REVIEW ARTICLE

Sports Med 2011; 41 (1): 17-38
0112-1642/11/0001-0017/$49.95/0

Developing Maximal Neuromuscular Power

Part 1 – Biological Basis of Maximal Power Production

Prue Cormie,[1] *Michael R. McGuigan*[2,3] and *Robert U. Newton*[1]

1 School of Exercise, Biomedical and Health Sciences, Edith Cowan University, Joondalup, Western Australia, Australia
2 New Zealand Academy of Sport North Island, Auckland, New Zealand
3 Institute of Sport and Recreation Research New Zealand, Auckland University of Technology, Auckland, New Zealand

Contents

Abstract . . . 17
1. Muscle Mechanics . . . 18
   1.1 Force-Velocity Relationship . . . 18
   1.2 Length-Tension Relationship . . . 19
   1.3 Type of Muscle Action . . . 20
      1.3.1 Time Available to Develop Force . . . 20
      1.3.2 Storage and Utilization of Elastic Energy . . . 20
      1.3.3 Interactions of Contractile and Elastic Elements . . . 20
      1.3.4 Potentiation of Contractile and Elastic Filaments . . . 21
      1.3.5 Stretch Reflexes . . . 21
      1.3.6 Effect of Training on Stretch-Shortening Cycle Function . . . 22
2. Morphological Factors . . . 22
   2.1 Muscle Fibre Type . . . 22
   2.2 Muscle Architecture . . . 23
      2.2.1 Cross-Sectional Area . . . 23
      2.2.2 Fascicle Length . . . 24
      2.2.3 Pennation Angle . . . 25
   2.3 Tendon Properties . . . 25
3. Neural Factors . . . 26
   3.1 Motor Unit Recruitment . . . 26
   3.2 Firing Frequency . . . 27
   3.3 Motor Unit Synchronization . . . 28
   3.4 Inter-Muscular Coordination . . . 29
      3.4.1 Activation of Synergists . . . 29
      3.4.2 Co-Activation of Antagonists . . . 30
4. Muscle Environment . . . 30
5. Conclusion . . . 30

Abstract

This series of reviews focuses on the most important neuromuscular function in many sport performances, the ability to generate maximal muscular power. Part 1 focuses on the factors that affect maximal power

production, while part 2, which will follow in a forthcoming edition of *Sports Medicine*, explores the practical application of these findings by reviewing the scientific literature relevant to the development of training programmes that most effectively enhance maximal power production. The ability of the neuromuscular system to generate maximal power is affected by a range of interrelated factors. Maximal muscular power is defined and limited by the force-velocity relationship and affected by the length-tension relationship. The ability to generate maximal power is influenced by the type of muscle action involved and, in particular, the time available to develop force, storage and utilization of elastic energy, interactions of contractile and elastic elements, potentiation of contractile and elastic filaments as well as stretch reflexes. Furthermore, maximal power production is influenced by morphological factors including fibre type contribution to whole muscle area, muscle architectural features and tendon properties as well as neural factors including motor unit recruitment, firing frequency, synchronization and intermuscular coordination. In addition, acute changes in the muscle environment (i.e. alterations resulting from fatigue, changes in hormone milieu and muscle temperature) impact the ability to generate maximal power. Resistance training has been shown to impact each of these neuromuscular factors in quite specific ways. Therefore, an understanding of the biological basis of maximal power production is essential for developing training programmes that effectively enhance maximal power production in the human.

Maximal power describes the highest level of power (work/time) achieved in muscular contractions.[1] From an applied perspective, maximal power represents the greatest instantaneous power during a single movement performed with the goal of producing maximal velocity at takeoff, release or impact.[2,3] This encompasses generic movements such as sprinting, jumping, changing direction, throwing, kicking and striking and therefore applies to the vast majority of sports. Empirical evidence supported by previous research has shown that superior ability to generate maximal power typically results in enhanced athletic performance.[2-6] A series of interrelated neuromuscular factors contribute to maximal power production. These factors, as well as any evidence of adaptations to these factors following training, will be discussed in part 1 of this review. Part 2, which will follow in a forthcoming edition of *Sports Medicine*, will explore the scientific literature relevant to the development of training programmes that most effectively improve maximal power production in dynamic athletic movements.

The search for scientific literature relevant to this review was performed using US National Library of Medicine (PubMed), MEDLINE and SportDiscus® databases and the terms 'maximal power' and 'muscular power'. Relevant literature was also sourced from searches of related articles arising from the reference list of those obtained from the database searches. The studies reviewed examined factors that could potentially influence the production of maximal muscular power.

1. Muscle Mechanics

1.1 Force-Velocity Relationship

The force-velocity relationship represents a characteristic property of muscle that dictates its power production capacities. Various levels of organization have been used to study the relationship including molecular and single-cell levels, whole muscle and multi-muscle movements, as well as single and multi-joint movements.[7-13] Regardless of the approach, the characteristic hyperbola (figure 1) can be used to describe the

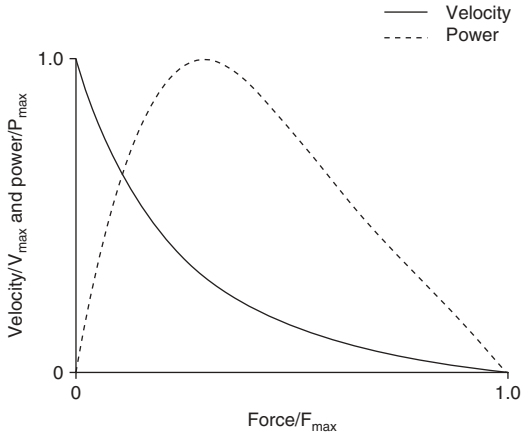

Fig. 1. The force-velocity and force-power relationships for concentric contractions of skeletal muscle. Force, velocity and power are normalized to the maximum isometric force (F_{max}), maximum velocity of shortening (V_{max}) and maximum power output (P_{max}), respectively.

inverse relationship between the force and velocity during concentric muscle contraction.[14] As the velocity of concentric muscle action is increased, less force is capable of being generated during that contraction. This is true for a given muscle or muscle group activated at a constant level as is due to actin-myosin cross-bridge cycling. Specifically, because it takes a fixed amount of time for cross-bridges to attach and detach, the total number of cross-bridges attached decreases with increasing velocity of muscle shortening. Due to the fact that the amount of force generated by a muscle depends on the number of attached cross-bridges, force production decreases as the velocity of the contraction increases and power, therefore, is maximized at a combination of sub-maximal force and velocity values.[15] Although the force-velocity relationship was first defined using isolated frog sartorius muscle,[14] all human movements are similarly limited by this fundamental property of muscles.[7,8,10-12,16,17] Maximal muscular power is therefore determined by the parameters of the force-velocity relationship: maximal isometric force (F_{max}), maximal velocity of shortening (V_{max}) and the degree of curvature (defined by a/F_{max} or b/V_{max}). Improvements in maximal power output of a muscle can be achieved through increasing F_{max} or V_{max} and/or decreasing the degree of curvature. Measurements of the force-velocity relationship during movements *in vivo* (more accurately termed load-velocity or torque-angular velocity relationship but referred to as force-velocity relationship throughout to prevent confusion) are complicated by mixed fibre composition,[16,18,19] architectural characteristics,[20,21] anatomical joint configuration[16] and levels of neural activation.[7,21-24] Despite these limitations, examination of the force-velocity relationship during such movements quantifies the ability of the intact neuromuscular system to function under various loading conditions. This information is essential in understanding maximal power production during human movements.

1.2 Length-Tension Relationship

The ability of skeletal muscle to generate force is critically dependent on sarcomere length.[25-27] The greatest potential for force production on activation of the cross-bridge cycle exists when the sarcomere length provides for optimal overlap between the actin and myosin filaments (described as the 'optimal length'). At this length, cross-bridge interaction is maximal, which allows for the greatest levels of active tension development.[25-27] Force production is impaired when sarcomere lengths are shortened below the optimal length due to overlap of the actin filaments from opposite ends of the sarcomere and the compression of the myosin filament as it comes in contact with the Z-disk.[15] Stretching a sarcomere beyond the optimal length also reduces the force production capacity. At longer lengths, cross-bridge interaction is decreased as a result of less overlap between actin and myosin filaments.[25-27] *In vivo* research has demonstrated that resting muscle lengths are generally slightly shorter than the optimal length[28] and, therefore, muscular force may be increased with a slight stretch prior to activation. While muscular power is defined by the force-velocity relationship, the length-tension relationship influences the ability of muscle fibres to develop force and, therefore, plays an important role in maximal muscular power production.

1.3 Type of Muscle Action

The ability of muscle to generate maximal power is influenced by the type of action involved; eccentric or concentric contractions as well as actions involving the combination of eccentric, isometric and/or concentric contractions.[29] Muscle function required in natural human movement rarely calls for the use of these muscle actions in isolation. The successive combination of eccentric and concentric actions forms the most common type of muscle function and is termed the stretch-shortening cycle (SSC).[29,30] When a muscle fibre is activated, stretched, then immediately shortened, the force and power generated during the concentric action is greater than a concentric-only contraction.[31,32] Therefore, maximal muscular power is superior in movements involving a SSC.[17,33-40] While there is a consensus within the literature regarding the potentiating effect of a SSC on performance, the mechanisms responsible for improved performance during SSC movements are an issue of debate amongst researchers.

1.3.1 Time Available to Develop Force

One of the proposed mechanisms driving the superior maximal power output observed during SSC compared with concentric-only movements is based on the fact that it takes time for muscle to generate force (due to time constraints imposed by stimulation, excitation and contraction dynamics[41]). The eccentric action during a SSC movement allows time for the agonist muscles to develop considerable force prior to the concentric contraction. In contrast, the concentric contraction starts as soon as force development (beyond that which is required to maintain a static position) begins in concentric-only movements. An alternate view of this same principle is that SSC contractions have enhanced power generation capability due to the greater distance over which force can be developed compared with concentric-only movements (i.e. based on the work-energy relationship). Hence, force during the concentric phase is greater in SSC movements, subsequently resulting in superior performance.[42-46] However, power output was observed to be higher in a SSC movement compared with a concentric-only movement immediately preceded by a maximal isometric action,[47] indicating that the time available to develop force is not the only factor contributing to enhance muscular power.

1.3.2 Storage and Utilization of Elastic Energy

The most generally reported mechanism believed to drive the SSC-induced enhancement of maximal power is the storage and utilization of elastic energy.[48] When an active muscle-tendon unit (MTU) is stretched, mechanical work is absorbed by the MTU and this work can be stored in part as potential energy in the series elastic component (SEC; includes fibre cross-bridges, aponeurosis and tendon).[31,34,49] It is believed that some of this potential energy can then be used to increase the mechanical energy and positive work during the following concentric contraction.[17,31,33,34,36,49] This recoil of the SEC is thought to contribute to the increased force at the beginning of the concentric phase in SSC movements and ultimately to enhanced maximal power production.[17,31,33,34,36,49]

1.3.3 Interactions of Contractile and Elastic Elements

In SSC movements, the interactions between the contractile and elastic elements play an important role in enhancing maximal performance. Tendinous recoil has been shown to influence the contribution of the contractile component of work produced during SSC movements.[50-52] Higher force at the beginning of the concentric phase during SSC movements results in greater tendinous lengthening with less fascicle lengthening.[53-57] As the concentric contraction progresses, the muscle fibre contracts at a nearly constant length (i.e. isometric), while the rapid shortening of the MTU largely depends on the shortening of the tendinous structure.[53-57] In contrast, while some tendinous displacement does occur, the majority of the MTU length change during concentric-only movements is due to fascicle shortening.[54] The minimal displacement of muscle fibres during the concentric phase of SSC movements is believed to be caused by the catapult action of the tendinous structures (i.e. lengthening-shortening behaviour).[58]

These interactions may influence performance in three distinct ways. First, elastic energy would be stored predominantly in the tendinous structures and therefore can be utilized with minimal dissipation via the tendon recoil during the concentric phase.[58,59] Second, the minimal displacement of muscle fibres during SSC movements means that they operate closer to their optimal length and, based on the length-tension relationship, can therefore produce more force.[53,55,56] Finally, while the net shortening velocity of the MTU is high, fascicle length change occurs at relatively slow velocities. Thus, fascicles are able to generate high forces according to the force-velocity relationship.[60] Therefore, during SSC movements, the contractile element acts as a force generator producing high forces at relatively low shortening velocities, while the tendinous structures act as an energy re-distributor and power amplifier.[60] The interaction of these components is vital in SSC movements because it allows for the muscle-tendon complex to generate superior maximal power output.

