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Title: Mechanical Coupling Between Muscle-Tendon Units Reduces Peak Stresses

Year: 2018

Version:

Please cite the original version:

Maas, H., & Finni Juutinen, T. (2018). Mechanical Coupling Between Muscle-Tendon Units Reduces Peak Stresses. *Exercise and Sport Sciences Reviews*, 46(1), 26-33.
<https://doi.org/10.1249/JES.0000000000000132>

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1

2 **Mechanical coupling between muscle-tendon units reduces peak stresses**

3 Short title: Mechanism to reduce peak tissue stress

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23 **Disclosure of funding:** The research reviewed was partly supported by the Division for Earth and
24 Life Sciences of the Netherlands Organization for Scientific Research [864-10-011; to HM]. This
25 paper was partly written during a visit from TF to HM, which was supported by a visitor's travel grant
26 from the Netherlands Organization for Scientific Research [040.11.519] and University of Jyväskylä.

27 **Conflicts of interest:** None

28 **Abstract** (56 / 60 words)

29 The presence of mechanical linkages between synergistic muscles and their common tendons may
30 distribute forces among the involved structures. We review studies, using humans and other animals,
31 examining muscle and tendon interactions and discuss the hypothesis that connections between
32 muscle bellies and within tendons may serve as a mechanism to distribute forces and mitigate peak
33 stresses.

34 **Summary** (20 / 20 words)

35 We hypothesize that connective tissue linkages between muscle-tendon units serve as a mechanism to
36 distribute forces and mitigate peak stresses.

37 **Key Points** (125 / 150 words)

38 - Muscle fibers and whole muscles are embedded within a connective tissue network linking these
39 structures to surrounding structures.

40 - Mediated by these connective tissue linkages, skeletal muscles can mechanically interact with each
41 other, but the extent depends on multiple factors including muscle group, activation level and muscle
42 length. .

43 - Recent studies indicate that these linkages distribute muscle fiber forces without affecting the net
44 moments exerted at the joint level.

45 - Similarly to muscle, the connective tissue network around tendon fibers, fascicles and subtendons,
46 such as in the Achilles tendon, may serve as a mechanical linkage transmitting forces within tendon.

47 - Upon an injury the connective tissue network can provide an alternative pathway for muscular force
48 transmission and, thereby, limiting loss of muscle-tendon function.

49 **Key Words:** force transmission, skeletal muscle, connective tissue, Achilles tendon, displacement,
50 strain, injury

51

52 **INTRODUCTION**

53

54 Body or limb movements are initiated by the excitation of motor neurons in the spinal cord leading to
55 contraction of skeletal muscle fibers. The subsequent transmission of cross-bridge forces via the
56 tendons to the skeleton, creating a moment with respect to joints, is a key component in the mechanics
57 of animal movement. Within a muscle belly, many cytoskeletal proteins and collagen-reinforced
58 structures provide a medium for muscular force transmission. In other words, cross-bridge forces can
59 be distributed among many structures. Force can be transmitted to the tendon via sarcomeres in series
60 within muscle fibers and the myotendinous junction. The vast intramuscular connective tissue
61 network (i.e., endomysium, perimysium and epimysium) provides a medium through which forces
62 can also be transmitted laterally between neighboring muscle fibers. Furthermore, the connective
63 tissue network within a muscle is continuous with the connective tissue network of surrounding
64 muscles extending these effects beyond the muscle boundaries. Tendon fascicles and subtendons (i.e.,
65 tendon fascicles within a multi-muscle tendon, such as the Achilles tendon, originating from different
66 muscles) are also surrounded by a matrix, which can act to distribute forces.

67

68 In the first decade of this millennium, several studies using rats showed that, besides the origin and
69 insertion of a muscle-tendon unit, force produced by muscle fibers can be transmitted to the skeleton
70 via connective tissue linkages at their muscle belly surface (epimysium), named epimuscular
71 myofascial force transmission (1, 2). As a consequence, changes in the state (e.g., length, level of
72 activation) of one muscle can affect the force exerted at the tendon of a neighboring muscle. This
73 would imply that skeletal muscles do not act as independent functional units. Epimuscular myofascial
74 force transmission has been studied also in humans, but due to the limitations of non-invasive
75 kinematic measures and modeling approaches, this has yielded only indirect evidence. At this point,
76 the physiological significance of this phenomenon is still subject to debate. The unanswered question
77 is: Are intermuscular connections a mere by-product of the musculoskeletal organization or are they

78 functionally relevant? From a biological-evolutionary perspective, functional significance is expected
79 if this morphological feature is present within all mammalian species.

80

81 Besides the most recent findings on epimuscular myofascial force transmission, this article also
82 reviews studies investigating the role of lateral connections between tendon fascicles and subtendons
83 in transmitting force to the skeleton. While in vivo studies in humans have revealed nonuniform
84 anterior-posterior displacements within Achilles tendon, recent in vitro studies have shown that the
85 matrix between tendon fascicles has the capacity to bear substantial forces. These recent findings
86 suggest the possibility for force distribution not only between muscle bellies, but also within tendon.

