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# Mechanical coupling between muscle-tendon units reduces peak stresses

Short title: Mechanism to reduce peak tissue stress

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**Abstract** (56 / 60 words)

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

**Summary** (20 / 20 words)

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

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

- Muscle fibers and whole muscles are embedded within a connective tissue network linking these structures to surrounding structures.
- Mediated by these connective tissue linkages, skeletal muscles can mechanically interact with each other, but the extent depends on multiple factors including muscle group, activation level and muscle length. .
- Recent studies indicate that these linkages distribute muscle fiber forces without affecting the net moments exerted at the joint level.
- Similarly to muscle, the connective tissue network around tendon fibers, fascicles and subtendons, such as in the Achilles tendon, may serve as a mechanical linkage transmitting forces within tendon.
- Upon an injury the connective tissue network can provide an alternative pathway for muscular force transmission and, thereby, limiting loss of muscle-tendon function.

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

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## INTRODUCTION

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

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

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

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

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

## CONNECTIVE TISSUES PROVIDING PATHWAYS FOR FORCE DISTRIBUTION

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

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

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

## MECHANICAL INTERACTIONS BETWEEN MUSCLES – HUMAN STUDIES

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

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

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

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

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

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

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

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

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

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

## MECHANICAL INTERACTIONS BETWEEN MUSCLES – STUDIES ON OTHER ANIMALS

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

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

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

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

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

## INTERACTIONS BETWEEN MUSCLES VIA A COMMON TENDON

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

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

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

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

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

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

## FORCE TRANSMISSION WITHIN TENDON

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

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

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

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

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

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

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

## STRESS DISTRIBUTION FOLLOWING MUSCLE-TENDON INJURY

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

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

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

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

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

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

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

## CONCLUSIONS

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

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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

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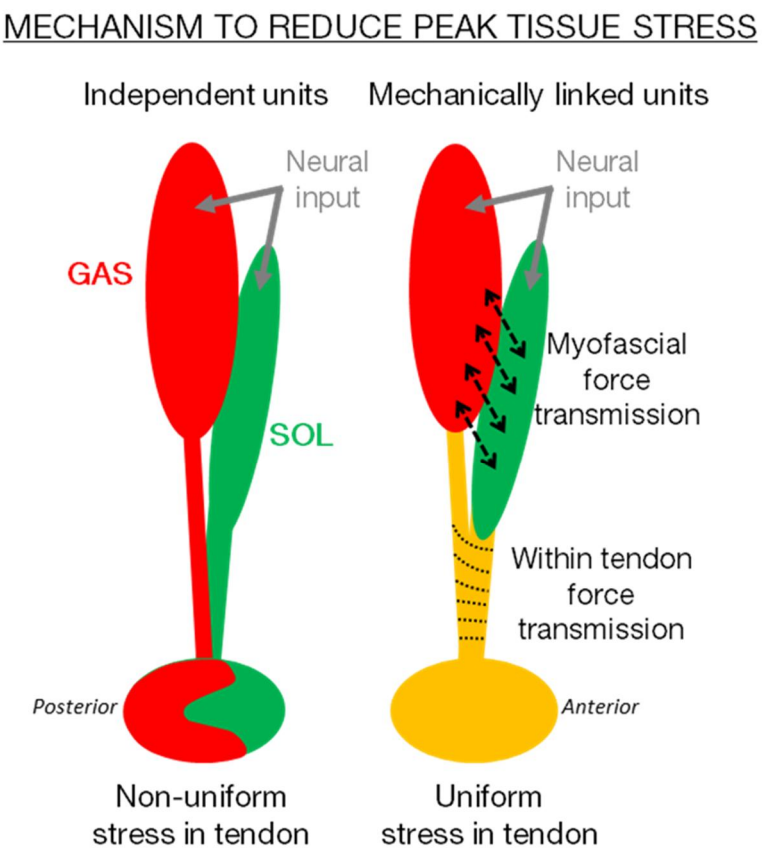
624 **Figure 1**