1.3.4 Potentiation of Contractile and Elastic Filaments

The potentiation of the actin-myosin cross-bridges is another mechanism thought to contribute to the SSC-induced enhancement in maximal power output.[34,47,50,61] In tetanized isolated muscle and single muscle fibres, an active stretch has been observed to enhance work output of the contractile machinery during subsequent shortening,[32,62-64] a finding supported by *in vivo* studies involving intact muscle-tendon complexes.[34,47,61] This potentiating effect is thought to be due to enhanced force production per cross-bridge rather than an increase in the number of active cross-bridges.[62,64] Woledge and Curtin[65] proposed that strained cross-bridges are detached in a state that permits them to re-attach more rapidly than cross-bridges not exposed to a pre-stretch. While suggestions have also been made that some cross-bridges may be left in a highly strained state after the stretch, it is not currently known precisely how the force per cross-bridge is enhanced.[61] Despite the convincing *in vitro* evidence, the extent to which the potentiation of the contractile filaments influences *in vivo* SSC performance has been questioned.[66] *In vivo* observations of isometric (rather than lengthening) action of muscle fascicles during a stretch[54,55] cast doubt on the possible contribution of force potentiation to enhanced SSC performance *in vivo*. Additionally, the potentiation of elastic filaments such as titin and/or nebulin has been proposed as another possible mechanism contributing to enhanced force production following an active stretch.[67-70] It has been theorized that an active stretch may be associated with a calcium-dependent increase in titin stiffness, which in turn contributes to enhanced force production compared with a non-activated stretch.[67-70] However, a recent investigation suggests that enhanced force production in the absence of actin-myosin overlap cannot be explained by calcium-induced stiffening of titin and proposes cross-bridge force-dependent titin-actin interactions to be responsible for non actin-myosin-based force enhancement observed following an active stretch.[71] Indeed, further research is required to establish if, and to what extent, potentiation of contractile and elastic filaments occurs during SSC movements *in vivo* as well as the relative contribution of this effect to maximal muscular power.

1.3.5 Stretch Reflexes

Another mechanism proposed to contribute to the enhanced maximal power output during SSC movements is the activation of spinal reflexes. The forced lengthening of the MTU during the eccentric phase of SSC movements causes a mechanical deformation of the muscle spindles, which activates reflex mechanisms (stretch reflexes of α-motoneurons).[72] The stretch reflex subsequently increases muscle stimulation, resulting in increased contraction force during the concentric phase and ultimately contributes to enhanced maximal power output.[37,39,73-78] Despite some reservations, the consensus within the literature appears to be that SSC movements do evoke a stretch reflex of sufficient magnitude to contribute to the increase in muscular force during the concentric phase.[37,39,48,73-75,77] Therefore, the development of maximal power during SSC movements

may be influenced in some degree by the activation of stretch reflexes.

1.3.6 Effect of Training on Stretch-Shortening Cycle Function

The beneficial effects of resistance training on SSC performance has been well documented.[79-84] However, to date, no conclusive evidence exists identifying how the aforementioned mechanisms contributing to enhanced SSC performance are affected by training. Several speculative theories exist but further research is required to identify the adaptations driving training-induced improvements in SSC performance.

2. Morphological Factors

The ability to generate maximal power during a movement is dictated by the contractile capacity of the muscles involved. The contractile capacity of muscle is influenced by a series of morphological factors but primarily its fibre type composition and architectural features. Additionally, the properties of tendon influence the function of the contractile elements within the MTU and therefore impact maximal power production.

2.1 Muscle Fibre Type

Due to the unique characteristics of each fibre type, the force-velocity properties of a muscle are determined by the fibre type contribution to whole muscle area.[8,12] Type II fibres have a greater capacity to generate power per unit cross-sectional area (CSA).[8,12,19,85-87] In an investigation of single fibres from the vastus lateralis, peak power per unit CSA was observed to be 5- and 10-fold greater in type IIa and IIx fibres, respectively, when compared with type I fibres.[87] However, these contractile properties were measured as sub-physiological temperatures (15°C) and thus may not reflect function *in vivo*.[88] Examination of results of studies using closer to *in vivo* muscle temperatures suggest that the differences in peak power per unit CSA are smaller than those observed at lower temperatures. In a study specifically addressing this issue, the propelling velocity of actin filaments by myosin from human muscle fibres was only 2-fold greater with type IIx versus type I myosin when measured at 35°C, compared with a 7.5-fold difference at 15°C.[88] In a rare study measuring the contractile properties of intact human muscle fibres at 37°C, bundles of type II fibres were found to have a 3-fold greater V_{max} and a 4-fold greater maximum power output (P_{max}) than bundles of type I fibres.[19] The differences in peak power per unit CSA are due to differences in specific force (i.e. F_{max}/CSA), V_{max} and the curvature of the force-velocity curve amongst the fibre types.[13,15,19,87] Using single fibre preparations, type II fibres have been observed to have significantly greater specific force than type I fibres.[13,87,89] Similar findings have been observed in whole skeletal muscle investigations (i.e. muscles composed mainly of type II fibres vs mainly type I fibres) although this is a somewhat controversial area in muscle physiology.[15] However, differences in V_{max} are theorized to have a much more pronounced influence on the difference in P_{max} values between fibre types.[15] Type II fibres are characterized by high sarcoplasmic reticulum and myofibrillar adenosine triphosphatase (ATPase) activities, and correspondingly high V_{max} and short contraction time/twitch duration (i.e. the heads of type II myosin isoforms split ATPase approximately 600 times/second vs approximately 300 times/second for type I myosin isoforms).[90-94] This allows for a short cross-bridge cycle time and, therefore, the ability to develop force rapidly. In contrast, type I fibres display comparatively low ATPase activity and V_{max} with long contraction times/twitch durations.[90-94] For example, V_{max} has been shown to vary from approximately 0.8 fibre lengths/second in type I fibres to approximately 3.5 fibre lengths/second and 5.6 fibre lengths/second in type IIa and IIx fibres, respectively[86,95,96] (note these investigations used sub-physiological temperatures and thus may not reflect function *in vivo*[88]). When this literature involving single fibre preparations is collated, a continuum of V_{max} (relative to fibre length) and P_{max} (relative to CSA) for the fibre types is evident as follows IIx > IIa > I. Furthermore, investigations of bundles of fibres reported a greater a/F_{max} ratio in type II versus type I fibres, indicating a greater degree of curvature of the force-velocity curve, and thus lower power output, for type I fibres.[19,94]

Therefore, the maximal power output of a muscle is influenced by its fibre type composition. Muscles with a high percentage of type II fibres display greater P_{max} in comparison to muscles with a high percentage of type I fibres.[8,12,97] However, future research is required in order to determine the magnitude of differences in P_{max} as well as V_{max} between fibre types and subtypes at physiological temperatures in intact fibres.

Cross-sectional comparisons have revealed that elite strength-power athletes have predominately type II fibres, whereas elite endurance athletes display a predominance of type I fibres.[98,99] While approximately 45% of the variance in muscle fibre type is believed to be associated with inherited factors,[100] findings of fibre type transformations from type I to II (and *vice versa*) after periods of intense training[101-105] and detraining[106,107] indicate plasticity in fibre type composition based on environmental conditions.[100] However, transformations between type I and II fibres have been debated throughout the literature and further research is required to understand the precise conditions under which they occur.[15] Additionally, resistance training has been shown to elicit transformations in myosin heavy chain gene expression within type I and II fibres. Transformations in type II subtypes have occurred following strength training whereby type IIx isoforms are reduced at the expense of an increase in the expression of type IIa isoforms.[87,108-110] When a muscle is chronically stressed with high loading requirements, it is theorized that the contractile protein properties are shifted to a more economical cross-bridge cycling system (i.e. increased oxidative capacity allowing for sustained power output over a longer period).[111,112] This shift in type II subtypes may be detrimental to P_{max} but is compensated for by the preferential hypertrophy of type II fibres following strength training (discussed further in section 2.2.1). Interestingly, a period of detraining following strength training has been observed to evoke an 'overshoot' in type IIx composition that is markedly higher than values observed prior to the strength training.[110] However, the influence of ballistic power training on possible myosin heavy chain isoform shifts is unclear, with conflicting reports of strong trends towards transformations from type IIx to IIa[86,113] and no such changes following training.[114,115] Further research is required to elucidate exactly how muscle fibre subtypes respond to ballistic power training. It is important to note that even if transformations between muscle fibre types and/or subtypes did occur, the contribution to improving maximal muscular power would be relatively small compared with alterations in other morphological properties (i.e. CSA or architectural characteristics).[15] Additionally, contractile properties can also improve following training without apparent changes in fibre type or subtype proportions. For example, Malisoux and colleagues[86,116] reported increases in V_{max} of all fibre types following plyometric training as well as improvements in several functional performance measures despite an increase in type IIa at the expense of IIx. Further research is necessary to determine the degree of training-induced adaptations in contractile properties evident across the fibre types and subtypes at physiological temperatures.

2.2 Muscle Architecture

2.2.1 Cross-Sectional Area

The maximal force generated by a single muscle fibre is directly proportional to its CSA, irrespective of the fibre type.[1,18,117-119] Due to the fact that power is heavily influenced by F_{max}, a muscle fibre with greater CSA can therefore generate higher P_{max}.[16,86,87,120] A comparison of single muscle fibres between sedentary men and men involved with regular resistance training for 7.6 ± 1.6 years highlights these findings.[120] The resistance-trained men had significantly greater CSA, F_{max} and P_{max} for type I and type II fibres compared with the sedentary men. However, the differences between the groups were no longer evident when F_{max} was normalized to CSA and P_{max} was normalized to fibre volume (which accounts for differences in both fibre CSA and length).[120] Evidence from single fibre studies is supported by research demonstrating that maximal voluntary isometric force is proportional to whole-muscle CSA.[121-123] For example, using CT scans to assess muscle CSA, Maughan and associates[123] reported significantly higher F_{max} in muscles with

greater CSA. The F_{max}-to-CSA ratio was not significantly different between experienced strength-trained subjects and untrained controls, suggesting that variation in CSA accounted for the majority of the differences seen in F_{max}.[123] Strong relationships have also been reported between knee extension F_{max} and quadriceps CSA in both men ($r = 0.71$) and women ($r = 0.76$).[124,125] However, it is important to note that not all of the variation in whole-muscle F_{max} can be explained solely by variation in muscle CSA.[126] Factors such as neural drive,[127-129] fibre-type composition,[130] pennation angle[131] and the lever system through which F_{max} is measured[132] may also contribute.

