The Effect of Complex Training on Horizontal Power Production in Rugby Union Players.

A thesis presented in partial fulfilment of the requirements for the degree of Master of Science at Massey University

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2004

Abstract

The use of strength and power training regimes is common place among elite and recreational athletes. However, the application of such methods as direct determinants of improvement in sporting performance is a controversial and much debated topic because the degree of transfer from the training exercise to the sporting application is unknown. In recent years combining strength and sport specific training methods into one training session (complex training) has been promoted as a method to enhance training transfer. The purpose of this project was to examine the effect of complex training on horizontal power production in rugby players.

9 participants completed two four week phases of training (complex and standard) in a randomized order. Participant performance in 5RM squat, horizontal force and horizontal power was tested prior to and at the end of each training phase.

A number of significant improvements were observed following complex training: maximum slope of the horizontal force curve increased by 12.29 ±33.59%, maximum power increased by 15.13 ±7.49%, width of the power curve increased by 28.30 ±18.16%, and maximum velocity during the horizontal power test improved by 20.63 ±14.21%. The improvements were significantly different from the respective standard training measures ($p \leq .05$).

It is concluded that power gains were a product of an enhanced ability to produce force at higher velocities. No significant weight gain or significant improvement in 5RM force production was associated with the improvement in maximum power. Therefore it is inferred that neural mechanisms accounted for the difference following complex training. The results presented here suggest that complex training not only improves horizontal power production but also transfers performance improvements to an untrained task by improving the rate of force development in the horizontal force condition. It appears that the complex training regime has in some way created a persistent change in the control mechanisms regulating the performance of both the horizontal strength and power conditions.

Acknowledgements

The completion of this Masters project required the support and assistance of many people; I would like to thank everyone who has offered their wisdom, effort and time. There are a number of people I would like to thank individually:

Dr. Alan Walmsley without whom I doubt I would have started let alone completed my project. Alan's support and academic guidance was, in many instances, the reason I was able to progress in the face of numerous barriers.

Emma Hawke who generously gave her time to assist me in data collection. The majority of the training sessions for participants started at 6am. Cold, early mornings are not the most pleasant time to be in the lab, thank you Emma.

The Bessie Thoms Trust for their scholarship support.

Thank you to my Mother, Father and Sister who have supported me in countless ways since I decided to give up joinery (among other things) and pursue an academic career.

Finally, thank you to Katie, for following me around the country and enduring the challenges that I tend to present. I am truly happy that I am now able to provide you the opportunity to pursue your own passion.

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1 Introduction

Action in sport is a direct manifestation of the only function of muscle fibres, namely the generation of force. The ability to produce force is affected by numerous factors; a strength and conditioning training programme is one such factor.

Muscular strength and conditioning programmes and structured training programs have been used to enhance sporting performance since the ancient Olympic Games. This implies that some methods of strength and power development have been known for many years. However, the recognition of such methods as direct determinants of improvement in sporting performance is still in question. This is largely because the degree of transfer from the training application to the sporting situation is unknown. The following review discusses the methods of strength and power development, the mechanisms involved in them, and their applicability to the field of sport performance enhancement.

1.1 Muscular strength

1.1.1 Definition of strength

"Muscle fibres, the cells of skeletal muscles, have but one function: to generate force." (Billeter & Hoppler, 1992).

Muscular strength may be defined as the force or torque a muscle can exert against a resistance in one maximal effort (Potach & Chu, 2000; Wilson, 1994). The mechanism of muscular contraction is described by the sliding filament theory. In this context, force production is affected by several factors, including:

1) the release of calcium into the myofibril, which is governed by the innervation of the muscle;

2) the number of actin and myosin cross bridge interactions at a given position and time, determined by the length-tension and force-velocity relationships;

3) the physiological cross sectional area of the muscle; and

4) the anatomical structure of the muscle, that is the angle of muscle fibres relative to the tendon and line of force.

The sliding filament theory of muscular contraction proposes that, after stimulation of the muscle fibre via the motor neuron, calcium ions are released by the sarcoplasmic reticulum. The calcium binds to troponin, which in turn creates conformational changes in tropomyosin allowing binding of the myosin head to the actin filament. Once bound, the myosin head bends drawing the actin filament towards the centre of the sarcomere; this is the power stroke. This process is facilitated by the cleaving of a phosphate group

from ATP within the myosin head, allowing binding to take place and leading to the power stroke (Billeter & Hoppler, 1992).

