

THE ROLE OF DETRAINING IN TENDON MECHANOBIOLOGY

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Keywords

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Abstract

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Introduction: Several conditions such as training, aging, estrogen deficiency and drugs could affect the biological and anatomophysiological characteristics of the tendon. Additionally, recent preclinical and clinical studies examined the effect of detraining on tendon, showing alterations in its structure and morphology and in tenocyte mechanobiology. However, there is a paucity of data examining the impact that cessation of training may have on tendon. In practice, we do not fully understand how tendons respond to a period of training followed by sudden detraining. Therefore, within this review, we summarize the studies where tendon detraining was examined.

Materials and methods: A descriptive systematic literature review was conducted by searching three databases (PubMed, Scopus and Web of Knowledge) on tendon detraining. Original articles in English from 2000 to 2015 were included. In addition, the search was extended to the reference lists of the selected articles. A public reference manager ("www.mendeley.com") was used to delete duplicate articles.

Results: An initial literature search yielded 134 references (www.pubmed.org: 53; www.scopus.com: 11; www.webofknowledge.com: 70). 15 publications were extracted based on the title for further analysis by two independent reviewers. Abstracts and whole articles were then reviewed to detect if they met inclusion criteria.

Conclusions: The revised literature comprised 4 clinical studies and an in vitro and three in vivo reports. Overall, the results showed that tendon structure and properties after detraining are compromised, with an alteration in the tissue structural organization and mechanical properties. Clinical studies usually showed a lesser extent of tendon alterations, probably because preclinical studies permit an in-depth evaluation of tendon modifications, which is hard to perform in human subjects. In conclusion, after a period of sudden detraining (e.g. after an injury), physical activity should be restarted with caution, following an appropriate rehabilitation program. However, further research should be performed to fully understand the effect of sudden detraining on tendons.

Ethics statement

(Authors are required to state the ethical considerations of their study in the manuscript including for cases where the study was exempt from ethical approval procedures.)

Did the study presented in the manuscript involve human or animal subjects: No

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30 **ABSTRACT**

31 **Introduction:** Several conditions such as training, aging, estrogen deficiency and drugs could affect
32 the biological and anatomic-physiological characteristics of the tendon. Additionally, recent
33 preclinical and clinical studies examined the effect of detraining on tendon, showing alterations in
34 its structure and morphology and in tenocyte mechanobiology. However, few data evaluated the
35 importance that cessation of training might have on tendon. Basically, we do not fully understand
36 how tendons react to a phase of training followed by sudden detraining. Therefore, within this
37 review, we summarize the studies where tendon detraining was examined.

38 **Materials and methods:** A descriptive systematic literature review was carried out by searching
39 three databases (PubMed, Scopus and Web of Knowledge) on tendon detraining. Original articles in
40 English from 2000 to 2015 were included. In addition, the search was extended to the reference lists
41 of the selected articles. A public reference manager ("www.mendeley.com") was adopted to remove
42 duplicate articles.

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44 www.scopus.com: 8 11; www.webofknowledge.com: 9 70). 11 15 publications were extracted
45 based on the title for further analysis by two independent reviewers. Abstracts and complete articles
46 were after that reviewed to evaluate if they met inclusion criteria.

47 **Conclusions:** The revised literature comprised 4 clinical studies and an *in vitro* and ~~two~~ three *in*
48 *vivo* reports. Overall, the results showed that tendon structure and properties after detraining are
49 compromised, with an alteration in the tissue structural organization and mechanical properties.
50 Clinical studies usually showed a lesser extent of tendon alterations, probably because preclinical
51 studies permit an in-depth evaluation of tendon modifications, which is hard to perform in human
52 subjects. In conclusion, after a period of sudden detraining (e.g. after an injury), physical activity
53 should be taken with caution, following a targeted rehabilitation program. However, further
54 research should be performed to fully understand the effect of sudden detraining on tendons.