In response to training, changes to F_{max} of single muscle fibres are proportional to changes in fibre CSA.[96,120,133] Increases in fibre CSA are brought about through increases in the size and number of myofibrils within the muscle fibre.[111,134,135] These hypertrophic adaptations occur in both type I and II muscle fibres in response to heavy strength training but to a greater degree in type II fibres.[109,136-141] Extensive research has established that heavy strength training is a very effective stimulus for eliciting a hypertrophic response in muscle.[87,109,112,124,129,131,142,143] Training-induced increases in CSA or F_{max} are typically accompanied by improvements in maximal muscular power.[10,16,84,86,87] However, much of this research involved relatively untrained subjects with low to moderate strength levels, in which improvements in muscular function are easily invoked. Increases in CSA following heavy strength training of stronger/more trained individuals are expected to be lower and take longer.[128] Therefore, the possible influence of increased CSA on muscular power is theorized to diminish as the training age of the athlete increases. Furthermore, the degree of muscle hypertrophy is highly dependent on the type of training and the specific programme variables (i.e. intensity, volume and frequency).[144] The relatively lighter loads used during ballistic power training are typically too small to elicit the necessary mechanical stimulus required to initiate a significant hypertrophic response.[144-150] However, observations of hypertrophic responses following plyometric training[86,151,152] indicate that further research is necessary to determine the important variables in plyometric and/or ballistic training that may elicit an increase in CSA (i.e. significant eccentric component to plyometrics, volume or time under tension, etc.). Consequently, increases in maximal muscular power mediated by improved CSA are achieved primarily through heavy strength training and, typically, not (or markedly less) in response to specific power training.

2.2.2 Fascicle Length

While sarcomere V_{max} differs quite significantly between various fibre types, the V_{max} of a muscle fibre is proportional to its length (assuming a constant level of activation).[16,18,118,153-155] For example, if a sarcomere shortens at two fibre lengths per second, a fibre containing ten sarcomeres in series would have a greater V_{max} than a fibre containing five sarcomeres in series (i.e. 20 vs 10 fibre lengths/second). Due to the fact that power is heavily influenced by V_{max}, a longer muscle fibre can therefore generate higher P_{max}.[16,18,118,153] Correlational studies have reported significant relationships between fascicle length of vastus lateralis and gastrocnemius lateralis and 100 m sprint time in both men and women ($r = -0.43$ to -0.57).[156,157] Furthermore, cross-sectional investigations have revealed the fascicle lengths of the vastus lateralis, gastrocnemius medialis and gastrocnemius lateralis to be significantly longer in sprinters compared with long-distance runners and untrained controls.[158] However, it is unclear if these observations are a result of genetic predisposition or if fascicle lengthening is an adaptation to the modalities of training commonly used by sprinters (i.e. high-intensity sprint training and high-intensity strength/power training). Regardless of the origin of this architectural difference, these data indicate the importance of relatively longer fascicle lengths to rapid force-generation and maximal power production during dynamic movements.

The adaptive response of fibre length following training is not well understood. Animal models have been used to investigate fibre length change following various training interventions but have returned inconclusive results.[159-161] Fascicle length in humans has been measured as an indicator of fibre length but the current literature

offers little additional insight into the influence of training on fibre length. Training studies have reported fascicle length to increase in response to resistance training with heavy loads,[142,162-164] resistance training with light loads,[165] as well as in subjects who ceased strength training and performed jump and sprint training.[142] In contrast, an effective heavy strength training programme of the elbow extensors had no effect on fascicle length of the triceps brachii,[166] a finding supported by similar studies involving the lower body musculature.[167,168] While some of these changes were coupled with improvements in performance, it is unknown exactly how the changes in fascicle length affected muscle V_{max} or P_{max}. Further research is required to elucidate the most effective training stimulus for longitudinal growth of muscle fibres. Furthermore, while the addition of sarcomeres in series is theorized to occur through similar pathways as the addition of sarcomeres in parallel, factors determining which type of muscle growth occurs are unknown (the interested reader should refer to Blazevich and Sharp[169] for a more detailed discussion).

2.2.3 Pennation Angle

The pennation angle of a muscle, defined as the angle between the muscle's fascicles and the line of action,[155,170,171] has important physiological effects on the force-velocity relationship and thus P_{max}. As pennation angle increases, more sarcomeres can be arranged in parallel (i.e. more contractile tissue can attach to a given area of an aponeurosis or tendon) and the muscle can therefore produce more force.[154,172] Additionally, an increased pennation angle allows for muscle fibres to shorten less for a given tendon displacement due to the rotation of pennate muscle fibres during contraction.[173] This increases the likelihood that a fibre with a greater pennation angle operates closer to its optimum length and, based on the length-tension relationship, is able to generate more force.[173] These factors act to increase F_{max} and, therefore, pennation angle influences the maximal power output generated by a muscle. However, greater pennation angles are also associated with slower contraction velocities and thus, increasing a muscle's pennation angle may negatively impact V_{max}.[155] Despite this, the increase in F_{max} is theorized to have substantially greater impact on maximal power than increases to V_{max} brought about through an increase in pennation angle.[16]

Pennation angle is commonly thought to increase in response to heavy strength training and decrease in response to sprint training. These theories are based on observations of population differences whereby bodybuilders displayed greater pennation angles and CSA than untrained subjects,[174] and highly trained sprinters possessed smaller pennation angles than both lesser trained sprinters[157] and untrained controls.[156] Further support for possible adaptability of pennation angle to heavy strength training stemmed from the significant relationships between muscle thickness (indicative of CSA) and pennation angle in the triceps brachii ($r = 0.81$), vastus laterals ($r = 0.61$) and gastrocnemius medialis ($r = 0.56$) of over 700 people with various training backgrounds.[175] These observations were corroborated by studies involving training interventions in which heavy strength training significantly increased pennation angle,[131,166] while sprint/jump training significantly decreased pennation angle.[142] Increases in pennation angle following heavy strength training were accompanied by increased CSA and F_{max}[131,166] resulting in enhanced P_{max}.[107,110] However, other longitudinal studies have failed to establish pennation angle changes in response to heavy strength training in previously trained[176] and untrained[167,168] people. While the effectiveness of the training protocols implemented and the reliability of the techniques used may have prevented pennation angle changes being discovered, these findings highlight that the effects of heavy strength training on pennation angle are not clearly understood. Furthermore, it is unknown if ballistic power training and other training modalities elicit changes in pennation angle or if changes are influenced by the training status of the subject.

2.3 Tendon Properties

As previously discussed in section 1.3.3, fascicle behaviour is affected by interactions between the contractile and elastic elements of the

MTU.[53-57] The intrinsic compliance of tendon impacts these interactions (i.e. affects the amount of fascicle displacement) and, because a muscle's ability to generate force is both velocity and length dependent, the level of tendon compliance can influence maximal muscular power. Few data currently exist regarding the potential adaptability of tendon compliance in response to exercise[177,178] and the cross-sectional data to date have revealed mixed results.[179,180] Kubo and colleagues[179] reported a negative relationship between sprint performance and tendon compliance ($r=-0.757$) indicating that greater compliance is beneficial for sprint performance. In contrast, Bojsen-Møller and associates[180] observed knee extensor rate-of-force development (RFD) to relate positively to stiffness of the vastus lateralis tendon-aponeurosis ($r=0.55$), suggesting that less compliance is associated with enhanced muscular performance. Thus, further research is essential in order to determine the specific influence of tendon compliance on maximal power production as to whether this tendon property is amendable to exercise.

3. Neural Factors

The ability to generate maximal power during a movement is not only governed by the muscles morphology, but also by the ability of the nervous system to appropriately activate the muscles involved. The nervous system controls the activation of muscles primarily through changes in motor unit recruitment, firing frequency and synchronization as well as inter-muscular coordination.

3.1 Motor Unit Recruitment

The force produced by a muscle is related to the number and type of motor units recruited. Motor units are recruited in a systematic order during graded, voluntary contractions of increasing force according to the size principle.[181,182] Relatively small α-motoneurons that innervate type I fibres are initially activated at low force levels while progressively larger α-motoneurons that activate type IIa and IIx fibres are typically activated after the slow-twitch motor units at higher thresholds of force.[181-183] The size principle is the general rule of recruitment not only for slow, graded contractions but also for isometric[184] and ballistic contractions.[185,186] However, compared with slow, graded contractions, the threshold of motor unit recruitment is typically lower during ballistic movements due to the rapid force escalation to high levels.[186,187] The maximum force capabilities of a motor unit has been estimated to vary by up to 50 times.[188] Thus, the force capable of being generated during a movement is affected by which motor units are recruited. During contractions typically required for maximal power production, recruitment of high-threshold motor units is very beneficial to force production as they innervate a relatively large number of high RFD/force-producing muscle fibres.[189] Therefore, the ability to rapidly recruit high-threshold motor units influences maximal muscular power.

There are three common theories of adaptation in motor unit recruitment that may occur in response to training. It is hypothesized that training may result in increased motor unit recruitment, preferential recruitment of high-threshold motor units and/or lowering of the thresholds of motor unit recruitment.[128,190] All of these possible adaptations would act to increase agonist activation resulting in increased tension development by the muscle and consequently improved power output.

Observations of increased electromyography (EMG) amplitude following training suggests that a possible adaptation associated with enhanced muscular power may be an increase in the level of motor unit recruitment.[128] However, current techniques are unable to definitively establish whether or not training elicits a true increase in motor unit recruitment as this would require the identification of previously uninvolved motor units that are recruited after training. Methodologies have been implemented to gain an indication of possible training-induced changes to the level of motor unit activation (which encompasses recruitment and firing frequency). These techniques involve the comparison of force produced during a maximal voluntary contraction (MVC) and either a maximal

tetanic muscle stimulation, or a supra-maximal stimulus applied to the nerve of a muscle engaged in a MVC (i.e. interpolated twitch technique).[73,191-195] In both of these cases, the stimulus can cause a significant difference in force production between the voluntary and stimulated contractions if all motor units have not been recruited voluntarily (or the firing frequency of the recruited motor units is submaximal, as discussed in section 3.2). Results from early investigations indicated that despite considerable inter-subject variability, full voluntary activation was possible in a variety of muscles during single joint, isometric contractions in untrained but well motivated individuals.[73,128,191-194] Consequently, it was difficult to attribute training-induced increases in EMG to changes in the level of motor unit recruitment. However, advancements in techniques have allowed for more sensitive measurements, which have revealed levels of voluntary activation to range from 85% to 95% of maximum capacity in the quadriceps femoris and 95–100% in a range of other muscles.[195] Despite these differences and the theory that untrained individuals may not be able to consistently recruit the highest threshold motor units, resistance training studies involving healthy adults indicate that maximal voluntary activation does not increase following training.[196-203] It is important to note, however, that these longitudinal studies may have been impaired by use of less sensitive techniques than what are now available, the use of non-specific isometric tests to evaluate the effects of dynamic training, and the small window for improvement in some of the muscles assessed.[195] Furthermore, voluntary activation during maximal dynamic contractions has been shown to be 88–90%, significantly lower than voluntary activation during maximal isometric contraction (95.2%).[204] It may therefore be possible that training results in improved voluntary activation during dynamic movements and especially in more complex, multi-joint sport-specific movements. If future research was to demonstrate this, increased motor unit recruitment (or firing frequency) may in fact contribute to training-induced improvements in maximal muscular power.

