

REVIEW ARTICLE

PHYSIOLOGICAL CHANGES IN SKELETAL MUSCLE AS A RESULT OF STRENGTH TRAINING

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INTRODUCTION

The ingredients for success in all physical activities, whether of a sporting or more mundane everyday nature, are skill, strength, speed and endurance. The object of training is to match the mixture of ingredients to the requirements of the event and the deficiencies of the athlete. This review is concerned with the physiological changes associated with training for the 'explosive' events such as sprinting, jumping and throwing where the main object is to maximize power output. The power output of a muscle is determined by the velocity of shortening and strength of the muscle. To increase power output it is therefore necessary to either increase the velocity of shortening and/or muscle strength.

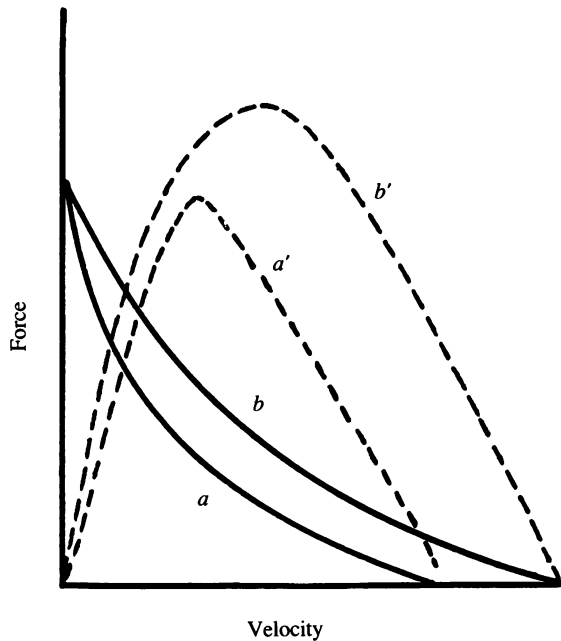


Fig. 1. The effect of increasing shortening velocity on the power output of a muscle. *a*, before training; *b*, after training. Dashed lines labelled *a'* and *b'* are the power outputs corresponding to curves *a* and *b*. Increase in the maximum velocity of shortening gives a greater maximum power output at a higher velocity of shortening.

Changes in shortening velocity

If the maximum velocity of unloaded shortening of the muscle were to increase, the maximum power that could be obtained from the muscle would also increase (Fig. 1). The speed of a muscle depends on the proportions of the different types of fibre, while the intrinsic velocity of shortening of an individual fibre is determined by the enzymic properties of its actomyosin cross-bridges. All muscle fibres contain the genetic information to produce fast and slow myosins and other proteins, and it is therefore conceivable that the slow fibres might be induced to change into fast fibres as a result of training. It is known that prolonged, relatively low-force activity, such as that imposed by chronic low-frequency electrical stimulation, causes a change to slow contractile characteristics (Salmons & Vrbová, 1969; Salmons & Henriksson, 1981; Rutherford & Jones, 1988). Evidence suggests that the converse is not true. Prolonged high-frequency stimulation in cat muscle caused a slowing of contractile characteristics similar to that seen with low-frequency stimulation (Eerbeek, Kernell & Verhey, 1984). It seems unlikely, therefore, that training will improve power output by increasing shortening velocity of a muscle as a consequence of a change in gene expression. There is, however, another way of increasing shortening velocity. The velocity of shortening depends on the intrinsic speed of the muscle but is also proportional to its length (i.e. the number of sarcomeres in series along the fibre). If muscle length and sarcomere numbers can be increased, possibly as a result of stretching exercises, then the maximum velocity of shortening will be proportionately increased. This possibility will be considered further in the section dealing with the length and velocity specificity of training.

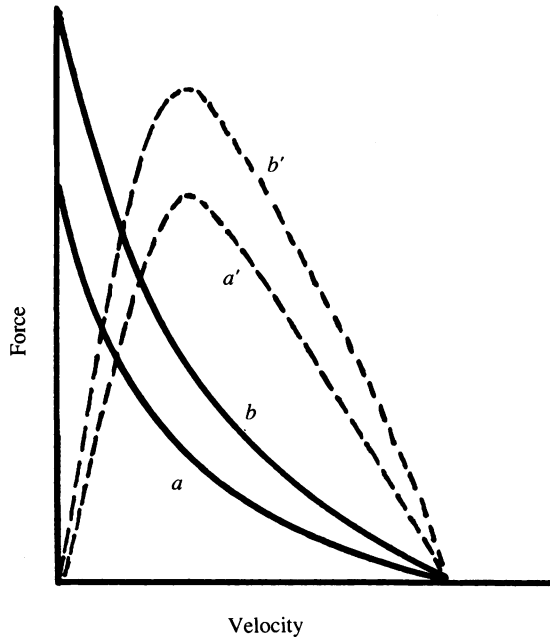


Fig. 2. The effect of increasing muscle strength on muscle power output. *a*, before training; *b*, after training. Dashed lines labelled *a'* and *b'* are the power outputs corresponding to curves *a* and *b*. Increasing the strength of the muscle increases the maximum power but does not affect the velocity at which this is achieved.

