

# Achilles tendon adaptation and Achilles tendinopathy in running

E Maas<sup>1\*</sup>, I Jonkers<sup>1</sup>, K Peers<sup>2</sup>, B Vanwanseele<sup>1</sup>

## Abstract

### Introduction

Achilles tendinopathy is a common overuse injury in runners and related to maladaptation of the Achilles tendon. The aim of the present review is to provide an overview of the literature on the adaptation of the Achilles tendon to running and the maladaptation caused by overloading of the tendon that leads to Achilles tendinopathy.

### Discussion

Cross-sectional studies reveal that runners have thicker Achilles tendons than non-runners, but no difference in its stiffness is found. Patients with Achilles tendinopathy have a larger Achilles tendon cross-sectional area, but lower stiffness than those of healthy people. Longitudinal studies on the adaptation of Achilles tendon mechanical properties are scarce and do not find a change in Achilles tendon size or stiffness.

### Conclusion

More longitudinal studies are necessary to find out what magnitude of strain is needed to trigger an adaptational response in tendons and to define a threshold between loading and overloading of the Achilles tendon.

## Introduction

The Achilles tendon is one of the most injured tendons in the human body. Especially, runners are at high risk of injuring their Achilles tendons: 8% of

novice runners develop Achilles tendinopathy<sup>1</sup> and 56% of elite runners report to have suffered from Achilles tendinopathy at some point in their career<sup>2</sup>. Achilles tendinopathy is defined as a combination of pain in the Achilles tendon area, swelling and impaired performance<sup>3</sup>. The exact injury mechanism for Achilles tendinopathy is currently unknown.

Similar to other musculoskeletal tissues, the Achilles tendon responds to mechanical loading with structural adaptations that make the tendon stronger and more resistant to strain<sup>4,5</sup>. Although a certain level of strain is required for these adaptations, it has been suggested that too high strains will overload the tendon and cause microdamage<sup>6</sup>. Following exercise, collagen synthesis is enhanced<sup>7</sup>. Simultaneously, the degradation of collagen protein also increases, outweighing the collagen synthesis<sup>8</sup> (see Figure 1). In the 18–36 h after exercise, there is a negative net balance in collagen levels (catabolic state so loss of collagen), which after 36 h turns positive up to 72 h after exercise (anabolic state so regeneration of collagen)<sup>6</sup>. These data suggest that apart from the level of strain, sufficient rest between training sessions needs to be respected in order to reduce the net loss of collagen content, which may lead to tendon injury. It therefore seems that there is a subtle balance between loading and overloading a tendon, depending on several factors including training volume, type of loading and recovery time<sup>6</sup>.

The purpose of the present review is to provide an overview of the healthy adaptation mechanisms of Achilles tendon to running and

on the other hand, maladaptations caused by overloading of the tendon that lead to Achilles tendinopathy. These insights will be discussed in the context of training recommendations for injury-free running.

## Discussion

### Adaptation of tendon to running

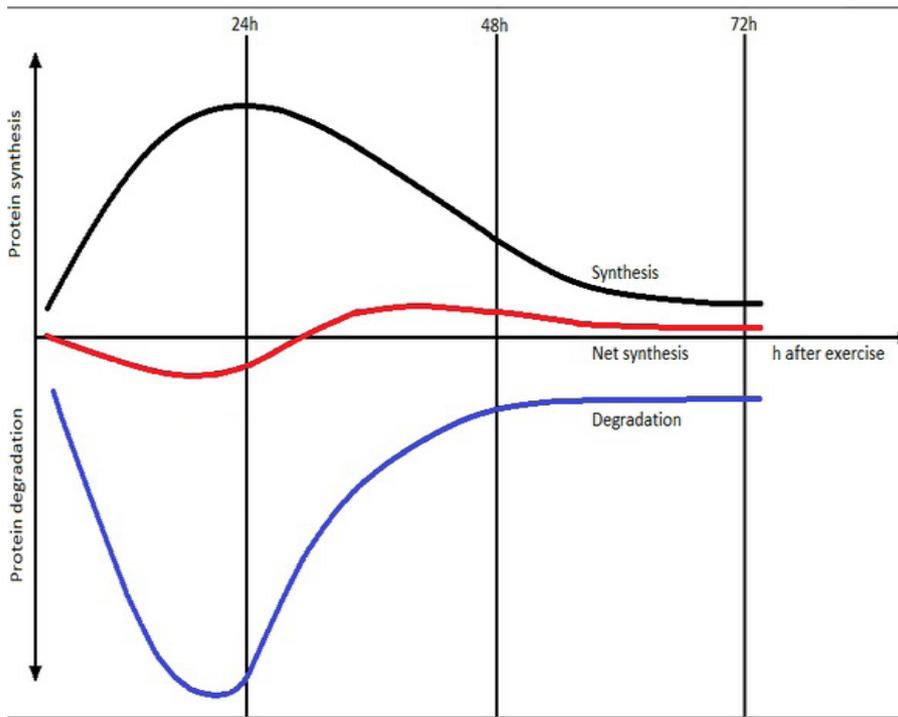
To evaluate the adaptations of the Achilles tendon to running, a number of studies compared the mechanical properties of Achilles tendons of runners with those of non-runners. Achilles tendon stiffness can be measured during isometric plantar flexion contractions during which tendon elongation is measured using ultrasonography. Tendon stiffness is then defined as the slope of the force/elongation relationship<sup>9</sup>. Achilles tendon's cross-sectional area (CSA) can be measured from MRI scans or ultrasonograms<sup>9</sup>. The findings of these studies are summarised in Table 1.