The preferential recruitment of high-threshold motor units following training is a somewhat common theory of neural adaptation.[2,205,206] While few exceptions to the size principle exist, it has been theorized that well trained athletes may be able to activate high-threshold motor units in place of low-threshold motor units during ballistic movements in an attempt to enhance maximal muscular power.[2,206] This theory stems from selective recruitment of high-threshold motor units observed during very rapid stereotyped movements in the cat[207] as well as during eccentric[208,209] or electrically induced contractions[210,211] in humans. In one of the only studies to assess this theory, van Cutsem and co-workers[187] observed the orderly motor unit recruitment of the size principle to be preserved during both slow ramp and ballistic contractions following ballistic power training. However, this same study observed that motor units were recruited at lower thresholds after training during ballistic contractions.[187] The post-training recruitment thresholds underwent a significant shift to lower percentages of MVC than those observed during ballistic contractions at baseline and in comparison with a non-training control group. The earlier activation was reported to be likely to contribute to the observed significant increase in the speed of voluntary ballistic contraction.[187] Therefore, increases in maximal power output following training may be due in some part to lower recruitment thresholds during ballistic contractions. While preferential recruitment of type II fibres remains a possibility, the current evidence for it occurring in response to exercise in humans is not convincing. It is important to note that a motor unit is trained in direct proportion to its recruitment,[111] so movements that require the recruitment of high-threshold units must be incorporated into the training programme for changes in recruitment to have an impact on performance.

3.2 Firing Frequency

The motor unit firing frequency represents the rate of neural impulses transmitted from the α-motoneuron to the muscle fibres. The firing frequency of a motor unit can impact the ability of a

muscle fibre to generate force in two ways. First, increasing the firing frequency enhances the magnitude of force generated during a contraction. It has been estimated that the force of contraction may increase by 300–1500% when the firing frequency of a motor unit is increased from its minimum to maximum rate.[188] Second, motor unit firing frequency impacts the RFD of muscle contraction. During ballistic contractions motor units have been reported to begin firing at very high frequencies followed by a rapid decline.[212] The high initial firing frequency, which is believed to be associated with an increase in the number of doublet discharges,[187,213] results in increased RFD, even if only maintained for a very short period of time.[214] Therefore, by influencing the force and RFD of muscle contraction, motor unit firing frequency plays a role in the development of maximal muscular power.

Training-induced enhancement of maximum motor unit firing frequency has been proposed as a possible mechanism driving improvements in neuromuscular performance.[215] A cross-sectional examination reported that weightlifters displayed greater maximum motor unit firing frequency during a MVC of the quadriceps compared with untrained controls,[216] thus indicating that training may increase the maximal firing frequency of motor units. As discussed in section 3.1, most resistance training studies involving healthy adults indicate that voluntary activation (which gives an indication of both motor unit recruitment and firing frequency) does not increase following training.[196-203] However, more recent research involving intramuscular EMG has reported training-induced increases in motor unit firing frequency during maximal contractions.[187,217,218] These observations were made following strength training during maximal isometric contractions of the abductor digiti minimi[217] and vastus lateralis[219] as well as during ballistic contractions in the tibialis anterior following ballistic power training.[187] In the two strength-training studies, rapid and pronounced improvements occurred in maximal firing frequency between subsequent testing sessions prior to training, which mirrored improvements in maximal force.[217,218] Maximal firing frequency remained elevated following vastus lateralis training[218] but returned to values similar to those observed at baseline in the abductor digiti minimi after training.[217] van Cutsem and co-workers[187] observed an increase in maximal motor unit firing frequency following 12 weeks of ballistic power training as well as enhanced maximal force and RFD values. These results suggest that increases in maximal motor unit firing frequency may contribute to improved force and power generation especially in the early phases of training.

Perhaps a more important consideration for improved athletic performance is the possible training-induced adaptations to the pattern of motor unit firing frequency and the subsequent impact on RFD. Compared with long-distance runners and untrained controls, Saplinskas et al.[220] observed sprinters to have the highest motor unit firing frequency during the onset of rapid isometric dorsiflexion. This observation was supported by an intervention study that reported the peak firing frequency at the onset of ballistic contraction to increase following ballistic training.[187] Furthermore, these higher firing frequencies were maintained for longer throughout the contraction after training.[187] Additionally, the authors reported a training-induced increase in the percentage of doublet discharges (i.e. a motor unit firing two consecutive discharges in a 5 ms or less interval) at the onset of a ballistic contraction that were reported to contribute to increases in RFD and time to peak force during ballistic contractions.[187] Therefore, ballistic power training may prompt adaptations to the pattern of motor unit firing frequency that contributes to enhanced maximal power production.

3.3 Motor Unit Synchronization

Motor unit synchronization occurs when two or more motor units are activated concurrently more frequently than expected for independent random processes.[221] Although it is yet to be convincingly demonstrated, synchronization has commonly been hypothesized to augment force production and positively influence RFD.[127,222] Furthermore, synchronization is theorized to be a nervous system adaptation that assists with the

coactivation of numerous different muscles in order to enhance RFD.[223,224] The manner in which synchronization may influence force or RFD is not readily apparent. No difference in force production has been observed between asynchronous and synchronous motor unit activation at frequencies similar to those observed in MVC and asynchronous discharges of action potentials has been shown to result in greater force production at submaximal firing frequencies.[225,226] Furthermore, voluntary contractions have been shown to produce greater RFD than evoked tetanic contractions in which all motor units are stimulated to fire concurrently.[214] However, synchronization may actually be one of the strategies for inter-muscular coordination and therefore could impact force and/or RFD during complex, multi-joint movements as opposed to isolated, single-joint movements where synchronization does not appear to have a significant impact. It has been hypothesized that synchronization between muscles may be a strategy to simplify and coordinate the activity of muscles in control of mechanically unstable joints (e.g. the medial and lateral vasti muscles and the patellofemoral joint),[224] which would allow for greater transmission of muscular power in complex movements. Therefore, further investigation is required in order to determine if motor unit synchronization contributes to enhanced maximal power production especially during complex multi-joint movements.

Observations from cross-sectional comparisons have led to the theory that motor unit synchronization may improve as a result of training. Using surface EMG, Milner-Brown et al.[221] observed recreational weightlifters to display greater motor unit synchronization in the hand muscles than untrained subjects. This observation was corroborated by Semmler and Nordstrom[227] who, using techniques that measured motor unit discharges directly, demonstrated motor unit synchronization to be significantly greater in strength-trained subjects than both musicians and untrained subjects. In one of the only intervention studies examining motor unit synchronization, Milner-Brown et al.[221] reported a significant improvement in motor unit synchronization (measured by surface EMG) following 6 weeks of MVC training of the hand muscles. However, the validity of using surface EMG to assess motor unit synchronization has been questioned.[228] Therefore, further research is required to elucidate if changes to motor unit synchronization occur in response to training.

3.4 Inter-Muscular Coordination

Inter-muscular coordination describes the appropriate activation (both magnitude and timing) of agonist, synergist and antagonist muscles during a movement. For highly effective and efficient movement, agonist activation needs to be supplemented by increased synergist activity and decreased co-contraction of the antagonists.[190] The coordinated activation of these muscles is required to generate the greatest possible force in the direction of movement.[190] 'Triple extension' (i.e. extension of the hips, knees and plantar flexion of the ankles) of the lower limbs typical of jumping and sprinting involves quite complex interaction of uni- and multi-articulate musculotendinous units performing various actions. It is only with precise timing and level of activation and relaxation of the agonists, synergists and antagonists that power flow through the kinetic chain will be optimized, impulse on the ground maximized and, thus, performance in terms of takeoff velocity maximized. Therefore, the ability to generate maximal power output during athletic movements is considerably influenced by inter-muscular coordination.

3.4.1 Activation of Synergists

Synergists play a role in maximal power production and it is possible that improved activation and/or coordination of synergist muscles could contribute to enhanced performance. While there is much evidence of task-specific synergist coordination, little information is available monitoring possible changes to synergist activity brought about by training. While untrained people have been shown to activate agonists quite effectively,[191-193] it is theorized that enhanced activation and/or coordination of synergist muscles may contribute to performance improvements following training and are associated with

the superior performance of trained individuals.[229] Furthermore, adaptations in synergist muscles may help explain the increases in force production observed independent of increased neural activation of the agonists, especially during the early phases of training. Additional research is required to clarify the nature of adaptations in synergists and the relative contribution to enhancing performance.

3.4.2 Co-Activation of Antagonists

The magnitude of antagonist co-activation is dependent on various factors including the type of contraction,[230] load, velocity and precision[231] of the movement as well as its range of motion.[232] Antagonist co-activation is counterproductive to movements in which maximal force must be generated due to the fact that the co-activation would produce torque about the joint acting in the opposite direction of the desired movement.[233-235] There is also evidence that co-activation may impair the full activation of agonist muscles through reciprocal inhibition.[236] However, antagonist co-activation is beneficial in coordinating movements and maintaining joint stability during actions, especially those ballistic in nature. Despite these advantages, excessive antagonist co-activation may negatively influence the ability to perform movements with maximal power.

It is hypothesized that training-induced improvements in performance are influenced to some degree by a decrease in antagonist co-activation. Comparisons of individuals with different training backgrounds have rendered inconclusive results and intervention studies have reported conflicting evidence of adaptations to antagonist co-activation. Hence, the possible training-induced adaptations in antagonist co-activation and subsequent impact on performance, remains unclear. Antagonist co-activation has been reported to be prominent during ballistic movements[237] and, therefore, the potential to reduce co-activation in such movements following training is relatively greater. Furthermore, the level of antagonist co-activation may be much greater during dynamic, multi-joint movements than during the single-joint, isometric movements commonly researched. Although these areas have not yet been investigated, it is theorized that a reduction in antagonist co-activation during such complex movements would contribute to improvements in maximal power following training.[143]

4. Muscle Environment

Acute changes in the muscle environment (i.e. alterations resulting from fatigue, changes in hormone milieu and muscle temperature) impact muscular performance and therefore the ability to generate maximal power. During fatigue, numerous muscle properties are altered including ionic changes on the action potential, extracellular and intracellular ions as well as intracellular metabolites (the interested reader should refer to recent comprehensive reviews of this topic[238,239]). Each of these alterations negatively affects maximal muscular power through impairing the force generation and/or the velocity of shortening during contractions.[238,239] Furthermore, recent evidence suggests that the combination of factors co-existing during fatigue *in vivo* result in even greater impairment than what has been observed for fatigue factors individually.[240] While the influence of endocrine factors on adaptational mechanisms in muscle and the resulting enhancement in muscular function have been well reviewed,[241,242] acute hormonal changes may potentially impact the ability to generate maximal muscular power immediately. Recent evidence indicating that treating bundle fibres with physiological concentrations of dihydrotestosterone increases specific force and phosphorylation of myosin light chains of type II fibres, suggests that changes in androgenic hormone concentrations in the blood may acutely impact maximal muscular power.[243] Additionally, alterations in muscle temperature also influence maximal power production as it has been shown that P_{max}, V_{max}, F_{max} as well as RFD decrease with a decrease in muscle temperature[244-246] (for extensive reviews of this topic please refer to[247-249]).