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56 **Key words:** tendon, tenocyte, detraining, sudden detraining, systematic literature review

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62 INTRODUCTION

63 Tendons are a specialized tissues that join muscle to bone and are composed by extracellular
64 collagen fibers arranged in regular arrays (Aslan, 2008). This mechanosensitive tissue shows
65 detailed mechanical properties that allow it to adapt and respond to loading transmitted by muscles
66 **(Fang, 2015)**. This load transfer provide the principal mechanical stimulus for tendon cells
67 **(Kondratko-Mittnacht 2015)**. These tensile loads are diverted to tendon cells through different
68 matrix compartments and components. At cellular level, by various transmembrane structures and
69 pathways, they are transduced from the exterior to intracellular biochemical responses **(Maeda,**
70 **2015; Kondratko-Mittnacht 2015)**.

71 While physiologic loads are required to maintain tendon homeostasis, (Galloway, 2013) unusual
72 loading could direct to tendon injury, either through an acute traumatic injury or chronic,
73 degenerative process (i.e., tendinopathy) resulting from an increase of microdamages and an altered
74 cell/matrix response (Arnoczky, 2007; Magnusson, 2010). **Histopathologically, tendinopathy is a**
75 **unsuccessful healing response, represented by altered tenocytes proliferation, disruption and**
76 **impaired organization of collagen fibers, increase in non collagenous matrix and**
77 **neovascularization (Maffulli, 2011). In the chronic stage of tendinopathy, inflammation is**
78 **absent or minimal, nevertheless it could play a role only in the initiation, but not in the**
79 **propagation and progression, of the disease process (Maffulli, 2010). Even if tendinopathies**
80 **also comprise conditions of damage to the tendon without symptoms, these pathologies**
81 **frequently occur with pain in the injured tendon, which is accentuated or appears during**
82 **palpation of the affected area or during active and passive movements involving the tendon**
83 **(Franceschi, 2014)**. Tendon injury may not only lead in the lack of mobility or irregular joint
84 kinematics, but could also result in damages to tissues adjacent to the joint. **Muscle atrophy**
85 **subsequent to tendon rupture is a frequent complication found by physicians and orthopedic**
86 **surgeons. This condition proves significantly weaker musculature resulting in unfavorable**
87 **functional consequences, with a consequent reduction in muscle force generation (Sandri,**
88 **2008; Zhang, 2013)**. Despite previous studies showed complete histological and biochemical
89 **characteristics of tendons rupture and some of these have been included into the clinical**
90 **scenario, little is known concerning the mechanical response of muscles to tendon injury**
91 **(Sandri, 2008; Zhang, 2013; Jamali, 2000; Charvet, 2012)**. However, recently Zhang et al.
92 **demonstrated that tendon rupture has a supplementary influence on muscle biomechanics in**
93 **comparison to disuse (Zhang, 2013)**.

94 Due to their poor healing ability, tendon injuries represent an increasing problem in orthopedics as
95 physicians are faced with a growing demand in sports and recreation and in the aging population

96 (Kaux, 2011). Thus, primary disorders of tendons are a widely distributed clinical problem in
97 society and hospital evidence and statistical data suggest that some tendons are more susceptible to
98 pathology than others; these are the rotator cuff, Achilles tibialis posterior and patellar tendons.
99 Although there are no specific figures in relation to tendon disease, several studies show that 16%
100 of the population is affected from tendon pain (Urwin, 1998) and this rises to 21% when the
101 statistics shift to elderly hospitals and community populations (Urwin, 1998, Chard, 1991). These
102 numbers supplementary enhance in the sports community, in fact it was reported that 30 to 50% of
103 all sporting injuries involve tendons (Kannus, 1997). Ordinarily, the major conditions affecting
104 tendons are tendinitis and tendinosis; the first assumed to be accompanied by inflammation and
105 pain, whereas the second can be caused by tendinous degeneration (Maffulli, 1998). It is assumed
106 that these conditions are seldom spontaneous (Gibson, 1998) and are not caused by single factors.
107 Rather, they are the end result of a variety of pathological processes (Riley, 2004; Rees, 2006)
108 which can ultimately lead to the main clinical problem: loss of tissue integrity with full or partial
109 tendon rupture .