87

88 Because lateral connections between different muscle-tendon units provide a medium for force and
89 stress distribution, we examine the hypothesis that connections between muscle bellies and within
90 tendons may serve as a mechanism to distribute forces and, consequently, to minimize peak stresses
91 (Fig. 1). In a case of muscle fiber or tendon injury, the connections provide an alternative pathway for
92 forces, thereby, limiting the effects of such injury on muscle-tendon function. If this mechanism is
93 active continuously, during the many motor tasks that we perform, it may also serve to prevent muscle
94 or tendon injuries. We expect that this mechanism is of greater importance in the presence of scar
95 tissue, for example after tendon transfers, because this enhances the connectivity between muscle-
96 tendon units (3, 4). In this review, we focus mainly on the triceps surae muscles and their shared
97 Achilles tendon (AT); not including the plantaris muscle. However, our analysis will apply also to
98 other muscle groups in which different muscles share a common tendon (e.g., the quadriceps).

99

100 **CONNECTIVE TISSUES PROVIDING PATHWAYS FOR FORCE DISTRIBUTION**

101

102 Several structures have been identified as pathways for epimuscular force transmission (1). At the
103 interface between muscle bellies, a layer of areolar connective tissue as well as a neurovascular tract
104 (a connective tissue structure embedding blood and lymph vessels as well as peripheral nerves) is

105 present. In addition, skeletal muscles are linked to connective tissue layers surrounding synergistic
106 muscle groups (e.g., compartmental fascia, interosseal membranes). All above mentioned structures
107 are continuous with the intramuscular connective tissue network. At the myotendinous junction, the
108 muscle fibers embedded in connective tissue are linked in an interdigitated fashion to the collagen
109 fibers eventually forming the tendon.

110

111 Tendon structural organization allows it to bear high tensile forces, but to a smaller extent also
112 transversal, rotational and shear forces as well as pressure and contusion. Hierarchy of tendon from
113 tropocollagen molecules to fascicles as well as the ground substance wherein the collagens are
114 embedded is well established. Tendon fascicles are surrounded by a matrix (the interfascicular matrix)
115 that allows these fascicles to move relative to each other for some range, but that can also bear
116 substantial forces beyond the initial stretching range. Interestingly, properties of the interfascicular
117 matrix can be different between tendons having different functions related to their capacity to store
118 energy and transmit forces (5).

119

120 **MECHANICAL INTERACTIONS BETWEEN MUSCLES – HUMAN STUDIES**

121 Human imaging studies have discussed the potential effects of epimuscular myofascial force
122 transmission based on observations of muscle motion during 1) passive joint movements, 2) selective
123 muscle stimulation or 3) voluntary contractions. One of the first human studies trying to elucidate
124 force transmission between adjacent muscles examined the relative displacement of medial
125 gastrocnemius (MG), soleus (SOL) and flexor hallucis longus muscle movements in all of the three
126 abovementioned conditions (6). During 80° passive knee extension, the authors demonstrated that the
127 neighboring distal aponeuroses of MG and SOL moved to a similar extent. This result showed that
128 also the one-joint SOL muscle deformed by knee movement, possibly involving epimuscular
129 myofascial force transmission.

130

131 When Tian et al. (7) studied the effects of 60° passive knee flexion on MG and SOL fascicle lengths,
132 they found that MG fascicles shortened while the length of proximally and distally located SOL
133 fascicles increased slightly. Using a similar experimental approach imposing 90° knee flexion, Finni
134 et al. (8) reported lengthening of only the distal SOL fascicles while the length of proximally located
135 muscle fascicles remained rather constant. These studies provide support to our hypothesis by
136 suggesting that knee movement causes deformations of SOL muscle due to force transmission from
137 the two-joint gastrocnemius muscles (GAS) via connective tissue linkages. However, the effects may
138 be location specific due to the more abundant presence of the connective tissue linkages distally. Note
139 that there are earlier reports showing no effects of passive knee joint flexion on proximally located
140 SOL fascicle lengths in humans (e.g., 9).

141
142 While effects of knee movement on SOL muscle have been repeatedly observed, estimates of the
143 magnitude of such force transmission indicated very small effects on ankle joint moment (7).
144 Assuming that SOL slack angle (i.e. angle at which the passive muscle bears no tension) occurs at 30°
145 plantarflexion, Tian et al. (7) calculated that the contribution of SOL to the ankle joint moment could
146 be as high as »12% of that of GAS force. However, they also reported that the force estimate was
147 sensitive to the selected slack length. As recently SOL slack length was reported to occur at 2°
148 dorsiflexed ankle position (10), the magnitude of intermuscular force transmission in passive
149 condition is probably even smaller than estimated by Tian et al. (7). This suggests that for the
150 substantial deformations within SOL only small forces were needed. Note, however, that in this study
151 the muscles were not activated. Interestingly, both MG and lateral gastrocnemius (LG) muscles start
152 to bear tension at a more plantar flexed ankle position than SOL (10) with possible proximal-distal
153 differences in muscle fascicle slack lengths along a single muscle (11). Overall, the observations of
154 heterogeneous slack lengths may be relevant when estimating the mechanical effects of relative tissue
155 displacements of synergist muscles at different joint angles.