5. Conclusion

Maximal muscular power is influenced by a wide variety of neuromuscular factors including muscle fibre composition, cross-sectional area,

fascicle length, pennation angle and tendon compliance as well as motor unit recruitment, firing frequency, synchronization and inter-muscular coordination. Maximal power is also affected by the type of muscle action involved and, in particular, the time available to develop force, storage and utilization of elastic energy, interactions of contractile and elastic elements, potentiation of contractile and elastic filaments as well as stretch reflexes. Furthermore, acute changes in the muscle environment (i.e. alterations resulting from fatigue, changes in hormone milieu and muscle temperature) impact the ability to generate maximal power. Development of effective training programmes that enhance maximal muscle power must involve consideration of these factors and the manner in which they respond to training.

Acknowledgements

The authors have no potential conflicts of interest to disclose and no funding was received for this review.

References

1. Gollnick PD, Bayley WM. Biochemical training adaptations and maximal power. In: Jones NL, McCartney N, McComas AJ, editors. Human muscle power. Champaign (IL): Human Kinetics, 1986: 255-67
2. Kraemer WJ, Newton RU. Training for muscular power. Phys Med Rehabil Clin N Am 2000; 11 (2): 341-68
3. Newton RU, Kraemer WJ. Developing explosive muscular power: implications for a mixed method training strategy. Strength Cond J 1994; 16 (5): 20-31
4. Baker D. Comparison of upper-body strength and power between professional and college-aged rugby league players. J Strength Cond Res 2001 Feb; 15 (1): 30-5
5. Sleivert G, Taingahue M. The relationship between maximal jump-squat power and sprint acceleration in athletes. Eur J Appl Physiol 2004 Jan; 91 (1): 46-52
6. Young WB, Newton RU, Doyle TL, et al. Physiological and anthropometric characteristics of starters and non-starters and playing positions in elite Australian rules football: a case study. J Sci Med Sport 2005; 8 (3): 333-45
7. Caiozzo VJ, Perrine JJ, Edgerton VR. Training-induced alterations of the in vivo force-velocity relationship of human muscle. J Appl Physiol 1981; 51 (3): 750-4
8. Thorstensson A, Grimby G, Karlsson J. Force-velocity relations and fiber composition in human knee extensor muscles. J Appl Physiol 1976 Jan; 40 (1): 12-6
9. Widrick JJ, Trappe SW, Costill DL, et al. Force-velocity and force-power properties of single muscle fibers from elite master runners and sedentary men. Am J Physiol 1996 Aug; 271 (2 Pt 1): C676-83
10. Kaneko M, Fuchimoto T, Toji H, et al. Training effect of different loads on the force-velocity relationship and mechanical power output in human muscle. Scand J Med Sci Sports 1983; 5 (2): 50-5
11. Komi PV. Measurement of the force-velocity relationship in human muscle under concentric and eccentric contractions. In: Cerguiglini S, editor. Biomechanics III. Basel: Karger, 1973: 224-9
12. Tihanyi J, Apor P, Fekete G. Force-velocity-power characteristics and fiber composition in human knee extensor muscles. Eur J Appl Physiol Occup Physiol 1982; 48 (3): 331-43
13. Bottinelli R, Pellegrino MA, Canepari M, et al. Specific contributions of various muscle fibre types to human muscle performance: an in vitro study. J Electromyogr Kinesiol 1999; 9 (2): 87-95
14. Hill AV. The heat of shortening and dynamic constants of muscle. Proc R Soc Lond B Biol Sci 1938; 126: 136-95
15. Lieber RL. Skeletal muscle structure, function and plasticity: the physiological basis of rehabilitation. 3rd ed. Philadelphia (PA): Lippincott Williams & Williams, 2010
16. MacIntosh BR, Holash RJ. Power output and force-velocity properties of muscle. In: Nigg BM, MacIntosh BR, Mester J, editors. Biomechanics and biology of movement. Champaign (IL): Human Kinetics, 2000: 193-210
17. Bosco C, Komi PV. Potentiation of the mechanical behavior of the human skeletal muscle through prestretching. Acta Physiol Scand 1979 Aug; 106 (4): 467-72
18. Edgerton VR, Roy RR, Gregor RJ, et al. Morphological basis of skeletal muscle power output. In: Jones NL, McCartney N, McComas AJ, editors. Human muscle power. Champaign (IL): Human Kinetics, Inc., 1986: 43-64
19. Faulkner JA, Claflin DR, McCully KK. Power output of fast and slow fibers from human skeletal muscles. In: Jones NL, McCartney N, McComas AJ, editors. Human muscle power. Champaign (IL): Human Kinetics Inc., 1986: 81-94
20. Herbert RD, Gandevia SC. Changes in pennation with joint angle and muscle torque: in vivo measurements in human brachialis muscle. J Physiol 1995; 484 (Pt 2): 523-32
21. Wickiewicz TL, Roy RR, Powell PL, et al. Muscle architecture and force-velocity relationships in humans. J Appl Physiol 1984 Aug; 57 (2): 435-43
22. Gregor RJ, Edgerton VR, Perine JJ, et al. Torque-velocity relationship and muscle fiber composition in elite female athletes. J Appl Physiol 1979; 47: 388-92
23. Perrine JJ, Edgerton VR. Muscle force-velocity and power-velocity relationships under isokinetic loading. Med Sci Sports 1978; 10 (3): 159-66
24. Perrine JL. The biophysics of maximal muscle power outputs: methods and problems of measurement. In: Jones NL, McCartney N, McComas AJ, editors. Human muscle power. Champaign (IL): Human Kinetics, 1986: 15-25
25. Gordon AM, Huxley AV, Julian FJ. The variation in isometric tension with sarcomere length in vertebrate muscle fibres. J Physiol 1966; 184: 170-92
26. Edman KAP. The relation between sarcomere length and active tension is isolated semitendinosus fibers of the frog. J Physiol 1966; 183 (2): 407-17

27. Lieber RL, Loren GJ, Friden J. In vivo measurement of human wrist estensor muscle sarcomere length changes. J Neurophysiol 1994; 71 (3): 874-81
28. Close RI. Dynamic properties of mammalian skeletal muscles. Physiol Rev 1972; 52: 129-97
29. Komi PV. The stretch-shortening cycle and human power output. In: Jones NL, McCartney N, McComas AJ, editors. Human muscle power. Champaign (IL): Human Kinetics, 1986: 27-40
30. Cavanagh PR, Komi PV. Electromechanical delay in human skeletal muscle under concentric and eccentric contractions. Eur J Appl Physiol Occup Physiol 1979 Nov; 42 (3): 159-63
31. Cavagna GA, Saibene FP, Margaria R. Effect of negative work on the amount of positive work performed by an isolated muscle. J Appl Physiol 1965; 20 (1): 157-8
32. Edman KAP, Elzinga G, Noble MIM. Enhancement of mechanical performance by stretch during tetanic contractions of vertebrate skeletal muscle fibres. J Physiol 1978; 281: 139-55
33. Komi PV, Bosco C. Utilization of stored elastic energy in leg extensor muscles by men and women. Med Sci Sports 1978 Winter; 10 (4): 261-5
34. Cavagna GA, Dusman B, Margaria R. Positive work done by a previously stretched muscle. J Appl Physiol 1968 Jan; 24 (1): 21-32
35. Anderson FC, Pandy MG. Storage and utilization of elastic strain energy during jumping. J Biomech 1993; 26 (12): 1413-27
36. Asmussen E, Bonde-Petersen F. Storage of elastic energy in skeletal muscles in man. Acta Physiol Scand 1974 Jul; 91 (3): 385-92
37. Bosco C, Viitasalo JT, Komi PV, et al. Combined effect of elastic energy and myoelectrical potentiation during stretch-shortening cycle exercise. Acta Physiol Scand 1982; 114: 557-65
38. Asmussen E, Bonde-Petersen F. Apparent efficiency and storage of elastic energy in human muscles during exercise. Acta Physiol Scand 1974; 92 (4): 537-45
39. Gollhofer A, Kyrolainen H. Neuromuscular control of the human leg extensor muscles in jump exercises under various stretch-load conditions. Int J Sports Med 1991; 12 (1): 34-40
40. Takarada Y, Hirano Y, Ishige Y, et al. Stretch-induced enhancement of mechanical power output in human multijoint exercise with countermovement. J Appl Physiol 1997; 83 (5): 1749-55
41. van Zandwijk JP, Bobbert MF, Baan GC, et al. From twitch to tetanus: performance of excitation dynamics optimized for a twitch in predicting tetanic muscle forces. Biol Cybern 1996; 75 (5): 409-17
42. Bobbert MF, Casius LJR. Is the effect of a countermovement on jump height due to active state development? Med Sci Sports Exerc 2005; 37 (3): 440-6
43. Bobbert MF, Gerritsen KG, Litjens MC, et al. Why is countermovement jump height greater than squat jump height? Med Sci Sports Exerc 1996 Nov; 28 (11): 1402-12
44. Mungiole M, Winters JM. Overview: influences of muscle on cyclic and propolsive movements involving the lower limb. In: Winters JM, Woo SLY, editors. Multiple muscle systems biomechanics and movement organisation. New York: Springer-Verlag, 1990: 550-67
45. van Ingen Schenau GJ. An alternate view to the concept of utilisation of elastic energy. Hum Mov Sci 1984; 3: 301-36
46. Bosco C, Montanari G, Tarkka I, et al. The effect of prestretch on mechanical efficiency of human skeletal muscle. Acta Physiol Scand 1987 Nov; 131 (3): 323-9
47. Walshe AD, Wilson GJ, Ettema GJ. Stretch-shorten cycle compared with isometric preload: contributions to enhanced muscular performance. J Appl Physiol 1998 Jan; 84 (1): 97-106
48. van Ingen Schenau GJ, Bobbert MF, de Haan A. Mechanics and energetics of the stretch-shortening cycle: a stimulating discussion. J Appl Biomech 1997; 13: 484-96
49. Cavagna GA, Citterio G. Effect of stretching on the elastic characteristics and the contractile component of frog striated muscle. J Physiol 1974; 239: 1-14
50. Ettema GJ, Huijing PA, de Haan A. The potentiating effect of prestretch on the contractile performance of rat gastrocnemius medialis muscle during subsequent shortening and isometric contractions. J Exp Biol 1992 Apr; 165: 121-36
51. Ettema GJ, van Soest AJ, Huijing PA. The role of series elastic structures in prestretch-induced work enhancement during isotonic and isokinetic contractions. J Exp Biol 1990 Nov; 154: 121-36
52. Huijing PA. Parameter interdependence and success of skeletal muscle modelling. Hum Mov Sci 1995; 14: 443-86
53. Fukunaga T, Kubo K, Kawakami Y, et al. In vivo behavior of human muscle tendon during walking. Proc Biol Sci 2001; 268: 229-33
54. Kawakami Y, Muraoka T, Ito S, et al. In vivo muscle fibre behaviour during counter-movement exercise in humans reveals a significant role for tendon elasticity. J Physiol 2002 Apr 15; 540 (Pt 2): 635-46
55. Kubo K, Kanehisa H, Takeshita D, et al. In vivo dynamics of human medial gastrocnemius muscle-tendon complex during stretch-shortening cycle exercise. Acta Physiol Scand 2000; 170 (2): 127-35
56. Kurokawa S, Fukunaga T, Fukashiro S. Behavior of fascicles and tendinous structures of human gastrocnemius during vertical jumping. J Appl Physiol 2001 Apr; 90 (4): 1349-58
57. Kurokawa S, Fukunaga T, Nagano A, et al. Interaction between fascicles and tendinous structures during counter movement jumping investigated in vivo. J Appl Physiol 2003 Dec; 95 (6): 2306-14
58. Hof AL, Geelen BA, van den Berg J. Calf muscle moment, work and efficiency in level walking; role of series elasticity. J Biomech 1983; 16 (7): 523-37
59. Fukashiro S, Kurokawa S, Hay DC, et al. Comparison of muscle-tendon interaction of human m. gastrocnemius between ankle- and drop-jumping. Int J Sport Health Sci 2005; 3: 253-63
60. Fukashiro S, Hay DC, Nagano A. Biomechanical behavior of muscle-tendon complex during dynamic human movements. J Appl Biomech 2006 May; 22 (2): 131-47
61. Cook CS, McDonagh MJ. Force responses to controlled stretches of electrically stimulated human muscle-tendon complex. Exp Physiol 1995; 80 (3): 477-90