It has been found that collagen turnover is increased after a bout of running<sup>15</sup>, suggesting that running causes tendon hypertrophy. Indeed, six out of seven studies found a larger Achilles tendon CSA in runners compared with non-runners<sup>7,8,11–13</sup>, although sometimes this difference was only present in specific parts of the Achilles tendon<sup>7,12</sup> or only if the Achilles tendon CSA was normalised to body weight<sup>8</sup>. Other studies examining the effect of a (non-running) training programme on tendon size and stiffness also found that tendon hypertrophy following training is region specific<sup>16</sup>. This may be due to the difference in loading of different parts of the Achilles tendon: the distal part of the tendon is not only

\*Corresponding author  
Email: ellen.maas@faber.kuleuven.be

<sup>1</sup> KU Leuven, Department of Kinesiology, Tervuursevest 101 - box 1501, 3001 Leuven, Belgium

<sup>2</sup> KU Leuven, Department of Development and Regeneration, UZ Herestraat 49 - box 7003, 3000 Leuven, Belgium



**Figure 1:** (adapted from Magnusson et al.(6)): protein synthesis and degradation after a bout of exercise.

subject to tensile loads but also to compressive loads caused by the compression of the space between the distal part of the tendon and the calcaneus during dorsiflexion<sup>12</sup>.

Only one out of four studies found a difference in tendon stiffness between runners and non-runners<sup>11</sup>. More specifically, sprinters had larger tendon stiffness compared with long distance runners and non-runners. There was no difference in tendon stiffness between long distance runners and non-runners. This suggests that the strain imposed on the Achilles tendon by submaximal running is not sufficient to trigger adaptation responses, whereas sprint training does generate such responses. CSA was not measured in this study, so it cannot be concluded whether the larger stiffness in sprinters was due to tendon hypertrophy or architectural changes. Theoretically, tendon stiffness is directly related to tendon CSA, but this is not consistently reflected in the results from the studies in Table 1. Rosager et al.<sup>13</sup> found

larger Achilles tendon CSA in runners compared with controls, but no difference in tendon stiffness. Kubo et al.<sup>8</sup> and Hansen et al.<sup>9</sup> found no difference in both absolute CSA and tendon stiffness between runners and controls.

Tendon properties, loading and adaptations are gender specific. Only one study included in Table 1 examined the Achilles tendon properties of female runners versus female non-runners and did not find a difference in CSA<sup>14</sup>. Moreover, the difference in CSA between various portions of the Achilles tendon was much larger in men than in women (in male runners, the CSA of the distal part of the tendon was 75% larger than the proximal part, compared with 11% in female runners), indicating that the region-specific adaptation of Achilles tendon may be gender specific. Interestingly, injuries to the Achilles tendon occur more frequently in men than in women. This could be due to a larger plantar flexor moment arm in men, which causes more strain on the

Achilles tendon for a given change in joint angle<sup>17</sup>. Since tendon adaptation has also been linked to oestrogen levels<sup>14</sup> and collagen synthesis is found to be lower in women compared with men, and rises less after exercise<sup>18</sup>, it is not surprising that tendon adaptation is gender specific. Thus, more research is needed to find effects of running on mechanical properties of tendon in women.