156

157 Regarding the effects of knee joint angle on SOL muscle deformations, it should also be noted that
158 based on rat studies the nerve tract that also crosses the knee joint may contribute to force
159 transmission (1, 2). In humans, knee extension has been reported to result in proximal displacement of
160 the sciatic nerve (12). Such movement may then cause a load on SOL muscle and deform it. In a
161 recent rat study involving only passive muscle conditions, it was found that changes in knee angle did
162 not affect fiber length of tibialis anterior muscle (crossing only the ankle joint) but did result in
163 changes in the distribution of lengths of sarcomeres in series within muscle fibers (13). Because the
164 direction of sarcomere length changes could not be explained by effects of length changes of the
165 synergistic extensor digitorum longus muscle, the results were explained by changes in loads exerted
166 on tibialis anterior via the nerve tract. Note, however, that no effects of knee movement on
167 distribution of sarcomere lengths were found for SOL muscle (13). Considering that sarcomere
168 lengths can be affected without changes in fiber length, measurements of sarcomere lengths in
169 humans using recently developed microendoscopy techniques (14) seem warranted when
170 investigating force producing capacity and its distribution.

171

172 While the magnitude of force transmission may be small between passive muscles less is known about
173 activity dependent stiffening of connective tissues and whether it may increase the magnitude of
174 intermuscular force transmission. It was recently proposed that passive conditions are more likely to
175 involve large deformations of muscle tissues, because of the low stiffness with respect to the
176 intermuscular connections, while active conditions will involve less deformations of muscle tissues,
177 but more lengthening of myofascial connections (4). Accordingly, greater intermuscular force
178 transmission can be expected during active muscle conditions. Previous studies have reported that
179 connective tissues, the aponeurosis in particular, are stiffer during muscle contraction than in passive
180 conditions (e.g., 15). Also the extent of mechanical interaction between rat triceps surae muscles was
181 found to increase from fully passive state to all muscles being maximally activated (16).

182

183 A recent human study examined the effects of activation on relative displacement between SOL and
184 LG muscles (8). During knee extension (i.e., GAS muscle stretch) with LG selectively stimulated,
185 relative displacement between LG and SOL muscles was smaller in active as compared to passive
186 condition, indicating that muscle activation reduced movement independency of the muscles. It
187 should be acknowledged that in this experiment only low activation levels were used to ensure
188 selectivity of the stimulation and the phenomenon was statistically significant only for the shortest
189 muscle-tendon unit length of GAS (8). It may be that the short muscle length (near slack length)
190 provided a condition where even a small level of activation could induce effects while at longer
191 lengths greater forces would be required to show the effect. The above results indicate a likely effect
192 of muscle length and activation level on epimuscular myofascial force transmission.

193
194 In active conditions, the level and type of activation may affect the tissue deformations. For example,
195 supramaximal electrical stimulation of the tibial nerve induced similar length and velocity effects in
196 both SOL and MG fascicles (17), whereas under voluntary control SOL and MG may be activated
197 differently (18) and move differently (19). The observations of relative muscle motions may also be
198 task dependent (i.e., isometric vs. dynamic) or vary between individuals as shown for relative
199 movement between SOL and MG (19) and vastus lateralis and rectus femoris muscles (20).

200
201 It is important to note that the above described concurrent muscle deformations between adjacent
202 muscles do not necessarily refer to mechanical interaction. To confirm intermuscular force
203 transmission in humans, future studies need to complement the kinematic measures with modeling or
204 advanced methods such as ultrasound shear wave elastography that may provide information about
205 the forces involved. Another noteworthy issue is that some human studies report fascicle lengths (7-9,
206 17) while others present results on relative tissue displacements (6, 8). These two measures may
207 reflect slightly different phenomena: the latter on a more global and functional level and the former on
208 a local level. Note that, due to effects of muscle architecture, changes in muscle belly length are more
209 directly related to changes in tendon length and, hence, tendon forces than changes in muscle fiber

210 length. Therefore, measurements at different levels of the musculoskeletal system are needed in future
211 studies for a complete understanding of the mechanical implications of local muscle deformations.

212

213 In the next section, we will describe in situ studies using rats and cats in which the mechanical
214 consequences have been measured at the single muscle or joint level.

215

216 **MECHANICAL INTERACTIONS BETWEEN MUSCLES – STUDIES ON OTHER** 217 **ANIMALS**

218

219 As described above, there is ample evidence for connective tissue linkages between adjacent muscle
220 bellies that are sufficiently strong to transmit force. According to our hypothesis, this epimuscular
221 myofascial pathway may also be involved in the distribution of forces among synergists. However,
222 earlier studies involved muscle conditions that were beyond physiological, e.g. maximal excitation of
223 all synergistic (and sometimes also antagonistic) muscles and higher changes in muscle-tendon unit
224 length and relative position than possible in vivo (reviewed in 1, 2). In some cases, the myofascial
225 effects of the physiological conditions within those experiments were tested separately and found to
226 be significant but small (2). More recently, mechanical interactions between synergistic muscles for
227 conditions found during normal movement have been studied using two experimental approaches: (i)
228 sever the tendon of origin and/or insertion to connect them to force transducers, and imposing muscle
229 lengths mimicking those found in vivo – note that all other structures crossing the joints (e.g., other
230 muscles, neurovascular tracts, ligaments) are not manipulated (Fig. 2, left); (ii) testing muscles in a
231 nearly intact limb, leaving the tendons attached to the skeleton and changing muscle-tendon unit
232 length by joint movements (Fig. 2, right).