The amount of calcium released is related to the frequency of stimulation from the innervating motor neuron. It is recognised that motor neurons stimulate more than one muscle fibre, and so the term motor unit (MU) incorporating the motor neuron and all the fibres it innervates is used to describe the functional unit of muscle action. MUs are recruited in an all or none fashion, meaning that all fibres within the MU contract simultaneously to produce force. Each individual stimulation and contraction of the MU is referred to as a twitch. Each single twitch produces a force pulse, and if stimulation is repeated prior to relaxation of the MU the force pulses of the two twitches summate creating a greater force than the single twitch. By decreasing the interval between twitches summation of force is enhanced and when stimulation is at maximum frequency twitches become indistinguishable in the force trace. This is a fused tetanus, and produces the largest amount of force that the MU can develop. MUs themselves may be broadly categorised into two classes or types; type I or slow twitch, and type II or fast twitch. Each type is identified using physiological characteristics; type I by its aerobic qualities and relatively low force capabilities, and type II by its anaerobic qualities and its relatively high force capabilities. All fibres within a MU are of the same type.

The force that an individual sarcomere can produce is proportional to the number of actin and myosin filaments interacting at a given instant. The length-tension relationship indicates that the optimal position (optimal interaction of actin and myosin) for a sarcomere to produce maximal force is at its resting length; when the sarcomere is either shortened or lengthened its force producing capacity will be diminished (Edman, 1992).

This implies that the length of the muscle itself will have a direct bearing on the force production possible at any given instant. The force a muscle can produce is also governed by the force-velocity relationship which relates the velocity of concentric contraction to force production. Contraction velocity will be discussed at a later stage.

Muscle strength is proportional to the physiological cross sectional area (pCSA) of the muscle (Hunter, 2000; Roy & Egderton, 1992; Wilson, 1994). The greater the pCSA the greater number of sarcomeres in parallel and, consequently, the greater the maximum possible force production (Hunter, 2000). Speed of contraction of a muscle is proportional to the number of sarcomeres in series. Because type II muscle fibres have relatively short sarcomeres compared to type I, they will have more sarcomeres in series for a given length of muscle and consequently will contract faster than type I fibres. Therefore, type II fibres have the greatest potential for both maximal force production and velocity of shortening.

The morphology of muscle (such as pennation) affects the force that a muscle can produce. The term pennation refers to the angle between the line of action of the muscle fibres and the line of the tendon to which they attach. In general, it is considered that a pennate muscle will have a greater number of sarcomeres in parallel compared with a non-pennate muscle and so it will have the potential to produce greater force. However, the number of sarcomeres in parallel must be great enough to compensate for the reduction in muscular force as a consequence of the oblique alignment of the muscle to the tendon. Therefore, if a pennate muscle has a sufficiently large pCSA it will allow the development of greater force than a non-pennate muscle of similar size. The morphology of muscle, such as fibre arrangement, origin and insertion, appears to be a genetic predisposition and so is unable to be modified by training. Consequently,

variation in muscle structure may account for differences in strength seen in individuals of similar physical stature with a similar proportion of type II MUs within a given muscle (Hunter, 2000; Roy & Egderton, 1992).

Therefore, we may conclude that muscular strength is dependent on the proportion of high force producing MUs within a muscle, the neural activation of those MUs, the muscle pCSA, and the structure of the muscle. As a result, hypertrophy of muscle, increasing the number of MUs recruited, and increasing the rate of MU recruitment will enhance muscle strength. Hypertrophy and neural adaptation account for strength gains when muscle is subjected to a training stimulus. These two categories of adaptations occur within well documented time frames. It is reported that in the first 6-8 weeks of training neural adaptation precedes muscular adaptation; following this, muscular adaptation accounts for the majority of strength improvements. However, it appears possible that our ability to gain strength via muscular adaptation may be limited by genetic factors and so performance improvements beyond a certain point may only be gained by neural adaptations; that is learning to use what we have more effectively.

1.1.2 Muscular adaptation to strength training.

By adhering to the general principles of training, such as progressive overload, which allow muscle to be repeatedly and appropriately stimulated, muscle may be made to adapt and become stronger. The primary mechanism of muscular adaptation is hypertrophy. Because Type II muscle fibres have a greater ability to hypertrophy they have a greater potential for strength adaptation than Type I muscle fibres (Goldspink, 1992; Goldspink & Harridge, 2003). When considering the method of recruitment of MUs (the size principle), training loads applied should be sufficiently heavy to allow full recruitment of all type II MUs in the muscle being trained.