To see the course of tendon adaptation to a running programme and in order to find out mechanisms behind Achilles tendinopathy, it is necessary to do longitudinal follow-up studies. Yet, the majority of the studies on the effects of running on mechanical properties of the Achilles tendon have a cross-sectional design, comparing runners with non-runners. With this study design, it is impossible to detect cause-effect relationships between running and the outcome variables. Changes in mechanical properties could be not only due to the running programme but also to the subject-specific running style, pre-existing conditions or other variables. Only one longitudinal study on the effects of a running programme on tendon mechanical properties could be found<sup>9</sup>. No changes in tendon CSA and tendon stiffness were found after 32 weeks of running. It should be noted that this study had a relatively small sample size ( $N = 11$ ) and included both men and women. Therefore, it may be possible that no differences in tendon size and stiffness were found because tendon adaptations are gender specific. It could also be that it takes more time for the tendon to adapt than the 32-week running programme that was administered in this study. Yet, since there was an insignificant increase in tendon stiffness of 5%–7%, it could be possible that a difference would be found in a larger sample ( $N = 11$ ). Several of the studies presented in Table 1 suffer from small sample size (e.g. of Rosager et al.<sup>13</sup>).

**Table 1 Achilles tendon properties in runners versus non-runners**

Study	Design	Exp. group	Contr. group	Findings
Abate et al. <sup>10</sup>	CS	21 runners; 25 overweight runners	16 non-runners; 19 overweight non-runners	AT thickness (midportion) was greater in runners than in non-runners. Overweight people had more sonographic abnormalities than non-overweight people. Overweight runners had more sonographic abnormalities than non-overweight runners
Arampatzis et al. <sup>11</sup>	CS	28 male sprinters; 28 male endurance runners	10 non-active male controls	No significant difference in AT stiffness between long distance runners and controls; stiffness in sprinters was larger than in long distance runners and controls
Hansen et al. <sup>9</sup>	Long	11 untrained subjects (7 male, 4 female)	-	After 32-week running intervention: no change in AT CSA; no change in plantar flexor moment, no change in corresponding tendon-aponeurosis displacement; insignificant increase in tendon stiffness
Kongsgaard et al. <sup>7</sup>	CS	8 male elite long distance runners 6 male AT rupture patients (avg 1.2 years post-surgery)	9 male elite kayakers	AT CSA of runners > kayakers AT CSA of runners > AT rupture subjects
Kubo et al. <sup>8</sup>	CS	15 male long distance runners	21 untrained subjects	Relative AT thickness (to body mass) of LD runners >controls, no difference in absolute AT thickness. No difference in AT stiffness
Magnusson and Kjaer <sup>12</sup>	CS	6 male long distance runners	6 male non-runners	Runners have greater AT CSA than non-runners in the distal, but not the proximal part of the tendon
Rosager et al. <sup>13</sup>	CS	5 male runners	5 male untrained	AT CSA of runners > non-runners; no difference in stiffness and stress/strain relationship
Westh et al. <sup>14</sup>	CS	10 female runners, 10 male runners	10 female non-runners	No difference in normalised AT CSA between female runners and non-runners. Male runners AT CSA > female runners

AT, Achilles tendon; CSA, cross-sectional area; LD, long distance.

### Mechanical and structural properties of tendons with Achilles tendinopathy

To evaluate the effects of overloading on Achilles tendons, differences in tendon mechanical properties between patients with Achilles tendinopathy and healthy subjects were assessed using ultrasonography and/or MRI. The results of various studies on this subject are displayed in Table 2.

All studies in Table 2 show that Achilles tendinopathy is associated with a larger CSA and a decrease in tendon stiffness. Since stiffness is

directly related to the tendon CSA, it would be expected that a larger tendon CSA would yield a stiffer tendon. However, in the case of Achilles tendinopathy, the increase in CSA is most likely due to accumulation of fluid and disorganisation of collagen fibres<sup>21</sup>. So possible explanations for decreased stiffness in Achilles tendinopathy patients relate to disorganisation of tendon and collagen fibres, hypervascularisation (the random formation of blood vessels), degeneration of collagen fibres and an increase in extracellular matrix<sup>3</sup>. Disorganisation of the tendon fibres

leads to an increased vulnerability for further (micro)trauma, which increases the risk for tendon rupture<sup>21</sup>.

Another factor that may contribute to the injury mechanism is a disturbed balance between energy storage and dissipation in the tendon: Wang et al.<sup>22</sup> found increased hysteresis in the injured Achilles tendon of patients with unilateral tendinopathy, indicative of increased dissipation of energy into heat. This causes an increase in metabolic demand and hyperthermia within the Achilles tendon, which may lead to tendon degeneration.