233

234 Using the first approach, mechanical interactions between passive and active ankle plantar flexors of
235 the rat were found (3, 16). Intermuscular interaction was assessed by measuring effects of lengthening
236 the two-joint muscles (i.e. stretching LG and plantaris proximally by 6 mm, simulating knee extension

237 from 45° to 130°) on force exerted at the distal SOL tendon which was kept at a constant length, and
238 on the force difference between the proximal and distal tendons of the two-joint muscles. These two
239 measures were shown to correlate positively (3), indicating that forces are transmitted between these
240 muscles. The extent of interaction was affected substantially by the level of muscle activation. The
241 increase of SOL force varied between 0.005 N for the condition in which all muscles were passive to
242 0.15 N for the condition in which all muscles were maximally activated. For reference, the optimal
243 force of SOL in these rats equals approximately 1.5 N. In conclusion, force transmission between
244 SOL and two-joint synergists may occur during normal joint movements, but only to a limited extent.
245 These findings support our hypothesis that some distribution of force among the ankle plantar flexors
246 can occur.

247

248 The second experimental approach exploits the presence of both one-joint and two- or multiple-joint
249 muscles within synergistic muscle groups. During isolated movements of the joint that is not spanned
250 by the one-joint muscle, only the length and relative position of neighboring two-joint muscle(s) will
251 be affected. As forces exerted at the tendons cannot be measured directly without causing tissue
252 damage, the joint moment the muscle exerts on contraction is assessed using a six degrees-of-freedom
253 load cell (see Fig. 2). Applying this approach in rat and cat studies revealed no effects of knee angle
254 and, hence, length changes of passive two-joint ankle plantar flexors, on plantar flexion moment and
255 rate of moment relaxation (the latter being a measure affected by muscle fiber length) of contracting
256 SOL muscle (21). Because MG, LG and SOL merge into the AT they have similar lines of action and
257 directions of 3D moment vectors (22), any effects of epimuscular myofascial force transmission will
258 be difficult to detect. In other words, the lack changes in SOL moment in response to changes in knee
259 angle cannot confirm the absence of epimuscular myofascial force transmission. These results indicate
260 that any connections between SOL and GAS have no mechanical effects at the joint level, but this
261 does not exclude the distribution of SOL forces to surrounding muscles or subtendons.

262

263 The same experimental approach was applied also to study mechanical interaction between one-joint
264 tibialis anterior (TA) and extensor hallucis longus (EHL) muscles and the multiple-joint extensor
265 digitorum longus (EDL) muscle (23). These muscles do not share a distal tendon and, hence, exert
266 moments in different directions (i.e., TA – ankle inversion, EDL – ankle eversion, both muscles –
267 ankle dorsiflexion). Thus for this muscle pair, intermuscular mechanical interaction should lead to
268 changes in the direction of the 3D moment vector. Instead, no effects of knee extension (decreasing
269 EDL length) on the moment exerted by TA muscle were observed (23). Therefore, these results must
270 be considered as evidence against the hypothesis of the present paper. This could be related to the
271 different functions of the ankle plantar and dorsal flexors, requiring more mechanical independence
272 within the latter muscle group.

273

274 **INTERACTIONS BETWEEN MUSCLES VIA A COMMON TENDON**

275

276 As described above, intermuscular interactions can be brought about by epimuscular myofascial
277 linkages at the interface between muscle bellies. If the tendons of several muscles merge into a single
278 tendon (e.g. the triceps surae with the Achilles tendon) and this tendon acts as a common spring, this
279 may provide another pathway for such interactions. An experimental approach to investigate if a
280 tendon acts as a common spring for two or more muscles is to assess muscle moment summation (24).
281 The active isometric joint moment (i.e., the total moment minus the passive moment) exerted by two
282 muscles simultaneously is compared to the sum of active moments exerted by each muscle
283 individually. The difference is then referred to as nonlinear moment summation, which is expected to
284 be negative. If the tendon acts as a common spring, its length change will be higher during
285 simultaneous activation than during individual activation due to greater force imposed on it. As the
286 muscle-tendon unit length is kept constant during an isometric contraction, this means a lower muscle
287 fiber length. Consequently, each muscle that is operating below its optimum length will produce
288 lower forces during simultaneous muscle activation. Hence, the sum of moments exerted by each
289 muscle individually will be higher than the moment exerted during simultaneous activation of the two

290 muscles. Experiments on cat vastus medialis and rectus femoris revealed nonlinear summation of -1%
291 (24). Similar results were found for cat and rat triceps surae (21, 22, 24). These results indicate that
292 nonlinearities are rather small, suggesting that for tested conditions the patellar and Achilles tendons
293 do not appear to act as common springs and thus the subtendons are rather independent of each other..
294 However, these findings do fully not exclude the possibility that the forces are distributed within the
295 AT. Future studies are needed to confirm the level of independence between subtendons of shared
296 tendons.

297

298 Another approach to study if a shared tendon can cause interactions between muscles is to manipulate
299 the length of only one muscle and, thereby, affect the part of the tendon that acts as a common spring.
300 For the triceps surae in humans, it has been found that passive knee extension (i.e. lengthening GAS
301 muscle) causes shortening of SOL muscle fascicles (7, 8). This may be the result of mechanical
302 interactions at the muscle belly interface, but could very well be mediated by the Achilles tendon
303 acting as a common spring. Knee extension will increase the muscle-tendon unit length of GAS
304 muscle, including its distal tendon. This may release tension on the SOL muscle fascicles, which may
305 then shorten. While knee extension in rats was shown to affect the length of the AT only when all
306 muscles were passive (21), in humans passive knee extension was accompanied with very minimal
307 distal tendon movement (1 mm as measured from distal SOL fascicle attachment, 6). Overall, more
308 studies on AT displacements are needed to understand if method and location of measurement (i.e.,
309 tracking the tendon itself vs. displacement of SOL, MG or LG fascicle insertion) provide
310 complementary observations especially in human studies.

