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Review Article

Hylonome

Disproportionate changes in skeletal muscle strength and size with resistance training and ageing

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Abstract

The ability of a muscle to shorten and produce force is crucial for locomotion, posture, balance and respiration. During a contraction, myosin heads on the myosin filament propel the actin filament via ATP hydrolysis, resulting in shortening of the muscle and/or force generation. The maximal shortening velocity of a muscle fibre is largely determined by the myosin ATPase activity, while maximal force is primarily determined by the cross-sectional area. Since most muscles are pennate rather than parallel-fibred and work at different lever ratios, muscle architecture and joint-tendon anatomy has to be taken into account to obtain the force and velocity characteristics of a muscle. Additionally, the recruitment of agonistic and antagonistic muscles will contribute to the torque generated during a contraction. Finally, tendon compliance may impact on the rate of force rise and force generated if it is such that the muscle contraction proceeds in the ascending limb of the length-tension relation. Even when magnetic resonance imaging and ultrasound, combined with EMG and/or electrical stimulation, have been applied to relate changes in muscle contractile properties to alterations in muscle size and architecture during ageing and resistance training, a disproportionate change in muscle strength and size remains to be explained.

Keywords: Muscle Architecture, Strength Training, Ageing, Tendon, Specific tension

Introduction

Skeletal muscle plays an important role in locomotion, posture, balance and respiration. The ability of the muscle to shorten and produce force is crucial for these functions. This ability alone, however, is not enough for daily living. Indeed, the force and velocity of a contraction has to be tightly controlled to enable both accurate, minute movements with a minimum of force, such as threading a suture through a needle, as well as those that require maximal force and/or velocity, such as weight lifting and sprinting. But what is it that controls the contraction of muscle? What determines the force and velocity of a contraction and what determines the maximal force generating capacity and shortening velocity of a muscle? These questions have been asked for many years and are still the subject of numerous studies. Different conditions that are associated with disproportionate changes in muscle strength and size, such as resistance training and ageing (Table 1), may help to shed some light on factors that determine the contractile properties of a muscle.

Control of muscle contraction

Jan Swammerdam (1667) was the first to show that irritation of the nerve results in contraction of the muscle. Later, Luigi Galvani (1791) showed that electrical impulses travelling along the nerve caused the muscle to contract[1]. We now know that depolarisation of the sarcolemma and propagation of the action potential over the sarcolemma into the t-tubuli leads to the release of Ca^{2+} from the sarcoplasmic reticulum. This Ca^{2+} binds to the troponin C on the actin filament, causing a conformational change of the tropomyosin that exposes the myosin binding sites on the actin filament resulting in contraction of the muscle. This whole process is called excitation-contraction coupling, as the excitation by

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MVC	CSA	Group	Age (yrs)	Training	Duration	Muscle	Ref
15%	5%	♂♀	27	Isometric	6 wks	Elbow flexors	7
36%	7%	♂♀	22	Isotonic	8 wks	Elbow flexors	11
16%	10%	♂	29	Isotonic	16 wks	Elbow extensors	25
35%	5%	♂♀	28	Isometric	12 wks	Knee extensors	10
11%	5%	♂♀	28	Eccentric	12 wks	Knee extensors	10
15%	5%	♂♀	28	Concentric	12 wks	Knee extensors	10
30%	19%	♂	29	Isotonic	6 mo	Knee extensors	12
33%	5%	♂	20	Isotonic	9 wks	Knee extensors	8
39%	7%	♂♀	20	Isotonic	5 wks	Knee extensors	31
16%	10%	♂	27	Isotonic	14 wks	Knee extensors	6
36%	5%	♂	42	Isotonic	6 mo	Knee extensors	14
36%	2%	♂	72	Isotonic	6 mo	Knee extensors	14
66%	9%	♀	39	Isotonic	6 mo	Knee extensors	14
57%	6%	♀	67	Isotonic	6 mo	Knee extensors	14
-47%	-16%[1]	♂	25 vs. 74			Gastrocnemius	53
-43%	-30%[2]	♀	23 vs. 70			Knee extensors Knee flexors	51
-44%	-24%	♂	28 vs. 68			Knee extensors	63
-32%	-14%	♂	28 vs. 68			Elbow flexors	63

[1] Physiological cross-sectional area; [2] muscle volume

Table 1. Changes in maximal voluntary force (MVC) and muscle cross-sectional area (CSA) after strength training, and below the line in the table some references to disproportionate changes in MVC and CSA during ageing.

nerve stimulation is coupled to the resulting contraction.