Changes in strength

If training is unlikely to increase the speed of shortening the only alternative, for those who wish to improve power output, is to try and increase the strength of the muscle. An increase in maximum isometric strength would be expected to lead to a proportional increase in strength at all velocities of shortening and consequently an increase in the maximum power developed (Fig. 2). Increasing power in this way would not affect the velocity at which the maximum power is attained.

Weight training is widely used as an adjunct to routine training for many sports, the general philosophy being that by increasing the size and strength of a specific muscle group, power output will be increased in events which use these muscles. This proves, however, to be a rather simplistic view.

HISTORY OF TRAINING STUDIES

In the early twentieth century Roux and Lange put forward the 'Aktivitätshypertrophie' theory, suggesting that a muscle would grow in size and strength when required to perform work of an intensity beyond its accustomed load (see Hettinger, 1961). This was an early form of the later developed 'overload' principle of deLorme (1946). In the 1940s and 1950s Captain Thomas deLorme, working with patients recovering from thigh and knee injuries, developed the progressive resistance training regime. The subjects trained at a load 60–90% of their maximum which was assessed weekly and, as they gained in strength, the load was increased. The value of this work was that it established the importance of high-intensity work rather than prolonged low-intensity exercise. In 1956 Hellebrandt and Houtz

confirmed this work using normal healthy subjects rather than patients with pre-existing muscle atrophy.

In the 1950s and 1960s Hettinger and Müller carried out a series of experiments to determine the minimum stimulus required for an increase in muscle strength and the different factors such as age and sex that could affect trainability. They concluded that one maximum isometric contraction of 1–2 s duration a day was sufficient to produce maximum improvements in strength. In the course of their studies they noticed that there was a wide variation in the susceptibility of different subjects and muscles to training, a finding that continues to frustrate and intrigue investigators (Hettinger, 1961).

Since this early work, many studies have compared the effects of isometric, isokinetic and isotonic training with seemingly every combination of repetition number, number of training days a week and relative training loads (for reviews see Atha, 1981; McDonagh & Davies, 1984). The number and variety of these studies makes a detailed comparison almost impossible but the basic conclusion that can be drawn is that as few as ten repetitions a day at loads greater than 60–70% of maximum will, if carried out regularly, produce strength increases of 0.5–1% per day. The original claim of Hettinger and Müller that one maximum contraction a day is sufficient has not been substantiated by other workers (Bonde-Petersen, 1960) and even the authors themselves have altered their original claims (see Royce, 1964). The physiological basis of these regimes is that by using high forces both high- and low-threshold units are recruited (Milner-Brown, Stein & Yemm, 1973) and subjected to a training stimulus. However lower-intensity contractions (around 30% maximum), if held for a longer duration (60 s) than usual (2–5 s), have been shown to produce increases in strength (Davies & Young, 1983). In this case the higher-threshold units will probably have been recruited towards the end of the contraction as others fatigue.

Despite the intense scientific and lay interest in strength training there remain many areas of ignorance and controversy and the remainder of this review will deal with three of these areas.

The first topic is the common observation that the benefits of training are very specific, being limited to the type of exercise undertaken and even to the speed of movement or the length of the active muscle.

The second area of controversy concerns the apparent change in the force-generating capacity of a muscle after training as a result of a greater change in muscle strength compared to size.

The final aspect to be discussed concerns the nature of the stimulus for muscle hypertrophy. Despite the obvious importance of this topic, there is little evidence on which to base rational training regimes either for athletes and body-builders or for patients undergoing rehabilitation following injury or illness.

SPECIFICITY OF THE TRAINING RESPONSE

A common finding in the majority of studies on training is that the greatest changes accompanying strength training can be seen in the training exercise itself rather than in any objective assessment of muscle strength or size (for review see Sale & MacDougall 1981).