311

312 In rats, during SOL activation or during SOL and GAS activation AT length was independent of knee
313 angle for most of the knee angles tested (i.e., between 60° and 130°)(22), which indicates a higher
314 stiffness of the AT in active muscle conditions. In agreement with this, no effects of knee angle on
315 active muscle moment summation were found (21). In a human study, low level MG stimulation in
316 flexed and extended knee positions caused smaller movements in distal SOL than those found during

317 passive knee movements (6). In particular, the movement of the distal SOL fascicle attachment to AT
318 was minimal (<1 mm), suggesting very small if any effects via the common tendon in passive and low
319 level active conditions.

320

321 Based on the above described studies, we conclude that, despite existing connections between
322 neighboring muscles and within common tendons, their effects on muscle moments exerted at the
323 joint are minimal. While passive and active muscle conditions during normal physiological conditions
324 may slightly differ from those applied experimentally, the significance of the force transmission
325 pathways may have an alternative function: that of distributing forces to reduce peak stresses. This
326 may occur not only at the muscle belly level but also within the tendon, which is discussed in the next
327 section.

328

329 **FORCE TRANSMISSION WITHIN TENDON**

330

331 In agreement with our hypothesis, we expect that there is potential for lateral force transmission
332 within tendons. This phenomenon may occur at different organizational levels of tendon and vary
333 between tendons. At a microscopic level, tendon matrix (5), cross-linking and/or fiber cross-sectional
334 area (25) can contribute to differences within (and between) tendons, while at a macroscopic level,
335 various anatomical designs can contribute to tendon specificity. For example, the patellar tendon has
336 straight fascicles of different lengths, while the AT has a twisted structure that is shared by three
337 muscles (26). Similarly to the triceps surae muscles, the quadriceps femoris and triceps brachii
338 muscles share the tendon of insertion, while the semitendinous muscle and the long head of biceps
339 femoris muscle share the tendon of origin. In such common tendons or in tendons with uneven
340 fascicle lengths, nonuniform displacements and/or strains seem very likely.

341

342 In addition to the macroscopic design of tendons, regional differences in tendon material may
343 contribute to the nonuniform behavior within tendons. In case of the patellar tendon, anterior and

344 posterior fascicles have different material properties, cross-link concentrations and fibril density (25).
345 Nonuniform behavior may also be facilitated by relative sliding between tendon fascicles that is
346 enabled by lubricin in the interfascicular matrix. This matrix is also rich in elastin that may act to store
347 energy and/or to transmit forces between the fascicles. Mechanical tests examining the properties of
348 the interfascicular matrix in equine tendons have shown that it is elastic and can bear substantial
349 forces; it has a stiffness of about half of that of tendon fascicles (5, 27). Preliminary data from a more
350 macroscopic level indicate that the matrix between the Achilles subtendons in rat has similar
351 mechanical properties as those described for the intrafascicular matrix (unpublished observations,
352 Maas & Screen) and, thus, there is potential for force transmission and distribution of stresses at
353 different organizational levels of a tendon.

354
355 The structural organization and mechanical properties described above can allow nonuniform
356 deformations within tendons. In cadavers, for example, differences in strain between anterior and
357 posterior patellar tendon and between different parts of AT can be found. Using novel *in vivo* imaging
358 methods (e.g., 28, 29), differential displacements between anterior and posterior parts have been
359 reported to exist in human patellar tendon during isometric contractions and in AT during passive
360 joint rotations, isometric contractions, eccentric contractions as well as during walking. Within the AT
361 in particular, these differential displacements may be possible due to the presence of distinct
362 subtendons for SOL, LG and MG muscles (26), but with the applied imaging methods it is not
363 possible to identify the specific anatomic locations of the displacements in humans. New evidence
364 from *in vivo* measurements in the rat indicates differential lengthening of the distal tendons of SOL
365 and LG muscles during controlled muscle stimulations (30) and during locomotion (31). This suggests
366 relative displacements between Achilles subtendons, which could, given the material properties of the
367 intersubtendon matrix, facilitate intersubtendon force transmission. The Achilles subtendons can be
368 dissected free, but as the connections between subtendons become tighter towards the insertion onto
369 the calcaneus, this is more difficult distally. Consequently, there may be proximal-distal variation in
370 the matrix between subtendons requiring comprehensive future experiments along the length of the

371 muscle-tendon unit. Note that the proximal-distal location also at the muscle belly level can be a
372 factor when evaluating the magnitude of mechanical interaction between the synergists (1, 2).

373

374

375 In addition to nonuniform deformations, there is also evidence of nonuniform loading within the AT
376 in humans *in vivo*. In experiments where either an optic fiber force transducer (32) or a syringe needle
377 (26) was inserted in the AT, sharp bending of the optic fiber or the needle was observed during high
378 loading conditions, such as drop jumps or maximal voluntary isometric contractions. This was most
379 likely caused by nonuniform deformations and/or forces within the tendon which, in turn, can be due
380 to differential activation of the synergists (18). In such a case the mechanical linkages between
381 muscle-tendon units can help to distribute forces and mitigate uneven AT stress (Fig. 1). For further
382 insight of asymmetrical loading of AT the reader is referred to a recent review by Bojsen-Moller &
383 Magnusson (26).