Based on the above description one may conclude that any stimulus of the motor nerve will result in a complete and indiscriminate contraction of the muscle. This would indeed be the case were it not that the motor nerve is composed of a bundle of axons having their origin in a multitude of motoneurons in the spinal cord. Each motoneuron innervates a number of muscle fibres and an action potential originating from the motor neuron will result in the contraction of all its innervated fibres; they thus work as a unit, hence the term motor unit. This arrangement provides a possibility to control the force and velocity of a contraction, where during low intensity contractions small motor units, consisting of slow-oxidative muscle fibres, are recruited and with increasing contraction intensity more and more large motor units, consisting of fast fibres, are recruited[2]. Further fine-tuning is possible by varying the stimulation frequency and thereby regulating the force-generation of individual motor units, where an increase in stimulation frequency results in an increase in force. Clearly, this tight neuronal and muscular interaction normally ensures that the muscle contracts with the right amount of force and velocity to perform a certain task adequately.

Muscle architecture

The angle at which muscle fibres insert into the aponeurosis, the pennation angle, affects the force exerted on the tendon. This force can be calculated as the product of force generated by all fibres and the cosine of their pennation angle, assuming that the aponeurosis is parallel with the tendon[3]. The pennation angle will also affect the shortening velocity of the muscle, as the amount of whole muscle shortening is, analogous to the tendon force, the product of muscle fibre shortening and the cosine of the pennation angle[4]. This reduction in shortening velocity in a pennate-fibred muscle, however, may to some extent be compensated by the sweeping movement of the muscle fascicles (at least during the initial stages of a contraction), which may cause the aponeurosis to move faster than the shortening velocity of the fascicles[5]. Nevertheless, the larger the pennation angle, the lower the resolved force and shortening velocity, leading to a reduction in resultant power per muscle volume (Figure 1). At this stage, one may wonder what the benefit could be of having pennate rather than parallel-fibred muscles. An explanation may be obtained when considering a contracting pennate-fibred muscle. After Swammerdam (1664) showed that mus-

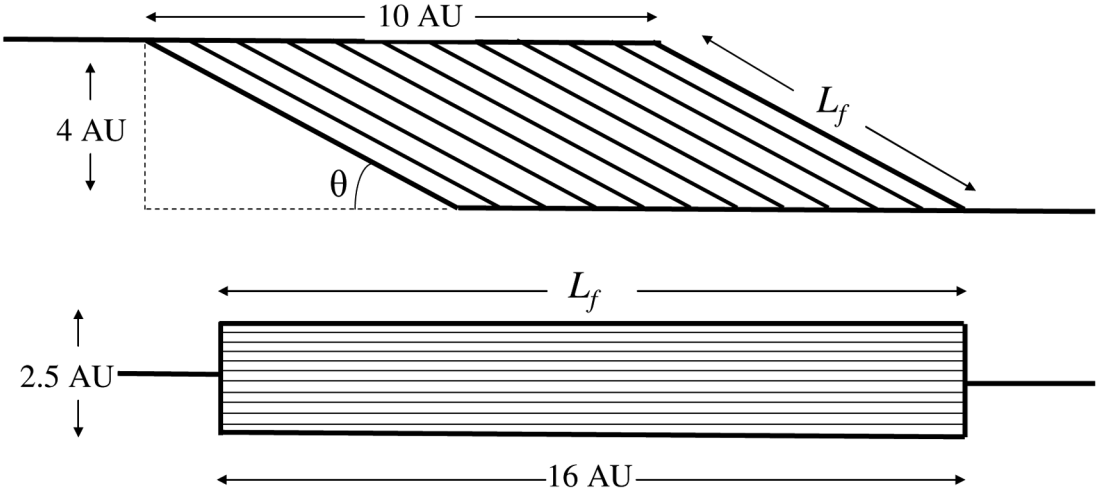

Figure 1. A pennate muscle produces less power than a parallel-fibred muscle of the same volume. Power is determined by the shortening velocity and force. We assume that the angle of pennation (θ) is 30˚ in the pennate muscle (the assertion applies to any angle). The volume of both muscles is 40 arbitrary units (AU), where the depth is 1 AU. The thickness of the pennate muscle is 4 AU and the length of the aponeurosis with fibres attached is 10 AU. The fascicle length (L_f) of the parallel muscle is 16 AU and the thickness (and, in this case, the physiological cross-sectional area, PCSA), is 2.5 AU. To calculate the PCSA of the pennate muscle we first calculate L_f as: (muscle thickness) 4AU * sin30˚=8AU. The PCSA of the pennate muscle is subsequently calculated as muscle volume/L_f: 40AU/8AU=5AU. For the calculation of force and velocity of the muscle we assume that the force-velocity characteristics of the fibres are the same in both muscles. The tendon forces of the pennate and parallel muscles are as follows:
Pennate: 5AU * cos30˚=4.33AU Parallel: 2.5AU * cos0˚=2.5AU
Hence, the pennate muscle produces 1.73 x (4.33/2.5) as much force as the parallel muscle. For the shortening velocity the following applies:
Pennate: 8AU * cos30˚=6.93AU Parallel: 16AU * cos0˚=16AU
Hence, the pennate muscle shortens at 0.43 x (6.93/16) the velocity of the parallel muscle. Multiplying these two factors (0.43 x 1.73) shows that the pennate muscle in this example produces only 74% of the power of a parallel muscle of the same volume.