110 Many intrinsic and extrinsic factors such as aging, gender, anatomical variants, obesity, systemic
111 diseases, estrogen deficiency, drugs, sporting activities, physical loading, occupation, and
112 environmental conditions could affect the biological and anatomo-physiological characteristics of
113 the tendon (Sandberg, 2015; Frizziero, 2014; Galdiero, 2014; Oliva, 2014a, 2014b; Snedeker,
114 2014; Abate 2014; Hast, 2014; Boivin, 2014; Berardi, 2014; Franchi, 2013; Frizziero, 2013;
115 Malliaras, 2013; Moerch, 2013; Torricelli, 2013; Frey, 2007; Holmes, 2006; Torricelli, 2006;
116 Nakama, 2005;). Thus, over the past decade, tendon and tenocyte adaptations in relation to
117 immobilization, training, aging and medications have been the center of an growing number of
118 studies (Maffulli, 2003; Sharma, 2005; Stanley, 2008; Torricelli, 2006; Torricelli, 2013).

119 While proper mechanical loads at physiological levels are typically helpful to tendons in terms of
120 enhancing its mechanical properties, recent preclinical and clinical studies examining the effect of
121 detraining on tendon, showed alterations in its structure and morphology and in tenocyte
122 mechanobiology. However, there is a paucity of data that evaluated the impact that detraining may
123 have on tendon. Thus, it has not yet been understood how tendons behave to a period of training
124 followed by cessation of training. Nevertheless, to guide rehabilitation and/or athletic programs it is
125 necessary to elucidate tendon adaptation after sudden detraining. Therefore, within this descriptive
126 systematic literature review, we summarize the studies where tendon detraining was examined.

127

128 **MATERIALS AND METHODS**

129 **Descriptive literature review**

130 According to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA)
131 a systematic search was carried out for this descriptive literature review (see **Figure 1** for details) in
132 three databases (www.pubmed.org, www.webofknowledge.com, www.scopus.com). The keywords
133 were “tendon detraining”, “tendon detraining associated damage”, “tendon sudden detraining”,
134 **“tendon disuse”, “tendon discontinuous training associated damage”, “tendinopathies and**
135 **discontinuous training”, “tendinopathies and detraining”, “tenocytes detraining associated**
136 **damage”, “tenocytes discontinuous training associated damage”**. We sought to identify studies
137 in which tendon detraining was examined. Publications from 2005 to 2015 (original articles in
138 English) were included. The reference lists from the articles included in this review were analyzed
139 to recognize additional studies that were not found by the initial search. A public reference manager
140 (“www.mendeley.com”) was used to delete duplicate articles.

141

142 **RESULTS**

143 An initial literature search yielded **34 134** references. **Seventeen Fifty-three** articles were identified
144 using www.pubmed.org, **9 70** articles using www.webofknowledge.com and **8 11** articles were
145 found in www.scopus.com. The resulting references were submitted to a public reference manager
146 (Mendeley 1.13.8, “www.mendeley.com”) to delete duplicate articles. Of the **20 76** remaining
147 articles, **11 15** publications were extracted based on the title for further analysis. Abstracts and
148 whole articles were then reviewed to ascertain whether the publication met the inclusion criteria and
149 **seven-8** articles (**3 4** preclinical studies, 1 *in vitro* and **2 3** *in vivo*, and 4 clinical studies) were
150 considered appropriate for the review (**Figure 1**). From the reference lists of the included articles,
151 no- supplementary publications were identified. We did not perform meta-analyses of the selected
152 studies, but quoted the results in a descriptive fashion.

153

154 **Figure 1:**Literature search strategy and criteria.