Task specificity

There is an important difference between strength, defined as the ability to perform a task which may involve the co-ordinated contractions of a number of muscle groups and the strength of an individual muscle. Training is frequently carried out by lifting weights in an apparatus such as a multi-gym, and it is common experience that there will be a much

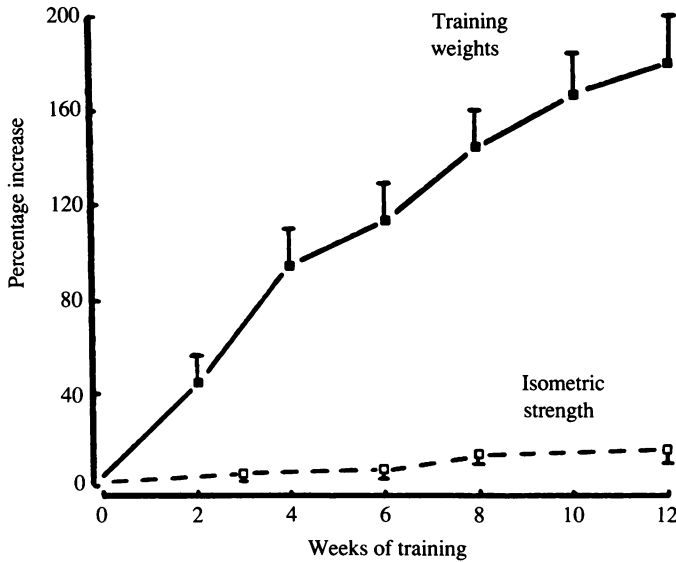


Fig. 3. Changes of strength with training. Changes in the weights lifted during training are compared with the increase in isometric strength of the quadriceps over a 12 week training period. Results are given as mean (\pm S.D.) percentage improvements.

greater increase in the ability to lift weights than in the intrinsic strength of the muscle groups being trained (Hellebrandt & Houtz, 1956; Lüthi, Howald, Claassen, Rösler, Vock & Hoppeler, 1986; Rutherford & Jones, 1986).

One of the early indications that training was specific to the movement pattern was reported by Rasch & Morehouse (1957). Subjects trained the elbow flexors in the standing position and were subsequently assessed both standing and supine. The increase in muscle strength was much greater in the familiar than in the unfamiliar position. A further indication of the specificity of movement pattern comes from studies examining changes in power output accompanying strength training. In one study, subjects trained for 12 weeks by lifting near-maximal loads on a leg-extension machine (Rutherford, Greig, Sargeant & Jones, 1986*a*). After 3 months of training, the improvement in training load was of the order of 200% accompanied by only a 15% increase in the isometric strength of the quadriceps (Fig. 3). Power output, assessed isokinetically using a modified cycle ergometer (Sargeant, Hoinville & Young, 1981), showed no change over the 12 weeks. Despite the improvement in the familiar training manoeuvre there was no significant change in power output measured in an unfamiliar manner, although the quadriceps was the main muscle group involved in both the training and isokinetic cycling. Such a lack of cross-over in performance suggests that the large increase in training weights lifted may be attributable to acquisition of skill in the training task, lifting weights, which was of little value in the different task of riding a bicycle.

Length specificity

A number of studies have investigated the improvements in strength as a result of training at specific muscle lengths. The majority have found that the increases in strength are greatest at the muscle length adopted during training. In one of the earlier studies of angle Jablecki, 1975) and the increased radiological density found after training in humans 6 weeks, and it was found that the strength increases were quite specific according to the

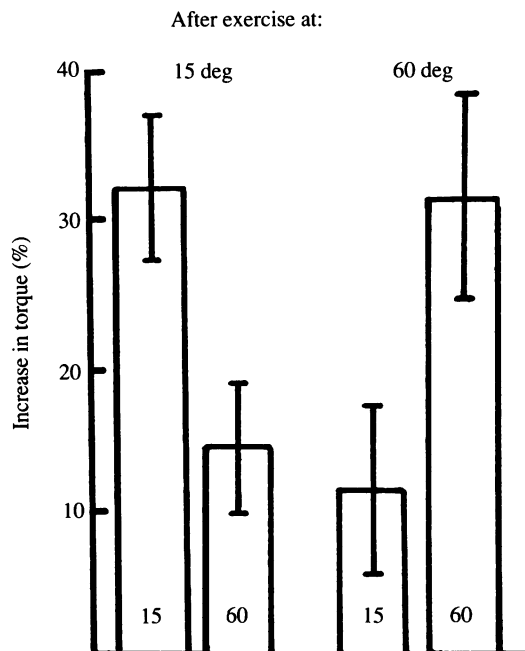


Fig. 4. Increases in strength after training at different angles. Training was carried out with the limb at either 15 or 60 deg (labels above the bars) and then tested at the two angles (labels within the bars). The greatest training effect was seen at the angle at which the training was carried out (after Lindh, 1979).

position at which the limb was exercised (Gardner, 1962). In a more extensive study Lindh (1979) trained subjects at either 15 or 60 deg of knee flexion and tested for isometric strength at both angles. Specific increases in isometric strength of about 30% were seen at the training angle compared to only 12% increases at the non-training angle (Fig. 4).