62. Cavagna GA, Mazzanti M, Heglund NC, et al. Storage and release of mechanical energy by active muscle: a non-elastic mechanism? J Exp Biol 1985; 115: 79-87
63. Cavagna GA, Mazzanti M, Heglund NC, et al. Mechanical transients initiated by ramp stretch and release at Po in frog muscle fibres. Am J Physiol 1986 Oct; 251 (4 Pt 1): C571-9
64. Sugi H, Tsuchiya T. Enhancement of mechanical performance in frog muscle fibres after quick increases in load. J Physiol 1981; 319: 239-52
65. Woledge RC, Curtin NA. The efficiency of energy conversion by swimming muscles of fish. In: Sugi H, Pollack GH, editors. Mechanisms of myofilament sliding in muscle contraction. New York: Plenum Press, 1993: 735-47
66. van Ingen Schenau GJ, Bobbert MF, de Haan A. Does elastic energy enhance work and efficiency in the stretch-shortening cycle? J Appl Biomech 1997; 13: 389-415
67. Rassier DE, Herzog W. Force enhancement following an active stretch in skeletal muscle. J Electromyogr Kinesiol 2002 Dec; 12 (6): 471-7
68. Herzog W, Leonard TR, Joumaa V, et al. Mysteries of muscle contraction. J Appl Biomech 2008 Feb; 24 (1): 1-13
69. Joumaa V, Rassier DE, Leonard TR, et al. Passive force enhancement in single myofibrils. Pflugers Arch 2007 Nov; 455 (2): 367-71
70. Joumaa V, Rassier DE, Leonard TR, et al. The origin of passive force enhancement in skeletal muscle. Am J Physiol Cell Physiol 2008 Jan; 294 (1): C74-8
71. Leonard TR, Herzog W. Regulation of muscle force in the absence of actin-myosin based cross-bridge interaction. Am J Physiol Cell Physiol. Epub 2010 Mar 31
72. Schmidt RA, Lee TD. Motor control and learning: a behavioral emphasis. Champaign (IL): Human Kinetics, 2005
73. Dietz V, Schmidtbleicher D, Noth J. Neuronal mechanisms of human locomotion. J Neurophysiol 1979; 42: 1212-22
74. Komi PV, Gollhofer A. Stretch reflexes can have an important role in force enhancement during SSC exercise. J Appl Biomech 1997; 13: 451-60
75. Trimble MH, Kukulka CG, Thomas RS. Reflex facilitation during the stretch-shortening cycle. J Electromyogr Kinesiol 2000; 10 (3): 179-87
76. Komi PV, Nicol C. Shortening cycle of muscle function. In: Zatsiorsky VM, editor. Biomechanics in sport. Oxford: Blackwell Science, 2000: 87-102
77. Nicol C, Komi PV. Significance of passively induced stretch reflexes on Achilles tendon force enhancement. Muscle Nerve 1998; 21 (11): 1546-8
78. Voigt M, Dyhre-Poulsen P, Simonsen EB. Modulation of short latency stretch reflexes during human hopping. Acta Physiol Scand 1998; 163 (2): 181-94
79. Cormie P, McCaulley GO, McBride JM. Power versus strength-power jump squat training: influence on the load-power relationship. Med Sci Sports Exerc 2007; 39 (6): 996-1003
80. Mayhew JL, Ware JS, Johns RA, et al. Changes in upper body power following heavy-resistance strength training in college men. Int J Sports Med 1997; 18: 516-20
81. McBride JM, Triplett-McBride T, Davie A, et al. The effect of heavy- vs. light-load jump squats on the development of strength, power, and speed. J Strength Cond Res 2002; 16 (1): 75-82
82. Stone ME, Johnson R, Carter D. A short term comparison of two different methods of resistive training on leg strength and power. Athl Train 1979; 14: 158-60
83. Stowers T, McMillan J, Scala D, et al. The short-term effects of three different strength-power training methods. NSCA J 1983; 5 (3): 24-7
84. Wilson GJ, Newton RU, Murphy AJ, et al. The optimal training load for the development of dynamic athletic performance. Med Sci Sports Exerc 1993; 25 (11): 1279-86
85. Fitts RH, Widrick JJ. Muscle mechanics: adaptations with exercise-training. Exerc Sport Sci Rev 1996; 24: 427-73
86. Malisoux L, Francaux M, Nielens H, et al. Stretch-shortening cycle exercises: an effective training paradigm to enhance power output of human single muscle fibers. J Appl Physiol 2006; 100 (3): 771-9
87. Widrick JJ, Stelzer JE, Shoepe TC, et al. Functional properties of human muscle fibers after short-term resistance exercise training. Am J Physiol Regul Integr Comp Physiol 2002; 283 (2): R408-16
88. Lionikas A, Li M, Larsson L. Human skeletal muscle myosin function at physiological and non-physiological temperatures. Acta Physiol 2006; 186 (2): 151-8
89. Stienen GJM, Kiers JL, Bottinelli R, et al. Myofibrillar ATPase activity in skinned human skeletal muscle fibres: fibre type and temperature dependence. J Physiol 1996; 493 (2): 299-307
90. Barany M. ATPase activity of myosin correlated with speed of muscle shortening. J Gen Physiol 1967; 50: 197-218
91. Bottinelli R, Betto R, Schiaffino S, et al. Unloaded shortening velocity and myosin heavy chain and alkali light chain isoform composition in rat skeletal muscle fibres. J Physiol 1994; 478: 341-9
92. Bottinelli R, Schiaffino S, Reggiani C. Force-velocity relationship and myosin heavy chain isoform compositions of skinned fibres from rat skeletal muscle. J Physiol 1991; 437: 655-72
93. Close RI. Dynamic proprties of fast and slow skeletal muscles of the rat during development. J Physiol 1964; 173: 74-95
94. Faulkner JA, Clafin DR, McCully KK, et al. Contractile properties of bundles of fiber segments from skeletal muscles. Am J Physiol 1982; 242 (12): C66-73
95. Trappe SW, Gallagher PM, Harber M, et al. Single muscle fibre contractile properties in young and old men and women. J Physiol 2003; 552: 47-58
96. Widrick JJ, Trappe SW, Blaser CA, et al. Isometic force and maximal shortening velocity of single muscle fibers from elite master runners. Am J Physiol 1996; 271: C666-75
97. McCartney N, Heigenhauser GJF, Jones NL. Power output and fatigue of human muscle in maximal cycling exercise. J Appl Physiol 1983; 55: 218-24
98. Costill DL, Daniels J, Evans WJ, et al. Skeletal muscle enzymes and fiber composition in male and female track athletes. J Appl Physiol 1976; 40: 149-54
99. Gollnick PD, Armstrong RB, Saubert CW, et al. Enzyme activity and fiber composition in skeletal muscle of untrained and trained men. J Appl Physiol 1972; 33: 312-9

100. Simoneau JA, Boucard C. Genetic determinism of fiber type proportion in human skeletal muscle. FASEB J 1995; 9: 1091-5
101. Jansson E, Esbjonsson M, Holm H, et al. Increase in the proportion of fast-twitch muscle fibres by sprint training in males. Acta Physiol Scand 1990; 140 (3): 359-63
102. Esbjonsson M, Hellsetn-Westing Y, Balsom PD, et al. Muscle fibre type changes with sprint training: effect of training pattern. Acta Physiol Scand 1993; 149 (2): 245-6
103. Dawson B, Fitzsimons M, Green S, et al. Changes in performance, muscle metabolites, enzymes and fibre types after short sprint training. Eur J Appl Physiol Occup Physiol 1998; 78 (2): 163-9
104. Andersen JL, Klitgaard H, Saltin B. Myosin heavy chain isoforms in single fibres from M. vastus lateralis of sprinters: influence of training. Acta Physiol Scand 1994; 151 (2): 135-42
105. Friedmann B, Kinscherf R, Vorwald S, et al. Muscular adaptations to computer-guided strength training with eccentric overload. Acta Physiol Scand 2004; 182 (1): 77-88
106. Larsson L, Ansved T. Effects of long-term physical training and detraining on enzyme histochemical and functional skeletal muscle characteristics in man. Muscle Nerve 1985; 8 (8): 714-22
107. Andersen LL, Andersen JL, Magnusson SP, et al. Changes in the human muscle force-velocity relationship in response to resistance training and subsequent detraining. J Physiol 2005; 99: 87-94
108. Adams GR, Hather BM, Baldwin KM, et al. Skeletal muscle myosin heavy chain composition and resistance training. J Appl Physiol 1993; 74: 911-5
109. Staron RS, Karapondo DL, Kraemer WJ, et al. Skeletal muscle adaptations during the early phase of heavy-resistance training in men and women. J Appl Physiol 1994; 76: 1247-55
110. Andersen JL, Aagaard P. Myosin heavy chain IIX overshoot in human skeletal muscle. Muscle Nerve 2000; 23 (7): 1095-104
111. Brooks GA, Fahey TD, White TP, et al. Exercise physiology: human bioenergetics and its application. 3rd ed. New York: McGraw Hill, 1999
112. Staron RS, Leonardi MJ, Karapondo DL, et al. Strength and skeletal muscle adaptations in heavy-resitance trained women after detraining and retraining. J Appl Physiol 1991; 70: 631-40
113. Liu Y, Schlumberger A, Wirth K, et al. Different effects on human myosin heavy chain isoform expression: strength vs. combined training. J Appl Physiol 2003; 94 (6): 2282-8
114. Ewing WJ, Wolfe DR, Rogers MA, et al. Effects of velocity of isokinetic training on strength, power and quadriceps muscle fibre characteristics. Eur J Appl Physiol 1990; 61: 159-62
115. McGuigan MR, Sharman MJ, Newton RU, et al. Effect of explosive resistance training on titin and myosin heavy chain isoforms in trained subjects. J Strength Cond Res 2003 Nov; 17 (4): 645-51
116. Malisoux L, Francaux M, Nielens H, et al. Calcium sensitivity of human single muscle fibers following plyometric training. Med Sci Sports Exerc 2006 Nov; 38 (11): 1901-8
117. McComas AJ. Skeletal muscle: form and function. Champaign (IL): Human Kinetics, 1996
118. Bodine S, Roy RR, Meadows DA, et al. Architectural, histochemical and contractile characteristics of a unique biarticular muscle: the cat semitendinosus. J Neurophysiol 1982; 48: 192-201
119. Partridge LD, Benton LA. Muscle, the motor. In: Mountcastle VB, Brooks VB, Greiger SR, editors. Handbook of physiology. Bethesda (MD): American Physiological Society, 1981
120. Shoepe TC, Stelzer JE, Garner DP, et al. Functional adaptability of muscle fibers to long-term resistance exercise. Med Sci Sports Exerc 2003; 35: 944-51
121. Ikai M, Fukunaga T. Calculation of muscle strength per unit cross-sectional area of human muscle by means of ultrasonic measurement. Int Z Angew Physiol 1968; 26: 26-32
122. Maughan RJ, Watson JS, Weir J. Strength and cross-sectional area of human skeletal muscle. J Physiol 1983; 338: 37-49
123. Maughan RJ, Watson JS, Weir J. Muscle strength and cross-sectional area in man: a comparison of strength-trained and untrained subjects. Br J Sports Med 1984; 18 (3): 149-57
124. Jones DA, Rutherford OM, Parker DF. Physiological changes in skeletal muscle as a result of strength training. Q J Exp Physiol 1989; 74 (3): 233-56
125. Rutherford OM, Jones DA. The role of learning and coordination in strength training. Eur J Appl Physiol 1986; 55: 100-5
126. Chapman SJ, Grindrod SR, Jones DA. Cross-sectional area and force production of the quadriceps muscle. J Physiol 1984; 353: 53P
127. Komi PV. Training of muscle strength and power: interaction of neuromotoric, hypertrophic, and mechanical factors. Int J Sports Med 1986; 7 Suppl. 1: 10-5
128. Sale DG. Neural adaptation to resistance training. Med Sci Sports Exerc 1988; 20 (5 Suppl.): 135S-45S
129. Narici MV, Roi GS, Landoni L, et al. Changes in force-cross-sectional area and neural activation during strength training and detraining of the human quadriceps. Eur J Appl Physiol 1989; 59: 310-9
130. Young A. The relative isometric strength of type 1 and type 2 muscle fibres in the human quadriceps. Clin Physiol 1984; 4: 23-32
131. Aagaard P, Andersen JL, Dyhre-Poulsen P, et al. A mechanism for increased contractile strength of human pennate muscle in response to strength training: changes in muscle architecture. J Physiol 2001; 534 (Pt 2): 613-23
132. McCullough P, Maughan RJ, Watson JS, et al. Biomechanical analysis of the knee in relation to measured quadriceps strength and cross sectional area in man [letter]. J Physiol 1984; 346: 60P
133. Trappe SW, Williamson DL, Godard M, et al. Effect of resistance training on single muscle fiber contractile function in older men. J Appl Physiol 2000; 89: 143-52
134. MacDougall JD. Morphological changes in human skeletal muscle following strength training and immobilization. In: Jones NL, McCartney N, McComas AJ, editors.