384

385 The above reviewed research supports the possibility for stress distribution within AT, in agreement
386 with our hypothesis. Relative displacement between tendon fascicles or subtendons may provoke
387 lateral force transmission via interfascicular matrix. Alternatively, such relative displacements may
388 indicate that the different triceps surae muscles transmit their forces independently. However, little is
389 known about the mechanical properties of the interfascicular and intersubtendon matrices and, thus,
390 no comprehensive conclusions about the mechanical consequences of nonuniform behavior can be
391 drawn.

392

393 **STRESS DISTRIBUTION FOLLOWING MUSCLE-TENDON INJURY**

394

395 Upon injuring muscle fibers and/or tendon fascicles, the connective tissue network may provide an
396 alternative pathway (a safety net) for force transmission and, thereby, limiting the acute loss of
397 muscle-tendon function. For animals other than humans, such a mechanism may mean the difference

398 between escaping from or being caught by its predator. Evidence of such acute redistribution of
399 muscle force in response to an injury has been derived from experiments using experimental
400 approaches similar to those presented in Figure 2 (2). In one of such experiments in rats,
401 simultaneously measuring forces exerted at distal tendons of SOL and distal and proximal LG and
402 plantaris (LG+PL) muscles, LG muscle belly was unexpectedly torn. This resulted in the following
403 events: (i) substantial LG muscle belly shortening, (ii) an immediate decrease of force exerted at the
404 tendons of LG+PL, and (iii) an immediate increase of force exerted at the distal tendon of SOL (Fig.
405 3). These results indicate that some of LG muscle fibers still exerted force and, thus, its muscle fibers
406 did not shorten to their active slack length (i.e., the length at which active force approaches zero). The
407 increase in force exerted at the distal tendon of SOL can be explained by the transmission of LG
408 muscle fiber forces via connective tissue linkages to SOL. In other words, forces produced by muscle
409 fibers in the injured muscle are redistributed to surrounding muscles. If the subtendons of a common
410 AT are separated, as in the experiment yielding the data shown in Fig. 3, such redistribution can only
411 be mediated by epimuscular myofascial pathways providing evidence for our hypothesis. In case of an
412 intact common tendon, force redistribution may occur also within the tendon given that its
413 interfascicular matrix has a notable stiffness (5).

414

415 It is worth mentioning, that our hypothesis implicitly assumes that distribution of forces leads to lower
416 stresses within the muscle-tendon unit, however, it may be that greater stress concentrations could be
417 formed if forces are redistributed from thicker to thinner structures. Clearly, future research at
418 different levels of muscle-tendon is warranted to clarify the phenomenon and its mechanisms.

419

420 Early studies examining the effects of rectus femoris tendon transfer showed that although the surgery
421 was supposed to make the rectus femoris muscle a knee flexor based on its origin and insertion, a
422 knee extensor moment was found during muscle stimulation (33). Recent evidence shows increased
423 importance of epimuscular pathways in the presence of scar tissue between muscle bellies (3, 4).
424 Based on these recent studies, it is very likely that the same mechanism was responsible for the

425 preservation of knee extension moment following rectus femoris tendon transfer to the flexor site of
426 the knee. This suggests an important role of scar tissue in stress distribution. It should be noted that
427 scar tissue is found also following muscle-tendon injuries, having the potential to modify force
428 transmission within the system. Overall, connective tissue mediated force distribution may become
429 more significant in pathological conditions of our musculoskeletal system.

430

431 When an injury causes damage to tendon fascicles their force transmission capacity is reduced, which
432 has been studied also using fatiguing situations. In tendon, the interfascicular matrix endures only one
433 third of the loading cycles (when loaded with 30% load) as compared to the fascicles (27). In the
434 course of a fatiguing trial, the stiffness of tendon fascicles and interfascicular matrix is reduced and
435 hysteresis shows a tendency to decrease until final increase before failure (27). Similarly to the
436 muscle injury described above and in Fig 3, in tendon injury the presence of epimuscular myofascial
437 linkages may provide alternative pathways for muscle forces to be effective at joint level. These
438 alternative pathways may also be important in the prevention of tendon injuries. Distributing forces
439 among a greater cross-sectional area will reduce peak stresses and, thereby, diminish the risk of tissue
440 overload. Even if the magnitude of this mechanism is limited, its role may be crucial during the many
441 physical activities that we perform in daily life.

442

443 Besides injury, future studies need to investigate the importance of force distribution in aging because
444 of changes in connective tissue properties. For example, in aged equine energy storing tendons, less
445 sliding between the fascicles has been found (34). This result corroborates to findings in human AT
446 during walking where the shearing between anterior and posterior tendon is reduced to nearly half in
447 the elderly. This reduction in the interaction within the tendon can have negative consequences to
448 SOL and GAS muscle function that are more tightly coupled to a common tendon in elderly (35).