155 **Preclinical studies**

156 This revised literature comprised **3 4** preclinical studies, an *in vitro* and ~~two~~ **three** *in vivo* reports,
157 respectively on tenocytes from patellar tendon (**Salamanna, 2015**) and on patellar (**Frizziero, 2011**
158 **and 2015**) and gastrocnemius (**Foutz, 2007**) tendon of detrained ~~rats animals~~ (~~Salamanna, 2015;~~
159 ~~Frizziero, 2011 and 2015~~) . Concerning the *in vitro* study patellar tendon tenocytes from rats
160 subjected to training and to sudden detraining were examined. Rats were trained for 10 weeks on a
161 treadmill (speed of about 25 m/min, corresponding to ~65–70% VO₂max) and successively caged
162 without exercise for further 4 weeks. Tenocytes from patellar tendon were cultured to evaluate
163 morphology, viability, proliferation and metabolic activity. It was found that detraining in the short-

164 term alters tenocyte synthetic and metabolic activity (C-terminal-propeptide of type I collagen,
165 collagen III, fibronectin, aggrecan, tenascin-c, interleukin-1 β , matrix-metalloproteinase-1 and-3).
166 These results indicated that tenocytes do not merely have a passive role but play an important
167 function during detraining (Salamanna, 2015). Similarly results were found by the same authors
168 also when the patellar tendons of detrained rats were studied by histology and histomorphometry
169 (Frizziero, 2011 and 2015). In fact, the studies showed alteration in tendon morphology and also in
170 its enthesis due to discontinuation of training. These alteration involved proteoglycan content,
171 collagen fiber organization with an increase of collagen III and a decrease of collagen I, which
172 means less resistance to stress, and a related increased risk of rupture. **Differently from the above
173 mentioned studies, Foutz et al. investigated the mechanical adaptability responses due to
174 disuse on the biomechanical properties of the gastrocnemius tendon of chicks (Foutz, 2007).
175 Chicks were trained for 3 weeks on a treadmill (speed of 0.22 m/s, for 5 min) and successively
176 immobilized in a whole body suspension system for further 2 weeks. It was found that
177 structural strength and toughness of the gastrocnemius tendon were reduced by 10 and 30%,
178 respectively, whereas the material strength, material toughness, and material stiffness of the
179 tendon increased by approximately 75, 65, and 70%, respectively. These results showed that
180 the chicken gastrocnemius tendon reacts to mechanical disuse as foretold by the
181 mechanobiology process (Foutz, 2007).**

182

183 **Clinical studies**

184 The PubMed, Web of Knowledge and Scopus search strategy identified 4 clinical papers that
185 examined the impact that detraining may have on tendons. Several studies showed that tendon
186 characteristics influence the performances during stretch-shortening cycle exercises (Bojesen-
187 Moller, 2005; Kubo, 2007; Stafilidis, 2007); thus, information on the time course of changes in
188 tendon characteristics during training and detraining is critical for the progress of performances in
189 the athletic field. To evaluate the time course of modifications in mechanical and morphological
190 properties of tendon during detraining, Kubo et al (Kubo, 2010) examined these variables in eight
191 volunteered men that executed unilateral knee extension exercise in a seated position. Subjects were
192 trained 4 times per weeks for 3 months and detrained for the following three months. Results of this
193 study showed that tendon stiffness was significantly increased after 3 months of training, while the
194 maximal elongation was unaltered. Conversely, during the detraining period, tendon showed greater
195 values of maximal elongation compared to the post-training, and tendon stiffness decreased to the
196 pre-training levels after 2 months of detraining (Kubo, 2010). With a similar methodology, the same
197 authors in 2012 focused more specifically on the alterations found in the human Achilles tendon

198 during training and detraining (Kubo, 2012). In addition, they measured the blood volume and
199 oxygen saturation of tendon, and evaluated the serum concentrations of markers of collagen type I
200 synthesis. Results were similar to the previous study ones: the elongation values did not change
201 after training but increased significantly during detraining; tendon stiffness increased only after
202 three months of training and rapidly decreased during detraining. Thus, Authors showed that during
203 detraining, the sudden decrease in tendon stiffness might be linked to modifications in the structure
204 of collagen fibers within the tendon. In addition, no significant alterations in blood supply or
205 collagen synthesis were observed (excluding an increase in procollagen peptides after 2 months of
206 training) (Kubo, 2012).

