Developing maximal neuromuscular power: Part 1 – Biological basis of maximal power production

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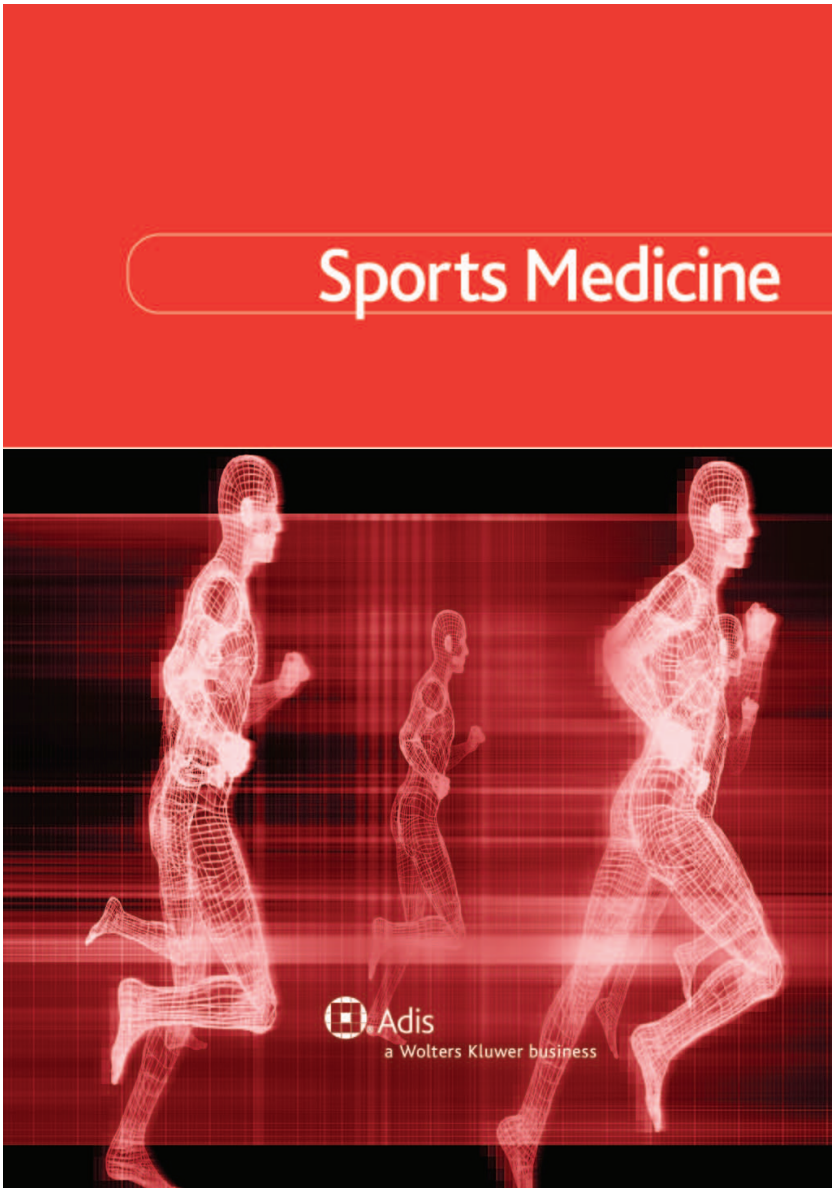

REVIEW ARTICLE

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Developing Maximal Neuromuscular Power

Part 1 – Biological Basis of Maximal Power Production

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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

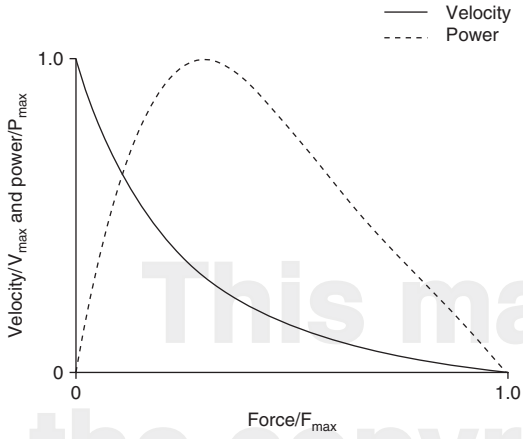

Fig. 1. The force-velocity and force-power relationships for concentric contractions of skeletal muscle. Force, velocity and power are normalized to the maximum isometric force (F_{max}), maximum velocity of shortening (V_{max}) and maximum power output (P_{max}), respectively.

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 submaximal 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.

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