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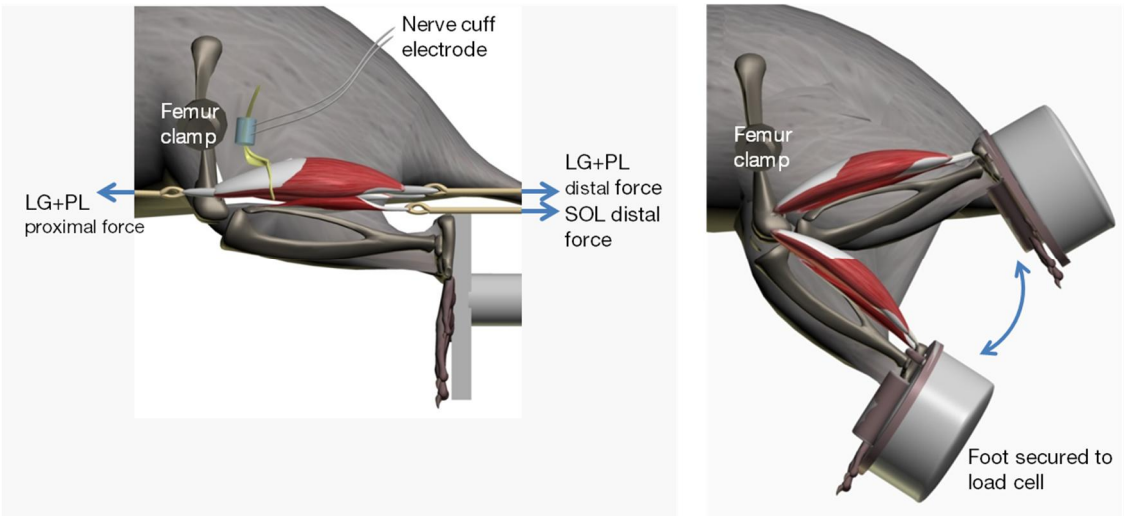
627 **Figure 2**

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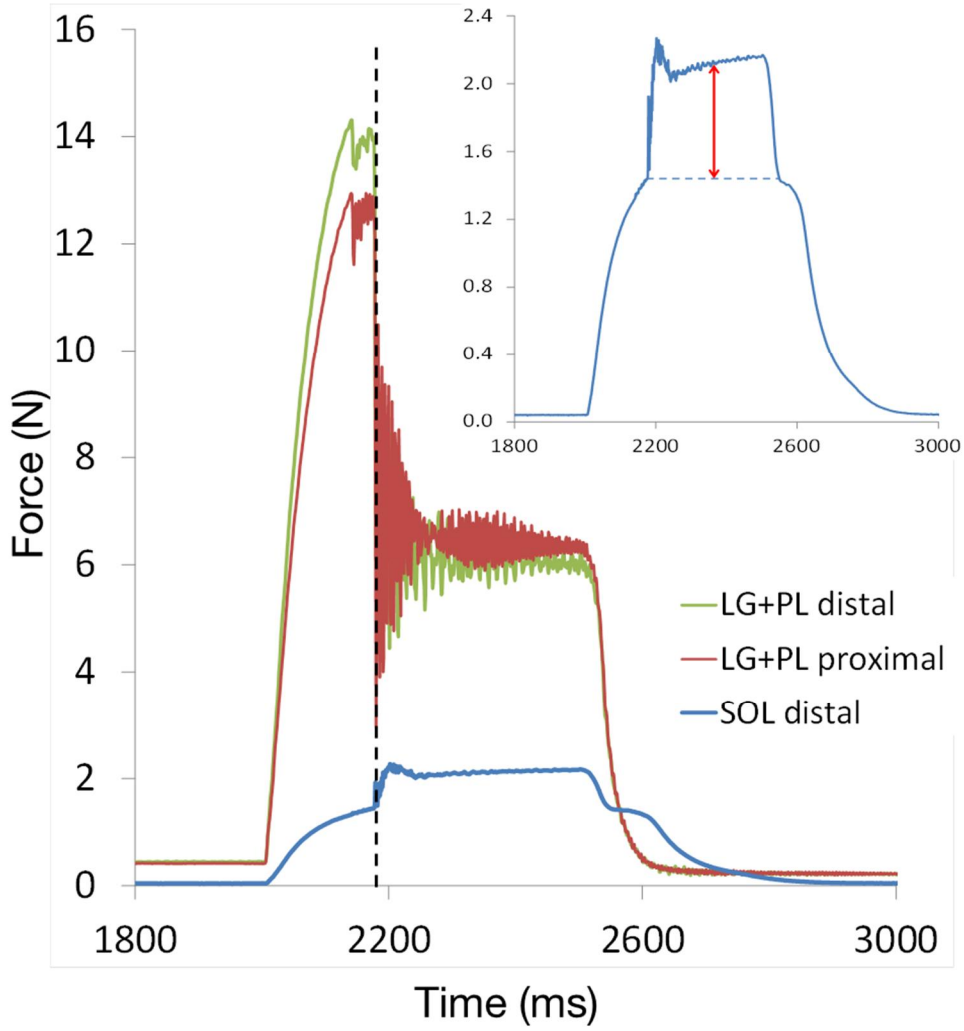
630 **Figure 3**

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