207 Recently McMahon et al (McMahon, 2013) evaluated the patella tendon properties during
208 detraining (1 months), after a 3-months period of training with different strains. The patella moment
209 arm, the perpendicular distance between the tibiofemoral contact point and the mid-portion of the
210 tendon, was estimated using dual-energy x-ray absorptiometry (DEXA) scan images. Tendon
211 elongation and stiffness were measured by ultrasonic analyses and tendon forces were calculated as
212 the ratio between the measured torque and the patella moment arm. Furthermore, they evaluated the
213 circulating transforming growth factor (TGF)- β 1 levels as it is associated to exercise-induced
214 response to mechanical loading of muscle and tendon. The authors found no significant alterations
215 in patella tendon dimensions or circulating TGF- β 1 levels following training or detraining.
216 However, the training groups with the muscle-tendon complex at a lengthened position or over a
217 wide range of motion better maintained adaptations compared to the training in a shortened position
218 subsequent to detraining, with a pattern of slower loss of progress at the early phase of detraining in
219 all training groups.

220 Finally Kannas and colleagues (Kannas, 2014) analyzed the effect of 4 weeks of detraining on the
221 mechanical properties of medial gastrocnemius aponeurosis into two groups that performed
222 plyometric training on incline and plane ground. They evaluated the aponeurosis strain of medial
223 gastrocnemius and found that it decreased after detraining; the ankle muscle tendon complex
224 properties withdrew to the pre-training values with lower performances. These findings suggested
225 that after four weeks of detraining, ankle muscle tendon complex properties withdraw to the pre-
226 training values with lower performance (Kannas, 2014).

227

228 **DISCUSSION**

229 The tendon is a connective tissue responsible for the transmission of force from the muscular tissue
230 to the bones, promoting body movement. It is not a static tissue, preferentially it adapts itself in

231 compliance to the level, direction and frequency of the load that is applied to it with a process of
232 remodeling possibly executed by tenocytes.

233 It was shown that appropriate mechanical loads are useful to tendons by improving their anabolic
234 processes and it is undertaken or prescribed for different reasons such as sports performance,
235 general health, functional maintenance, recovery (e.g., following injury, illness/diseased states) and
236 also to compensate the effects of ageing. However, extreme mechanical loads are harmful to
237 tendons by bringing catabolic processes such as matrix degradation. Immobilization or disuse of
238 tendons also leads catabolic effects on it. Differently there are few data that examined the impact
239 that detraining may have on tendons. Thus, the present descriptive systematic literature review tried
240 to summarize the effects of discontinuing physical activity on tenocyte metabolism and/or in tendon
241 morphology in order to elucidate the mechanism behind these changes.

242 All examined studies, both preclinical and clinical, observed that discontinuing activity negatively
243 influence tendon structure and morphology, albeit with differences in the training and/or detraining
244 protocols, in the types of tendons, in subjects involved, in the study design or in the experimental
245 setting involved. The results of all these studies suggested that after a period of sudden detraining
246 (such as after an injury) physical activity should be restarted with caution and with appropriate
247 rehabilitation programs because cessation of activity causes modifications in tenocytes and tendons
248 metabolism, morphology, i.e. in collagen type I and III synthesis, collagen organization, cellularity,
249 vascularity, proteoglycan content, tear density, mechanical properties.

250 Notwithstanding the alterations highlighted in the reviewed articles after tendon detraining, some
251 limitations of the examined studies should be also considered. In fact, this systematic review has as
252 its main focus not only to bring together major works involving major changes in morphological
253 and structural properties of tendons during detraining, but also to examine the methodological
254 process on which the articles were based to assess the trustworthiness of the results found.