Human muscle power. Champaign (IL): Human Kinetics, 1986: 269-88

135. MacDougall JD. Hypertrophy or hyperplasia. In: Komi PV, editor. Strength and power in sport. Oxford: Blackwell Scientific Publications, 1992: 230-8
136. Thorstensson A. Muscle strength, fibre types and enzyme activities in man. Acta Physiol Scand Suppl 1976; 443: 1-45
137. MacDougall JD, Elder GCB, Sale DG, et al. Effects of strength training and immobilization on human muscle fibers. Eur J Appl Physiol 1980; 43: 25-34
138. Dons B, Bollerup K, Bonde-Petersen F, et al. The effect of weight-lifting exercise related to muscle fiber composition and muscle cross-sectional area in humans. Eur J Appl Physiol 1979; 40: 95-106
139. Häkkinen K, Komi PV, Tesch PA. Effect of combined concentric and eccentric strength training and detraining on force-time, muscle fibre and metabolic characteristics of leg extensor muscles. Scand J Sports Sci 1981; 3: 50-8
140. Thorstensson A, Hulten B, von Dobeln W, et al. Effect of strength training on enzyme activities and fibre characteristics in human skeletal muscle. Acta Physiol Scand 1976; 96: 392-8
141. Staron RS, Malicky ES, Leonardi MJ, et al. Muscle hypertrophy and fast fiber type conversions in heavy resistance-trained women. Eur J Appl Physiol 1989; 60: 71-9
142. Blazevich AJ, Gill ND, Bronks R, et al. Training-specific muscle architecture adaptation after 5-wk training in athletes. Med Sci Sports Exerc 2003; 35 (12): 2013-22
143. Folland JP, Williams AG. The adaptations to strength training: morphological and neurological contributions to increased strength. Sports Med 2007; 37 (2): 145-68
144. Wernbom M, Augustsson J, Thomee R. The influence of frequency, intensity, volume and mode of strength training on whole muscle cross-sectional area in humans. Sports Med 2007; 37 (3): 225-64
145. Häkkinen K, Komi PV, Alen M. Effect of explosive type strength training on isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of leg extensor muscles. Acta Physiol Scand 1985; 125 (4): 587-600
146. Häkkinen K, Komi PV, Alen M, et al. EMG, muscle fibre and force production characteristics during a 1 year training period in elite weight-liters. Eur J Appl Physiol 1987; 56: 419-27
147. Häkkinen K, Pakarinen A, Kyröläinen H, et al. Neuromuscular adaptations and serum hormones in females during prolonged power training. Int J Sports Med 1990; 11 (2): 91-8
148. Komi PV, Karlsson J, Tesch P, et al. Effects of heavy resistance and explosive type strength training methods on mechanical, functional and metabolic aspects of performance. In: Komi PV, editor. Exercise and sport biology. Champaign (IL): Human Kinetics, 1982: 90-102
149. Kyrolainen H, Avela J, McBride JM, et al. Effects of power training on muscle structure and neuromuscular performance. Scand J Med Sci Sports 2005; 15 (1): 58-64
150. Potteiger JA, Lockwood R, Haub M, et al. Muscle power and fiber characteristics following 8 weeks of plyometric training. J Strength Cond Res 1999; 13: 275-9
151. Vissing K, Brink M, Lonbro S, et al. Muscle adaptations to plyometric vs. resistance training in untrained young men. J Strength Cond Res 2008 Nov; 22 (6): 1799-810
152. Kubo K, Morimoto M, Komuro T, et al. Effects of plyometric and weight training on muscle-tendon complex and jump performance. Med Sci Sports Exerc 2007; 39 (10): 1801-10
153. Wickiewicz TL, Roy RR, Powell PL, et al. Muscle architecture of the human lower limb. Clin Orthop Relat Res 1983; 179: 275-83
154. Sacks RD, Roy RR. Architecture of the hind limb of muscle of cats: functional significance. J Morphol 1982; 173: 185-95
155. Spector SA, Gardiner PF, Zernicke RF, et al. Muscle architecture and the force-velocity characteristics of cat soleus and medial gastrocnemius: impliations for motor control. J Neurophysiol 1980; 44: 951-60
156. Abe T, Fukashiro S, Harada Y, et al. Relationship between sprint performance and muscle fascicle length in female sprinters. J Physiol Anthropol 2001; 20 (2): 141-7
157. Kumagai K, Abe T, Brechue WF, et al. Sprint performance is related to muscle fascicle length in male 100-m sprinters. J Appl Physiol 2000 Mar; 88 (3): 811-6
158. Abe T, Kumagai K, Brechue WF. Muscle fascicle length is greater in sprinters than long-distance runners. Med Sci Sports Exerc 2000; 32: 1125-9
159. Butterfield TA, Leonard TR, Herzog W. Differential serial sarcomere number adaptations in knee extensor muscles of rats is contraction type dependent. J Appl Physiol 2005; 99: 1352-8
160. Lynn R, Morgan DL. Decline running produces more sarcomeres in rat vastus intermedius muscle fibers than does incline running. J Appl Physiol 1994; 79: 1439-44
161. Lynn R, Talbot JA, Morgan DL. Differences in rat skeletal muscles after incline and decline running. J Appl Physiol 1998; 85: 98-104
162. Blazevich AJ, Cannavan D, Coleman DR, et al. Influence of concentric and eccentric resistance training on architectural adaptation in human quadriceps muscles. J Appl Physiol 2007; 103 (5): 1565-75
163. Reeves ND, Narici MV, Maganaris CN. In vivo human muscle structure and function: adaptations to resistance training in old age. Exp Physiol 2004; 89 (6): 675-89
164. Seynnes OR, de Boer M, Narici MV. Early skeletal muscle hypertrophy and architectural changes in response to high-intensity resistance training. J Appl Physiol 2007; 102: 368-73
165. Alegre LM, Jimenez F, Gonzalo-Orden JM, et al. Effects of dynamic resistance training on fascicle length and isometric strength. J Sports Sci 2006; 24 (5): 501-8
166. Kawakami Y, Abe T, Kuno SY, et al. Training-induced changes in muscle architecture and specific tension. Eur J Appl Physiol 1995; 72 (1-2): 566-73
167. Rutherford OM, Jones DA. Measurement of fibre pennation using ultrasound in the human quadriceps in vivo. Eur J Appl Physiol Occup Physiol 1992; 65 (5): 433-7
168. Blazevich AJ, Gill ND, Deans N, et al. Lack of human muscle architectural adaptation after short-term strength training. Muscle Nerve 2007; 35 (1): 78-86