1.1.2.1 Hypertrophy

As the mechanisms of muscular hypertrophy are well known they will only be briefly discussed here; for a more detailed description see Goldspink (1992). Increase of fibre CSA is reported to be associated with a large increase in the myofibrillar content of the fibre as a consequence of myofibrils undergoing longitudinal splitting into two or more myofibrils (Goldspink, 1992). This process is reported to be stimulated by micro trauma within the muscle fibre caused by overload (performing heavily loaded contractions to fatigue). Repair of this micro trauma proceeds by two mechanisms; an increase in the rate of protein synthesis, or a decrease in the rate of protein breakdown (Goldspink, 1992; Goldspink & Harridge, 2003). The myofibril increases in size because of the increased volume of the myofilaments actin and myosin, this increases the ability of the sarcomere to produce force and, at some stage, the force produced is large enough to create a "snap" of the z-discs effectively subdividing the myofibril. Hypertrophy of the muscle fibre may take place independent of any change in the size of the entire muscle because the increase in fibre CSA occurs at the expense of extra-cellular space (Goldspink & Harridge, 2003). It is important not to confuse myofibrilar proliferation with an increase in the number of muscle fibres (hyperplasia).

1.1.2.2 Hyperplasia

Hyperplasia is an increase in the number of muscle fibres and has not been reported to take place in human muscle as a result of training interventions. This is not to say that the mechanism for hyperplasia is not present. The presence of satellite cells, which appear to function as reserve muscle cells, could provide such a mechanism (MacDougall et al., 1992). Cadaver studies have shown that the muscles of dominant limbs contain a greater number of muscle fibres than those of the non-dominant limb (McCall et al., 1996; Sjostrom et al., 1990). This implies that chronic training or use of

musculature induces hyperplasia as a means of increasing pCSA. The lack of support for hyperplasia in the current literature may be a consequence of the mismatch between the time course of hyperplasia and the average training intervention (Sjostrom et al., 1990). That is, the typical duration of training interventions used in scientific research is approximately 12 weeks, but the time course required to observe hyperplasia may be years rather than weeks or months. This then limits our means of enhancing pCSA in the short to medium term to the mechanisms of hypertrophy, which, in turn indicates that the principles of hypertrophy (muscular adaptation to resistance training) must be considered in any discussion of strength adaptation.

1.1.2.3 Morphological Changes

Strength training is reported to induce shifts in fibre type composition (Tesch & Alkner, 2003). To understand this concept we must consider the Type II muscle fibres to exist as two sub-types or isoforms. One method of determining the isoform of muscle is to measure the myosin heavy chain (MHC) content. The isoforms are usually labelled Type I, Type IIa and Type IIb. Strength training induces a shift from the expression of the Type IIb isoform towards the Type IIa (Goldspink & Harridge, 2003; Kraemer, 2000; Schiaffino & Reggiani, 1994). However, there is no report of change in fibre type (Type II to Type I, or Type I to Type II) following resistance training. It is suggested that the Type IIb isoform may be the default Type II fibre expression in unconditioned/untrained muscle, therefore, any training will promote a shift away from the Type IIb isoform (Tesch & Alkner, 2003). This is supported by an investigation into detraining that shows an isoform shift back towards Type IIb following a period of detraining (Anderson & Aagaard, 2000). This implies that the shift towards the Type IIa isoform following training may be an adaptation to allow more efficient force production. Therefore, the individual becomes more efficient a lifting an absolute load

following resistance training. Another way to think of this is to consider that the absolute load which was once maximal becomes sub-maximal, a greater load must now be lifted to achieve maximal activation of MUs. The physical outcome of this is increased strength. There may be implications of this shift for strength and power performance, however, these are yet to be explored and are beyond the scope of this study.

1.1.2.4 Cardiovascular Changes

The two most commonly reported cardiovascular changes following strength training are increased left ventricular wall thickness and decreased capillary density. There are other cardiovascular changes that occur as a response to strength training, but they appear to have little effect on strength or power performance (Fleck, 2003).

When considering strength training regimes it is clear that improvement in strength as a function of muscular adaptation is a product of muscular hypertrophy and therefore, increase in pCSA.

1.1.3 Neural component of strength adaptation

Although strength is proportional to pCSA, increasing pCSA alone does not account for all strength gains; the non-hypertrophy gains are partly attributable to neural adaptation. Therefore, strength of a muscle is determined both by muscle size and the ability of the nervous system to activate the muscle appropriately. There appears to be a complex interaction of neural drive to the agonists, synergists, and antagonists involved in a movement (Sale, 2003; Wilson, 1994). Whatever interactions are present MU recruitment appears to be controlled, in the first instance, by the size principle.