255 In relation to the results obtained in the *in vitro* study examined in this review (Salamanna, 2015),
256 that showed a decrease of tendon mitochondrial area, rough endoplasmic reticulum area, C-terminal
257 propeptide of type I collagen, fibronectin, aggrecan and tenascin-c synthesis and presence of
258 inflammatory cytokine production, we have to consider that tenocytes from animals subjected to
259 sudden detraining were studied. In addition, results were obtained in *in vitro* cultured cells, which
260 were not any longer structured into tissues, but in monolayer and static conditions. Thus, it is
261 probable that the performance of explanted tendon cells is not equal to the performance of tendon
262 cells in their native matrix environment *in vivo* (Fu, 2008; Leigh, 2008). However, these results
263 indicated that the tendon does not operate as a inert connector between muscles and bone, but
264 dynamically responds to mechanical loading.

265 The ~~two~~ **three** preclinical studies examined in this review employed a rat or **chicken** animal model
266 that may not be fully representative of human conditions but the invasive analyses conducted in
267 these studies permitted a depth investigation for the advancement of knowledge of many aspects on
268 tendon response to detraining (Frizziero, 2011 and 2015; **Foutz, 2007**). Moreover, looking at the
269 literature, rat and rodents are the most used animals when mechanical load with treadmill running is
270 used (Warden, 2009; Lui, 2011). In fact, the results of these *in vivo* studies demonstrated that the
271 adopted running protocol did not induce tendinopathy or other pathologic changes in hindlimbs.
272 Another methodological process that must be considered is that in these studies all morphometric
273 parameters were measured by 2D image analysis, while other investigation methods, such as micro-
274 MRI, may allow a more in-depth understanding of tendon structure. However, as for the reviewed
275 *in vitro* paper, these *in vivo* results provide interesting data for both sports medicine practitioners
276 and orthopedic surgeons, wishing to prevent the pathological or degenerative modification that
277 affect these structures.

278 Great variability was noted in the four clinical studies (Kubo, 2010 and 2012;McMahon, 2013;
279 Kannas, 2014) that analyzed the effects of detraining. In fact, these studies involved different
280 tendons (Achilles, gastrocnemius, patellar), different types of exercise (isometric knee extension ,
281 resistance training, plyometric training on incline and plane ground), different training and
282 detraining periods (3 and 4 months) and different types of analyses (Dual Energy X-Ray
283 Absorptiometry , ultrasonography, electromyography). Furthermore, it is important to point out that
284 the different effects of detraining on tendons depends not only on the above mentioned variables,
285 but also on the patient intrinsic characteristics, that are affected by age, gender, drug assumption,
286 the presence of systemic or genetic or endocrine diseases (i.e. obesity, diabetes, Cushing syndrome,
287 hypercholesterolemia, osteoporosis). In fact, recently it was shown that proliferation and synthetic
288 activity of tenocytes are negatively affected by aging and estrogen deficiency (Torricelli, 2013). In
289 addition, clinical studies did not permit a depth understanding of the alteration in tendon
290 metabolism and morphology (i.e. expression of type I collagen, fibronectin, aggrecan and tenascin-
291 c synthesis and/or presence of inflammatory cytokine, cellularity, vascularization, fibers
292 arrangements ect). However, despite these limitations these clinical studies indicate that tendons
293 may be susceptible to detraining. These findings could have a direct relevance to functional
294 rehabilitation practices showing that after a period of sudden detraining, physical activity should be
295 restarted with caution.

296 Despite the fact that the examined studies showed a potential negative effect of detraining on
297 tenocytes and tendons, there is a paucity of preclinical and clinical studies that examined the
298 importance that cessation of training may have on tendon. These results should be confirmed by

299 other preclinical and clinical research in order to completely comprehend the effect of detraining on
300 tendons. In particular, several aspects should be further studied and refined in order to improve our
301 understanding on the role of detraining in tenocytes and tendon mechanobiology: 1) standardization
302 of the training and detraining protocols in both preclinical and clinical research; 2) development of
303 systems that reproduce tendon detraining in culture with high reliability to native tendon; 3)
304 comprehend how tenocytes respond to detraining and how they mechano-regulate their response; 4)
305 evaluate the presence of altered tendon structure and/or morphology due to detraining in its various
306 stages; 5) evaluation of the role of other tissues (bone, muscle, nerve, vascularity, etc.) on tendon
307 mechanobiology during detraining. Finally an integrated, collaborative multi-disciplinary multiscale
308 approach is likely to yield the greatest advances in this field.