169. Blazevich AJ, Sharp NC. Understanding muscle architectural adaptation: macro- and micro-level research. Cells Tissues Organs 2005; 181 (1): 1-10
170. Huijing PA. Architecture of the human gastrocnemius muscle and some functional consequences. Acta Anat (Basel) 1985; 123: 101-7
171. Powell P, Roy RR, Kanim P, et al. Predictability of skeletal muscle tension from architectural determinations in guinea pig hindlimbs. J Appl Physiol 1984; 57: 1715-21
172. Gans C. Fiber architecture and muscle function. Exerc Sport Sci Rev 1982; 10: 160-207
173. Muhl ZF. Active length-tension relation and the effect of muscle pinnation on fiber lengthening. J Morphol 1982; 173: 285-92
174. Kawakami Y, Takashi A, Fukunaga T. Muscle-fiber pennations angles are greater in hypertrophied than in normal muscles. J Appl Physiol 1993; 74 (6): 2740-4
175. Kawakami Y, Abe T, Kanehisa H, et al. Human skeletal muscle size and architecture: variability and interdependence. Am J Hum Biol 2006 Nov-Dec; 18 (6): 845-8
176. Blazevich AJ, Giorgi A. Effect of testosterone administration and weight training on muscle architecture. Med Sci Sports Exerc 2001; 33 (10): 1688-93
177. Westh E, Kongsgaard M, Bojsen-Møller J, et al. Effect of habitual exercise on the structural and mechanical properties of human tendon, in vivo, in men and women. Scand J Med Sci Sports 2008 Feb; 18 (1): 23-30
178. Kubo K, Ishida Y, Suzuki S, et al. Effects of 6 months of walking training on lower limb muscle and tendon in elderly. Scand J Med Sci Sports 2008 Feb; 18 (1): 31-9
179. Kubo K, Kanehisa H, Kawakami Y, et al. Elasticity of tendon structures of the lower limbs in sprinters. Acta Physiol Scand 2000 Feb; 168 (2): 327-35
180. Bojsen-Møller J, Magnusson SP, Rasmussen LR, et al. Muscle performance during maximal isometric and dynamic contractions is influenced by the stiffness of the tendinous structures. J Appl Physiol 2005 Sep; 99 (3): 986-94
181. Henneman E, Clamann HP, Gillies JD, et al. Rank order of motoneurons within a pool, law of combination. J Neurophysiol 1974; 37: 1338-49
182. Henneman E, Somjen G, Carpenter DO. Functionl significance of cell size in spinal motoneurons. J Neurophysiol 1965; 28: 560-80
183. Burke RE. Motor units: anatomy, physiology, and functional organization. In: Brooks VB, editor. Handbook of physiology: section I – the nervous system volume II. Washington, DC: American Physiological Society, 1981: 345-422
184. Milner-Brown HS, Stein RB. The relationship between the surface electromyogram and muscular force. J Physiol 1975; 246: 549-69
185. Desmedt JE, Godaux E. Ballistic contractions in man: characteristic recruitment pattern of single motor units of the tibialis anterior muscle. J Physiol 1977; 264: 673-93
186. Desmedt JE, Godaux E. Ballistic contractions in fast or slow human muscles: discharge patterns of single motor units. J Physiol 1978; 285: 185-96
187. van Cutsem M, Duchateau J, Hainaut K. Changes in single motor unit behaviour contribute to the increase in contraction speed after dynamic training in humans. J Physiol 1998 Nov 15; 513 (Pt 1): 295-305
188. Enoka RM. Morphological features and activation patterns of motor units. J Clin Neurophysiol 1995; 12: 538-59
189. Enoka RM, Fuglevand AJ. Motor unit physiology: some unresolved issues. Muscle Nerve 2001; 24: 4-17
190. Sale DG. Neural adaptations to strength training. In: Komi PV, editor. Strength and power in sport. 2nd ed. Oxford: Blackwell Science, 2003: 281-313
191. Bigland B, Lippold OCJ. Motor unit activity in the voluntary contractions of human muscle. J Physiol 1954; 125: 322-35
192. Belanger AY, McComas AJ. Extent of motr unit activation during effort. J Appl Physiol 1981; 51 (5): 1131-5
193. Bellemare F, Woods JJ, Johansson R, et al. Motor-unit discharge rates in maximal voluntary contractions of three human muscles. J Neurophysiol 1983; 50: 1380-92
194. Grimby L, Hannerz J, Hedman B. The fatigue and voluntary discharge properties of single motor units in man. J Physiol 1981; 316: 545-54
195. Shield A, Zhou S. Assessing voluntary muscle activation with the twitch interpolation technique. Sports Med 2004; 34 (4): 253-367
196. Davies J, Parker DF, Rutherford OM, et al. Changes in strength and cross sectional area of the elbow flexors as a result of isometric strength training. Eur J Appl Physiol Occup Physiol 1988; 57 (6): 667-70
197. Garfinkel S, Cafarelli E. Relative changes in maximal force, EMG, and muscle cross-sectional area after isometric training. Med Sci Sports Exerc 1992; 24 (11): 1220-7
198. Herbert RD, Dean C, Gandevia SC. Effects of real and imagined training on voluntary muscle activation during maximal isometric contractions. Acta Physiol Scand 1998; 163 (4): 361-8
199. Harridge SD, Kryger A, Stensgaard A. Knee extensor strength, activation, and size in very elderly people following strength training. Muscle Nerve 1999; 22 (7): 831-9
200. Brown AB, McCartney N, Sale DG. Positive adaptations to weight-lifting training in the elderly. J Appl Physiol 1990; 69 (5): 1725-33
201. Jones DA, Rutherford OM. Human muscle strength training: the effects of three different regimes and the nature of the resultant changes. J Physiol 1987; 391: 1-11
202. Sale DG, Martin JE, Moroz DE. Hypertrophy without increased isometric strength after weight training. Eur J Appl Physiol Occup Physiol 1992; 64 (1): 51-5
203. Carolan B, Cafarelli E. Adaptations in coactivation after isometric resistance training. J Appl Physiol 1992; 73: 911-7
204. Babault N, Pousson M, Ballay Y, et al. Activation of human quadriceps femoris during isometric, concentric, and eccentric contractions. J Appl Physiol 2001; 91 (6): 2628-34
205. Duchateau J, Hainaut K. Mechanisms of muscle and motor unit adaptation to explosive power training. In: Komi PV, editor. Strength and power in sport. 2nd ed. Oxford: Blackwell Science, 2003: 315-29
206. Kraemer WJ, Fleck SJ, Evans WJ. Strength and power training: physiological mechanisms of adaptation. Exerc Sport Sci Rev 1996; 24: 363-97

207. Smith JL, Betts B, Edgerton VR, et al. Rapid ankle extension during paw shakes: selective recruitment of fast ankle extensors. J Neurophysiol 1980; 43: 612-20
208. Enoka RM. Eccentric contractions require unique activation strategies by the nervous system. J Appl Physiol 1996 Dec; 81 (6): 2339-46
209. Nardone A, Romano C, Schieppati M. Selective recruitment of high-threshold human motor units during voluntary isotonic lengthening of active muscles. J Physiol 1989; 409: 451-71
210. Feiereisen P, Duchateau J, Hainaut K. Motor unit recruitment order during voluntary and electrically induced contractions in the tibialis anterior. Exp Brain Res 1997; 114: 117-23
211. Hannerz J. Discharge properties of motor units in relation to recruitment order in voluntary contraction. Acta Physiol Scand 1974; 91 (3): 374-85
212. Zehr EP, Sale DG. Ballistic movement: motor control and muscle activation. Can J Appl Physiol 1994; 19: 363-78
213. Moritani T. Motor unit and motorneurone excitability during explosive movement. In: Komi PV, editor. Strength and power in sport. Oxford: Blackwell Science, 2003: 27-49
214. Miller RG, Mirka A, Maxfield M. Rate of tension development in isometric contractions of a human hand muscle. Exp Neurol 1981; 72: 267-85
215. Cracraft JD, Petajan JH. Effect of muscle training on the pattern of firing of single motor units. Am J Phys Med 1977; 56: 183-93
216. Leong B, Kamen G, Patten C, et al. Maximal motor unit discharge rates in the quadriceps muscles of older weight lifters. Med Sci Sports Exerc 1999; 31: 1638-44
217. Patten C, Kamen G, Rowland DM. Adaptations in maximal motor unit discharge rate to strength training in young and older adult. Muscle Nerve 2001; 24: 542-50
218. Kamen G, Knight CA. Training-related adaptations in motor unit discharge rate in young and older adults. J Gerontol A Biol Sci Med Sci 2004; 59 (12): 1334-8
219. Kamen G, Knight CA, Laroche DP, et al. Resistance training increases vastus lateralis motor unit firing rates in young and old adults [letter]. Med Sci Sports Exerc 1998; 30 Suppl.: S337
220. Saplinskas JS, Chobotas MA, Yashchaninas II. The time of completed motor acts and impulse activity of single motor units according to the training level and sport specialization of tested persons. Electromyogr Clin Neurophysiol 1980; 20: 529-39
221. Milner-Brown HS, Stein RB, Lee RG. Synchronization of human motor units: possible roles of exercise and supraspinal reflexes. Electroencephalogr Clin Neurophysiol 1975; 38 (3): 245-54
222. Semmler JG, Enoka RM. Neural contributions to the changes in muscle strength. In: Zatsiorsky VM, editor. Biomechanics in sport: the scientific basis of performance. Oxford: Blackwell Science, 2000: 3-20
223. Semmler JG. Motor unit synchronization and neuromuscular performance. Exerc Sport Sci Rev 2002; 30 (1): 8-14
224. Mellor R, Hodges P. Motor unit synchronization between medial and lateral vasti muscles. Clin Neurophysiol 2005; 116 (7): 1585-95
225. Lind AR, Petrofsky JS. Isometric tension from rotary stimulation of fast and slow cat muscle. Muscle Nerve 1978; 1: 213-8
226. Rack PM, Westbury DR. The effects of length and stimulus rate on tension in the isometric cat soleus muscle. J Physiol 1969; 204: 443-60
227. Semmler JG, Nordstrom MA. Motor unit discharge and force tremor in skill- and strength-trained individuals. Exp Brain Res 1998; 119 (1): 27-38
228. Yue G, Fuglevand AJ, Nordstrom MA, et al. Limitations of the surface electromyography technique for estimating motor unit synchronization. Biol Cybern 1995; 73 (3): 223-33
229. Behm DG. Neuromuscular implications and applications of resistance training. J Strength Cond Res 1995; 9 (4): 264-74
230. Ostering LR, Hamill J, Corcos DM, et al. EMG patterns accompanying isokinetic exercise under varying speed and sequencing conditions. Am J Phys Med 1984; 63: 289-97
231. Gordon J, Ghez C. EMG patterns in antagonist muscles during isometric contractions in man: relation to response dynamics. Exp Brain Res 1984; 55: 167-71
232. Karst G, Hazan Z. Antagonist muscle activity during forearm movements under varying kinematic and loading conditions. Exp Brain Res 1987; 67: 391-401
233. Baratta R, Solomonow M, Zhou BH, et al. Muscular coactivation: the role of the antagonist musculature in maintaining knee stability. Am J Sports Med 1988; 16 (2): 113-22
234. Aagaard P, Simonsen EB, Andersen JL, et al. Antagonist muscle coactivation during isokinetic knee extension. Scand J Med Sci Sports 2000; 10 (2): 58-67
235. Kellis E, Baltzopoulos V. The effects of antagonist moment on the resultant knee joint moment during isokinetic testing of the knee extensors. Eur J Appl Physiol Occup Physiol 1997; 76 (3): 253-9
236. Milner TE, Cloutier C, Leger AB, et al. Inability to activate muscles maximally during cocontraction and the effect of joint stiffness. Exp Brain Res 1995; 107: 293-305
237. Carpentier A, Duchateau J, Hainaut K. Velocity-dependent muscle strategy during plantarflexion in humans. J Electromyogr Kinesiol 1996; 6: 1-11
238. Fitts RH. The cross-bridge cycle and skeletal muscle fatigue. J Appl Physiol 2008; 104 (2): 551-8
239. Allen DG, Lamb GD, Westerblad H. Skeletal muscle fatigue: cellular mecanisms. Physiol Rev 2008; 88 (1): 287-332
240. Karatzaferi C, Franks-Skiba K, Cooke R. Inhibition of shortening velocity of skinned skeletal muscle fibers in conditions that mimic fatigue. Am J Physiol Regul Integr Comp Physiol 2008; 294: R948-55
241. Kraemer WJ, Ratamess NA. Hormonal responses and adaptations to resistance exercise and training. Sports Med 2005; 35 (4): 339-61
242. Kraemer WJ, Ratamess NA. Endocrine responses and adaptations to strength and power training. In: Komi PV, editor. Strength and power in sport. Oxford: Blackwell Scientific Publications, 1992
243. Hamdi MM, Mutungi G. Dihydrotestosterone activates the MAPK pathway and modulates maximum isometric

force through the EGF receptor in isolated intact mouse skeletal muscle fibres. J Physiol 2010; 588 (3): 511-25

244. De Ruiter CJ, Jones DA, Sargeant AJ, et al. Temperature effect on the rates of isometric force development and relaxation in the fresh and fatigued human adductor pollicis muscle. Exp Physiol 1999; 84: 1137-50
245. Ranatunga KW. Temperature-dependence of shortening velocity and rate of isometric tension development in rat skeletal muscle. J Physiol 1985; 329: 465-83
246. De Ruiter CJ, De Haan A. Temperature effect on the force/velocity relationship of the fresh and fatigued human adductor pollicis muscle. Pflügers Arch 2000; 440: 163-70
247. Rall JA, Woledge RC. Influence of temperature on mechanics and energetics of muscle contraction. Am J Physiol 1990; 259: R197-203
248. Bennett AF. Thermal dependence of muscle function. Am J Physiol 1984; 247: R217-29
249. Ferretti G. Cold and muscle performance. Int J Sports Med 1992; 13 Suppl. 1: 185S-92S

Correspondence: Dr *Prue Cormie*, School of Exercise and Biomedical Health Sciences, Edith Cowan University, 270 Joondalup Drive, Joondalup, WA 6027, Australia.
E-mail: p.cormie@ecu.edu.au