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Table 2 Differences in tendon mechanical properties between patients with Achilles tendinopathy and healthy controls

Study	Exp. group	Contr. group	Findings
Leung and Griffith <sup>19</sup>	30 Achilles tendons; 21 patients (9 male 12 female)	100 Achilles tendons; 50 subjects 22 male 28 female	Tendinopathic tendons CSA > controls at midpoint and calcaneal insertion; disruption of fibrillar pattern in 20% of tendinopathic tendons vs. 0% controls; neovascularisation <sup>a</sup> in 46.7% of tendinopathic tendons vs. 0% in controls; focal calcification in 6.7% of tendinopathic tendons vs. 2% in controls
Child et al. <sup>20</sup>	16 male recreational runners with Achilles tendinopathy	16 male recreational runners	Achilles tendon thickness was greater in symptomatic Achilles tendon than controls, at site perpendicular to medial malleolus, but not at insertion. Achilles tendon strain at musculotendinous junction was larger in symptomatic Achilles tendon than in controls => higher compliance/lower stiffness
Arya and Kulig <sup>21</sup>	12 male runners with Achilles tendinopathy	12 male runners	Achilles tendon stiffness patients < controls; Achilles tendon elongation patients > controls; Achilles tendon CSA patients > controls; Achilles tendon stress patients < controls; Achilles tendon strain patients > controls; Young's modulus (stress/strain) patients < controls
Wang et al. <sup>22</sup>	17 male athletes with Achilles tendinopathy	Non-symptomatic leg of same subjects	Stiffness: lower in injured leg, hysteresis: higher in injured leg; elastic energy stored: lower in injured leg; elastic energy released: lower in injured leg

<sup>a</sup>Influx of new blood vessels in the affected area.

As indicated in the previous section injury, injury mechanisms of Achilles tendinopathy are best studied using longitudinal studies; all studies summarised in Table 2 have a cross-sectional study design. Therefore, their results reflect the pathological reaction of the tendon, but the mechanisms behind these reactions are still unknown.

### Biomechanical risk factors for development of Achilles tendinopathy

Various risk factors for the development of Achilles tendinopathy can be found in the literature. Both intrinsic and running-related variables seem to contribute: in a prospective study, Mahieu et al.<sup>23</sup> found that people with low plantar flexor strength before the start of a running programme were more likely to develop Achilles tendinopathy. Using a similar design, Van Ginckel et al.<sup>1</sup> found a decrease in posterior-anterior displacement of the centre of force and a laterally directed force distribution underneath the forefoot

at 'forefoot flat' in subjects who developed Achilles tendinopathy. This suggests that runners with a running pattern with less forward force transfer underneath the foot and a more lateral foot roll-over are more at risk of developing Achilles tendinopathy. Further, Almonroeder et al.<sup>24</sup> found in a cross-sectional study that non-rearfoot strike runners had greater Achilles tendon loading than rearfoot strike runners, suggesting that running style influences the loading on the tendon and thus may also influence the adaptation of the tendon.

The studies of Mahieu et al.<sup>23</sup>, Van Ginckel et al.<sup>1</sup> and Almonroeder et al.<sup>24</sup> focus on the loading of the musculoskeletal structures. However, it is necessary to take into account the mechanical properties (i.e. tendon stiffness, CSA) of the Achilles tendon, as these reflect the loading capacity of the tendon. Such an analysis of loading and loading capacity would allow one to define individual loading threshold of the Achilles tendon.

### Conclusion

In conclusion, the human Achilles tendon adapts to running by increasing its CSA. It is still unknown how long these adaptations take and which loading volume is required for the tendon tissues to adapt. One longitudinal study indicates that a 9-month running programme for novice runners does not lead to structural adaptations. Similarly, it is unknown which running volume and intensity will cause overloading of the tendons. When following a structured running programme, mechanical loading of the tendon is increased, which normally leads to tissue adaptations. If these adaptations do not occur, the runner is at risk for developing Achilles tendinopathy. Strength and plyometric training cause an increase in Achilles tendon CSA and stiffness. It is therefore advisable for endurance runners to add strength and/or plyometric exercises to their training programme in order to protect against Achilles tendon injury.

More research with a prospective design is needed to determine

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the relationship between running volume and tendon adaptation. Furthermore, adaptations in female subjects should be investigated, as there appears to be a gender difference in tendon adaptation. Also considering possible risk factors for the development of Achilles tendinopathy, prospective studies are needed.

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