309

310

In review

311 **Table 1:**

Experimental set-up	Type of tendon	Control group	Training protocol	Detraining protocol	Analysis	Main results	Reference
<i>In vitro</i> model	Rat patellar tendon tenocyte	Untrained patellar tendon tenocyte Trained patellar tendon tenocyte	10 week on a treadmill (~65–70% VO ₂ max)	Caged without exercise for 4 weeks	Transmission-electronic-microscopy, C-terminal-propeptide of type I collagen, collagen III, fibronectin, aggrecan, tenascin-c, interleukin-1 β , matrix-metalloproteinase-1 and-3.	Altered tenocyte synthetic and metabolic activity.	Salamanna et al 2015
<i>In vivo</i> model	Chicken gastrocnemius tendon	No control group	3 week on a treadmill (speed of 0.22 m/s, for 5 min)	Controls or immobilized for 2 weeks.	Tendon midregion cross-sectional area and biomechanical properties	Gastrocnemius tendon responds to mechanical disuse as predicted by the mechanobiology process	Foutz et al. 2007
<i>In vivo</i> model	Rat patellar tendon	Untrained patellar tendon Trained patellar tendon	10 week on a treadmill (~60% VO ₂ max)	Caged without exercise for 4 weeks	Collagen fiber organization and proteoglycan content.	Low proteoglycan content and collagen fiber organization .	Frizziero et al 2011
<i>In vivo</i> model	Rat patellar tendon	Untrained patellar tendon Trained patellar	10 week on a treadmill (~65–70% VO ₂ max)	Caged without exercise for 4 weeks	Structure and morphology (modified Movin score, tear	Altered structure and morphology with the highest Movin score	Frizziero et al 2015

		tendon			density, collagen type I and III).	values, the highest percentage of collagen III and the lowest of collagen I	
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313 **Table 2:**

Type of tendon	Patients	Training protocol	Detraining protocol	Analysis	Main results	Reference
Patellar tendon	8 (training group); 6 (control group).	Unilateral isometric knee extension, 4 times/week, 3 months.	Return to usual levels of physical activity, 3 months.	- Tendon elongation by ultrasounds; - Cross-sectional areaby MRI.	Greater values of tendon elongation, decrease in tendon stiffness during detraining.	Kubo et al 2010
Achilles tendon	9 (training group); 7 (control group).	Unilateral (left side) isometric plantar flexion exercise, 4 times/week, 3 months.	Return to usual levels of physical activity, 3 months.	- Tendon elongation by ultrasounds; - Cross-sectional areaby MRI; - Blood supply and oxygen saturation; - Serum concentration of BAP and PIP by ELISA.	Tendon elongation increased and stiffness rapidly decreased after detraining.	Kubo et al 2012
Patellar tendon	10 (training with the MTC at a shortened position); 11 (MTC at a lengthened position); 11 (wide range of	Resistance training, three times per week, 8 weeks.	4 weeks of detraining	- Patella moment arm by DEXA; - Tendon elongation and stiffness by ultrasounds; - Circulating TGF- β 1 levels by ELISA.	No significant alterations in patella tendon dimensions or circulating TGF- β 1 levels following training or detraining in any of the groups.	McMahon 2013

	motion); 10 (control group).					
Achilles tendon	10 (training on inclined ground) 10 (training on plain ground)	Plyometric training	4 weeks of detraining	Aponeurosis strain of MG	Strain was decreased from 22.7% (± 0.05) to 16.3% (± 0.05) after detraining period.	Kannas 2014

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456

In review

Figure 1.TIF