1.1.3.1 Recruitment of Motor Units

The size principle that governs the recruitment pattern of MUs may be described as follows; as the demand on muscle to produce muscular tension increases, MUs are recruited in order of increasing size. As described previously type I MUs tend to be smaller, low force producing MUs, while type II MUs tend to be larger, higher force producing MUs. Consequently, increasing muscle force is produced by initially recruiting small, low threshold MUs, then increasing the firing rate of those MUs, followed by the progressive recruitment of larger MUs, and finally by increasing the firing rate of the large MUs. Theoretically a voluntary contraction that incorporates the maximal number of MUs (high and low threshold) will produce the largest force possible. However, research has shown that maximal voluntary contraction (MVC) does not produce the maximum force possible from a given muscle (Belanger & McComas, 1981). By superimposing electrical stimulation over MVC (known as the interpolated twitch method) additional force can be produced (Belanger & McComas, 1981). This suggests that improvement of MU recruitment could enhance strength. This is supported by observations of increased agonist activation and decreased difference between MVC force and force produced with stimulation superimposed over MVC following a training protocol (Hakkinen et al., 1992; Moritani & deVries, 1979). This suggests regulatory mechanisms are present governing the magnitude of voluntary production of force, and that training modifies these mechanisms allowing increased activation of MUs involved in producing the intended movement (agonists), and decreased activation of MUs inhibiting the intended movement (antagonists).

1.1.3.2 Disinhibition

Movement is essentially the sum of force produced in the line of the intended action by the agonist and any opposing force produced by the antagonist, that is, the true

expression of agonist force may be inhibited by the level of antagonist force. Exposure to high levels of tension has been reported to reduce the sensitivity of Golgi Tendon Organs (the receptors which regulate muscle tension). This creates a disinhibitory effect allowing greater agonist activation and, consequently, greater force production. Disinhibition can occur in the absence of muscular hypertrophy (Wilson, 1994). Decreasing antagonist co-activation allows a truer expression of agonist force. Although the concept of reduced sensitivity of Golgi Tendon Organs appears to be widely accepted some authors reject it as an adaptation to strength training citing lack of evidence (Chalmers, 2002). Disinhibition theoretically allows increased agonist activation and increased muscle force which may, in some instances, be brought about via the synchronization of MU recruitment.

1.1.3.3 Motor Unit Synchronization

Synchronization of MUs refers to the coordination of different MUs within a muscle creating a synchronised firing pattern. Strength training reportedly increases synchronization of MUs resulting in increased force production (Wilson, 1994). This coordination enables greater force to be expressed through the combined effect of numerous MUs contracting simultaneously. Not only is the force of a contraction increased but also the Rate of Force Development (RFD) is enhanced.

1.1.3.4 Rate of Force Development (RFD)

In many situations it is not necessarily the magnitude of force that can be produced that determines sporting success, rather it is the rate at which force is developed. In theory two identical athletes who produce identical peak force in opposing directions (such as a scrummaging situation in rugby) would achieve zero net movement; this suggests that the only means for athletes of similar size and force producing potential to gain advantage over the other is to reach peak force in a shorter time. In this situation the

athlete with more rapid RFD would be superior. This concept was explored in a comparison of untrained men and ski jump athletes (Komi, 1984). The author showed there was no difference between groups in peak force production during a leg extension exercise between groups. However, the defining attribute of the ski jump athletes was superior RFD.

The type of training has also been shown to effect RFD. Hakkinen et al. (1985) showed that both explosive jump training and heavy resistance training improved peak force (11% and 27% respectively). However, only explosive jump training was able to significantly alter RFD (24% improvement following explosive jump training compared to 0.4% improvement following heavy resistance training). It is suggested that increased motor neuron firing rates, selective recruitment of high threshold MUs, or both, account for this adaptation (Aagaard et al., 2002; Sale, 2003).

Therefore, it is clear that neural mechanisms exist that allow greater expression of force and RFD independent of muscular adaptation. It is also clear that strength training methods are able to induce neural adaptations.

1.1.4 Summary of Strength Adaptation

Strength is the ability of the muscle to produce force. MUs are the functional units of the neuromuscular system and because they are composed of motor neurons and muscle fibres strength may be increased by inducing both muscular and neural adaptations.

Muscular adaptation is primarily a consequence of muscular hypertrophy which is a result of increased net accumulation of protein within the myofibril; this ultimately causes myofibrilar proliferation and hypertrophy of the muscle fibre. Hypertrophy of the muscle fibre may occur without increasing pCSA of the muscle. An increase in the

number of muscle fibres, hyperplasia, is not believed to contribute to increasing pCSA in short term interventions.

Neural adaptations that enhance strength include increased agonist activation, decreased antagonist activation, increased MU synchronization, increased firing rates and increased rate of force development. Neural adaptations are not limited by the time course associated with structural and metabolic repair/recovery processes seen in muscular adaptation; consequently, neural adaptations can occur more rapidly than muscular adaptation

Therefore, it appears clear that optimisation of muscular strength requires optimisation of the muscle pCSA, and optimisation of the neural components of muscle action. In the absence of any of these adaptations maximal strength will not be fully realised. Furthermore, for those who have already achieved significant muscular adaptation, neural adaptation may be the only means of further improvement. This suggests that interventions may not need to be as long as those intended to induce muscular adaptation because it is recognised that the time frame associated with neural adaptation is shorter.