cle volume did not change during a contraction, Niels Stensen (1667) was the first to point out that during a contraction changes in muscle architecture must occur to accommodate the increased cross-sectional areas of the fibres on the given surface area of the aponeurosis[3]. Furthermore, at a given shortening of muscle fibres, the shortening of the whole muscle is less, thereby enabling a finer control of movement.

Strength training

As the maximal force generating capacity of a muscle is primarily determined by the number of sarcomeres in parallel, one might expect that the increase in strength following resistance training is proportional to the increase in size of the muscle. Yet, short-term resistance training results in an increase in strength that is higher than expected from the increase in muscle size in both young[6-13] and elderly people[14,15] (Table 1). It has been suggested that this disproportionate increase in muscle strength is due to an increase in neural activation[11,16] but this is not always observed[10,17,18]. It is also possible that a decrease in the co-activation of antagonist muscles may contribute to the increase in maximal voluntary contraction torque following strength training[14,19,20], although most studies observe no such change in antagonist co-activation with training[6,21,22]. Finally, changes in muscle architecture may contribute to the disproportionate increase in muscle strength and size.

Changes in muscle architecture

The increase in muscle size in response to strength training is often given as a change in anatomical cross-sectional area (ACSA), as measured with magnetic resonance imaging, ultrasonography or computer tomography. However, muscle ACSA (represented as the distance between the aponeuroses in Figure 1) does not take into account the pennation angle of the fascicles. Furthermore, the greater the pennation angle, the more the true physiological CSA (PCSA) of the muscle is underestimated by just measuring the ACSA[23,24]. Consequently, due to the concomitant increase in pennation angle in young[6,25], old[22] and even frail[26] individuals, any training-induced increase in muscle fibre CSA (fCSA)[6,27-29] will not necessarily be reflected by a similar increase in ACSA[6]. The benefit of an increase in pennation angle is that more contractile material can be attached to the aponeuroses, thus enabling an increase in the force generating capacity of the muscle[6,30]. However, an increase in pennation angle would cause a simultaneous reduction in

the force of the muscle fibres resolved at the tendon, proportional to the cosine of the pennation angle (Figure 1). Despite this trade-off, the overall effect on the resolved tendon force remains positive as long as the pennation angle does not exceed 45° [23].

Some investigators have observed that muscle fibre length increases following strength training in both young[31] and old[22,26] individuals, which would increase the maximum shortening velocity of the muscle fibre. As maximal power output is largely determined by the maximal force generating capacity and the maximal shortening velocity of the muscle, an increase in fibre length and PCSA is expected to result in an increased peak power. However, a training-induced increase in pennation angle would not only limit the resolved tendon force but also reduce the maximal shortening of the muscle as a whole, thereby limiting any training-induced increases in force and shortening velocity (Figure 1). It has been reported that the increase in pennation angle in the vastus lateralis muscle following resistance training is only 2.7° [6] and such a change would result in a 1% loss of the resolved tendon force. This is rather insignificant when considering that maximal strength was increased by 16%[6]. Although most studies do show an increase in power output following resistance training in both young[32,33] and old individuals[34], the slowing of the contractile properties as a consequence of an increase in pennation angle and a fast-to-slow shift in fibre type composition (see below) might explain why a training-induced increase in strength is not necessarily accompanied by an increase in peak power[35].

Muscle fibre adaptations

Changes in muscle strength and size after resistance training are likely accompanied by alterations in the size and phenotype of the muscle fibres. Indeed, apart from increases in fCSA[6,16], resistance training induces a fast-to-slow fibre type transition[27,28], which is also reflected by a shift in myosin heavy chain (MHC) isoform composition from type IIx to IIa[28,36,37]. The maximal power output of a type IIa fibre, however, is less than that of a type IIx fibre of the same size[38] and a IIx-to-IIa shift in MHC composition would attenuate, rather than augment, the increase in muscle power as a result of muscle fibre hypertrophy. Another factor that might explain the disproportionate increase in muscle strength and size following resistance training is a greater myofibrillar packing density of muscle fibres[39]. The training-induced increase in the specific tension (force per CSA) of single muscle fibres[40,41] lends support to this suggestion, but this is not a consistent observation[42,43].