449

450 **CONCLUSIONS**

451

452 Synergistic muscles are coordinated by the central nervous system to perform the versatile physical
453 tasks that we perform in daily life. This involves non-simultaneous activity, at various magnitudes
454 and, thus, different forces exerted by their muscle fibers to produce the required joint moments for
455 interaction of our body with the environment. As a consequence, the structures bearing these forces
456 (e.g., intramuscular connective tissues, tendon) are loaded non-uniformly, potentially leading to high
457 stresses locally. Recent evidence described in this review provides support for a mechanism that
458 redistributes muscle fiber forces and, thereby, minimizes peak stresses. This mechanism is
459 characterized by force transmission via tissues linking muscle-tendon units within a synergistic group
460 but its magnitude may vary between species, muscle groups, proximal-distal location, activity levels
461 and muscle lengths. Due to the fact that this mechanism is active continuously, we propose that it may
462 be involved in preventing muscle or tendon injuries. In case of an acute muscle-tendon injury, the
463 connective tissue network may act as a safety net, limiting the acute loss of muscle-tendon function.
464 In the long-term, enhanced use of stress distribution by means of scar tissue formation (i.e. an
465 alternative route for transmission of forces) will allow recovery of our musculoskeletal system while
466 making use of the maximal available capacity of the intact muscle tissues.

467

468

469 **Acknowledgements**

470 The authors wish to thank Guus Baan for his 3D drawings of the experimental setups and Michel
471 Bernabei for using some unpublished data.

472

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588

589 **Figure legends**

590 **Figure 1. Schematic illustration of the hypothesis.**

591 Simplified sagittal view of soleus (SOL) and gastrocnemius (GAS) muscles with distal Achilles
592 tendon cross-sectional viewed superiorly.

593 Left: If the muscle-tendon units of the triceps surae are mechanically independent from each other, all
594 force produced by the muscle fibers will be transmitted exclusively via the respective subtendon
595 within the Achilles tendon. No stress distribution will occur, the colors indicating different stresses
596 between the muscle-tendon units.

597 Right: If the muscle-tendon units of the triceps surae are mechanically linked to each other, either at
598 their shared muscle belly interface or within the Achilles tendon, force produced by the muscle fibers
599 can be distributed among all units. Stress will be distributed leading to a reduction of peak stresses. It
600 is important to note that in normal intact conditions the variable neural input affects forces also.
601 Typically, synergistic muscles are activated in synergy but not always and not to the same extent.

602

603 **Figure 2. Experimental setups for experimental approaches to study mechanical interactions**
604 **between synergistic muscles for conditions found during normal movement in a fully**
605 **anesthetized rat.**

606 Left: Proximal and distal tendons of LG and plantaris as well as the distal tendon of SOL were
607 connected via metal rods to separate force transducers (for details see 4). A bipolar cuff electrode was
608 placed on the tibial nerve to maximally activate all muscles.

609 Right: The femur was fixed and the foot was attached to a 6 degrees-of-freedom load cell using
610 custom-made clamps (21, 23). The ankle and knee joints were aligned with the set-up's rotational
611 axes. The knee joint angle was changed while keeping the ankle angle constant (two positions of the
612 knee joint are shown).

613

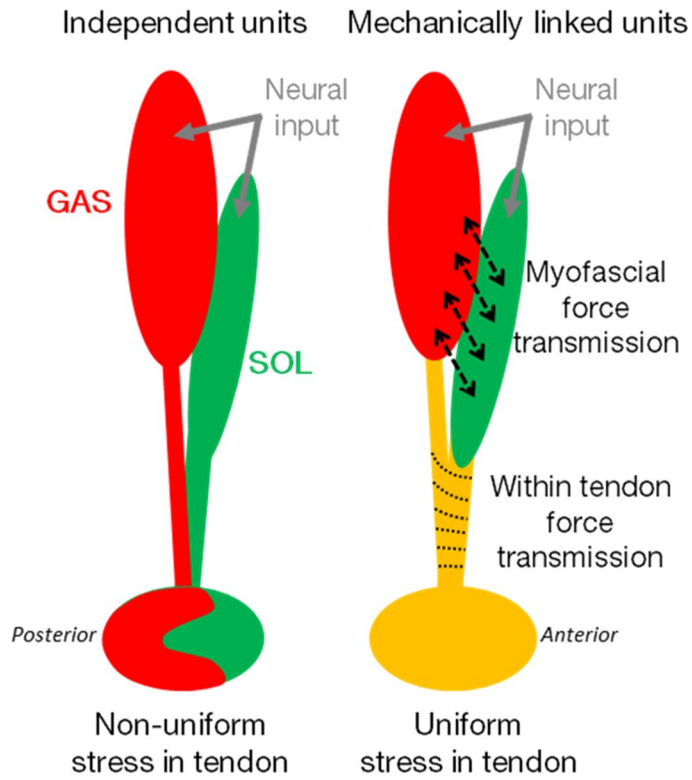
614 **Figure 3. Force distribution among synergistic muscles upon muscle rupture.**

615 Force recordings from distal tendons of soleus (SOL) and distal and proximal lateral gastrocnemius
616 and plantaris (LG+PL) muscles in a setup illustrated in Figure 2 (left). During an isometric
617 contraction induced by stimulation of the tibial nerve, LG muscle belly was unexpectedly torn (just
618 before the 2200 ms time stamp). Force exerted at the distal tendons of LG+PL decreased immediately
619 upon tear (vertical dashed line). Simultaneously, force exerted at the distal tendon of SOL increased
620 (see inset with enlarged view on SOL force signal). This indicates that some of LG muscle fibers still
621 exerted force, which was (at least partly) redistributed via connective tissue linkages to the distal
622 tendon of SOL (unpublished observations Bernabei & Maas).

