non-runners,⁽⁹⁾ though these findings have not been linked to tendon structure. As a result, there is a need to link the structural differences of habituated tendon with function to better understand the remodeling process and to elucidate the mechanisms that drive pathology.

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