Overall, neither alterations in neural control, nor changes in muscle architecture or fibre quality appear to fully explain the disproportionate increase in strength and muscle size. It has been suggested that an increase in lateral force transmission from the muscle fibres to the aponeuroses may explain this phenomenon[44]. The assumption is that connective tissue attachments between the intermediate sarcomeres and the aponeurosis might effectively increase the PCSA of the muscle but reduce its functional length, thus resulting in a stronger but slower muscle[44]. Although lateral force transmission does occur[45], it is speculative whether resistance training causes a change that could explain the disproportionate increase in muscle strength and size following resistance training.

Ageing

In contrast to the increase in strength in response to resistance training, ageing is associated with a progressive remodelling of the neuromuscular system with a number of implications for muscle strength, power and ultimately quality of life. Sarcopaenia (the age related loss of muscle mass) is well documented[46] and is to a large extent due to a decline in physical activity[47,48] and a loss of motor units secondary to a decline in innervating motoneurones[49,50]. The contribution of sarcopaenia to weakness in the elderly may be further mitigated by a decline in the ability to fully recruit the agonist muscle during forceful contractions and a decline in net torque resulting from an increased co-activation of antagonist muscles[51,52]. However, the contribution of changes in muscle architecture and the interrelationship with tendon compliance to changes in strength and velocity of contraction with ageing have seldom been considered.

Muscle Architecture and ageing

As discussed above, an increase in muscle size in response to resistance training is accompanied by a change in muscle architecture. Similarly, the decrease in muscle volume as a result of ageing also affects muscle architecture. The decrease in pennation angle and fascicle length (L_f) during ageing has a number of implications for strength and velocity of contraction[53]. The smaller pennation angle in the elderly may compensate in part for a lower PCSA (Figure 1). In reality, however, this theoretical advantage is rather insignificant. Indeed, the 2° decrease in pennation angle at MVC in the gastrocnemius of the elderly compared to younger counterparts confers a 1% advantage only[53]. In fact, similar to what was discussed during strength training, considering neural activation and muscle architecture does not entirely explain the loss of strength during ageing[52,53] and is likely to reflect the age-related reduction in specific tension of single muscle fibres[54-57].

At first glance one might expect that the decline in L_f with ageing has a number of implications on the working length of the muscle (L_m). It has been reported, however, that the L_f to L_m ratio is constant during ageing for a given muscle[53] and any change in PCSA (muscle volume divided by L_f) is therefore proportional to a change in muscle volume. The implication of this constant ratio is further that (1) muscle fibres within a given muscle, *ceteris paribus*, always shorten by the same relative amount, no matter the length of the muscle, enabling fibres to operate over the same region of the length-

tension relation and (2) it may represent a natural strategy to preserve the force generating capacity of the muscle (since the latter depends on PCSA, which is the ratio of muscle volume to L_f) at the expense of shortening velocity.

Tendon properties

Ageing is associated with an increase in tendon compliance and hence increased fascicular shortening during a given shortening of the muscle-tendon complex[58]. This may result in a larger degree of fibre shortening than would be the case if the tendon were stiff. If the muscle functions on the ascending part of the length-tension relation, such as is the case in the gastrocnemius and vastus lateralis muscles[22], the greater fascicular shortening during contraction may contribute to the age-related reduction in specific tension[58]. When one considers the *in vivo* length tension relation of the gastrocnemius muscle[59] a 10% greater shortening could cause as much as a 20% reduction in specific tension[53]. The impact of this phenomenon during the shortening of muscles involved in tasks such as stair ascent, gait and balance is particularly relevant to elderly individuals working much closer to their maximal capacity than young people[60-62]. It has been shown in elderly people that the resistance training-induced increase in stiffness of the patella tendon places the fascicles of the vastus lateralis on a more favourable portion of the fascicle length-tension relation[22] and may thereby reverse to some extent the age-related loss of force generating capacity and slowing of the muscle-tendon complex.

Summary

To obtain an accurate estimate of the specific tension and power of a muscle *in vivo*, one needs to take into consideration muscle architecture, neural control of agonist and antagonist muscles and fibre type composition. However, even when we take these factors into account, the disproportionately larger change in strength than muscle size after resistance training and ageing in humans remains to be fully explained. More comprehensive studies are required to address this issue by carefully considering a wide range of factors that determine muscle strength and power, such as neural control and muscle architecture. It should also be considered that the muscle architecture assessed with ultrasound gives a two dimensional representation of the muscle, while fascicles are also pennate in the third dimension. Another limitation is that the models often assume that fascicle lengths and pennation angles are constant along the length of the muscle. Taking these considerations into account in future experiments may help to explain the disproportionate changes in muscle size and force generating capacity following resistance training, disuse and ageing. It also remains to be seen whether an altered quality of the muscle fibres and/or an increased lateral force transmission offer an explanation for this phenomenon.

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