623

624 **Figure 1**

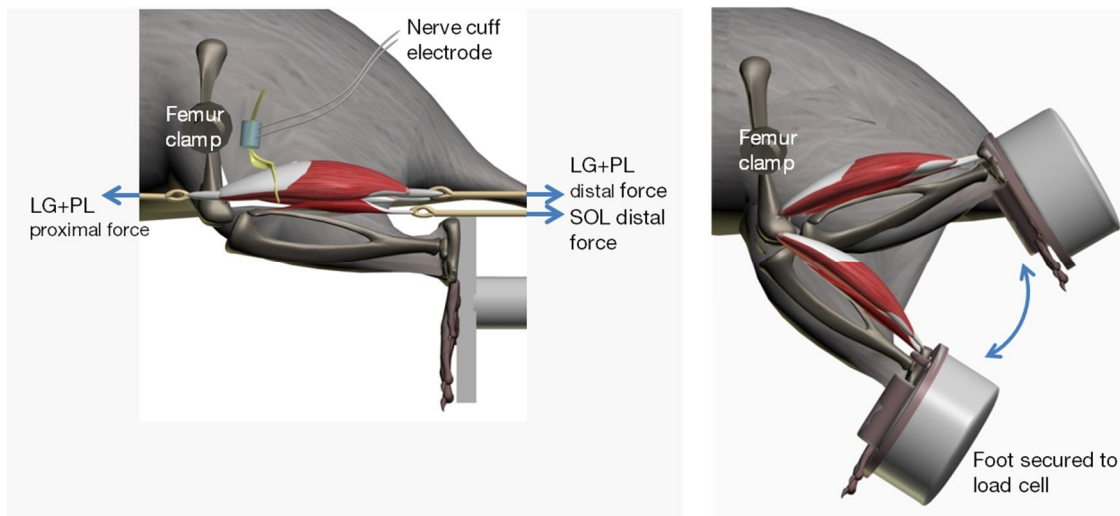
MECHANISM TO REDUCE PEAK TISSUE STRESS



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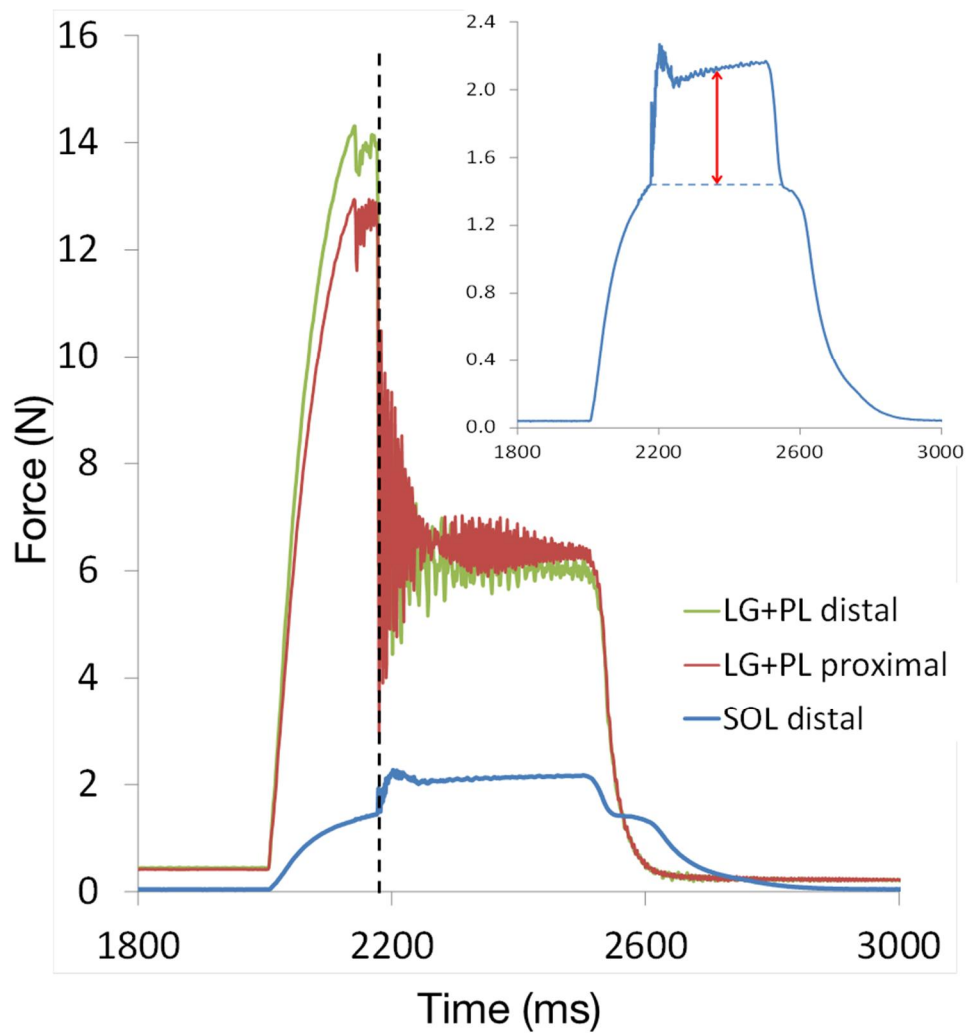
627 **Figure 2**



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629

630 **Figure 3**



631