The concept of angle specificity has recently been examined for the elbow flexor muscle group. In a comprehensive study Thepaut-Mathieu, Hoecke & Maton (1988) examined the changes following training at 120, 80 or 25 deg of elbow flexion. The greatest specificity was after training at the shortened position (120 deg), and the least specificity in the group who trained at the lengthened position. The results of this study would suggest that the degree of specificity was dependent on the muscle length at which the training has been carried out: the shorter the length, the greater the specificity.

In contrast to the above reports, a few authors have found no evidence of specificity. Rasch, Pierson & Logan (1961) trained the elbow flexors isometrically at 90 deg, and found significant and equal strength increases at joint angles ranging from 45 to 135 deg. A later study by Rasch & Pierson (1964) confirmed the earlier observations.

Specificity of any description is generally attributed to 'neural adaptation' and the possibilities for this are discussed below. However, alternative muscle-based explanations are frequently overlooked yet may play an important role. The first possibility arises from the fact that muscle groups such as the elbow flexors or the knee extensors are composites of a number of separate muscles. The angle-tension relationships of such muscle groups are determined by the individual length-tension relationships of the component muscles. The different muscles may have their tension optima at different joint angles thereby extending the useful working range of the muscle group (Fig. 5A). If, as the result of training, there

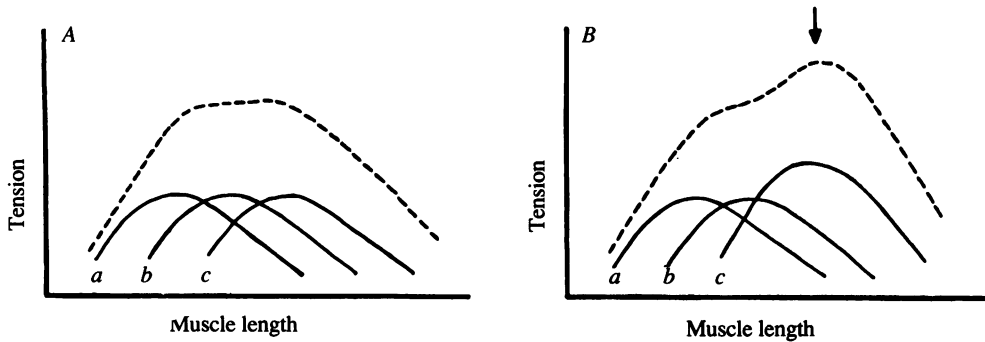


Fig. 5. The effect of selective muscle hypertrophy on the length-tension relationship of a compound muscle group. The muscle group is made up of three muscles, *a*, *b* and *c*, which have overlapping length-tension relationships which combine to give the composite relationship shown as a dashed line for the whole muscle group. *A*, before training; training at one length (arrow in *B*) may cause specific hypertrophy of muscle *c*, and a change in the length-tension relationship of the whole muscle (dashed line).

was preferential hypertrophy of one of the muscles this would alter the composite angle-tension relationship of the whole muscle group (Fig. 5*B*) so that the training response would appear to be maximal at the angle corresponding to the optimal length of the individual muscle. The second possibility arises as a result of a change in length of the muscle fibres. If the training were to be carried out at either very long or very short fibre lengths, where the forces generated were well below the maximum, then either a lengthening or shortening of the muscle might occur by the addition or loss of sarcomeres at the ends of the muscle fibres (Goldspink, Tabray, Tabray, Tardieu & Tardieu, 1974; Williams & Goldspink, 1978). Such a change in length could shift the angle tension-relationship so that maximum filament overlap with sarcomeres at around $2.5 \mu\text{m}$ was achieved with the muscle held at the training length (Fig. 6).

There are two ways of testing these ideas: one is to measure the changes in size of individual muscles within a muscle group such as the quadriceps to see whether there is any evidence of preferential hypertrophy of one or more muscles during training. The use of NMR imaging now makes this possible and preliminary results suggest that for the quadriceps there may be different degrees of hypertrophy amongst the component muscles (Cerretelli, Minetti, Narici, Roi & Scalmani, 1989). The second approach might be to measure the length-tension relationship of one of the component muscles of a group. If there was any change in sarcomere numbers this would be expected to show as a change in the length-tension relationship whereas if there was simply preferential hypertrophy there should be no such change. Both the quadriceps and the elbow flexor muscle groups contain muscles that extend over two joints (rectus femoris and the medial head of the biceps) which allows the length-tension relationships of these muscles to be determined independently of the other muscles in that group (Herzog & ter Keurs, 1988*a, b*).

Velocity specificity

Specificity of velocity in strength training has received much attention, particularly since the widespread availability of isokinetic testing machines such as the Cybex. Human skeletal muscle shows the same hyperbolic relationship between force and velocity as has been described for a wide variety of species (Wilkie, 1950; Close, 1972; Thomas, White, Sagar & Davies, 1987) and the expectation is that, should a muscle increase in isometric

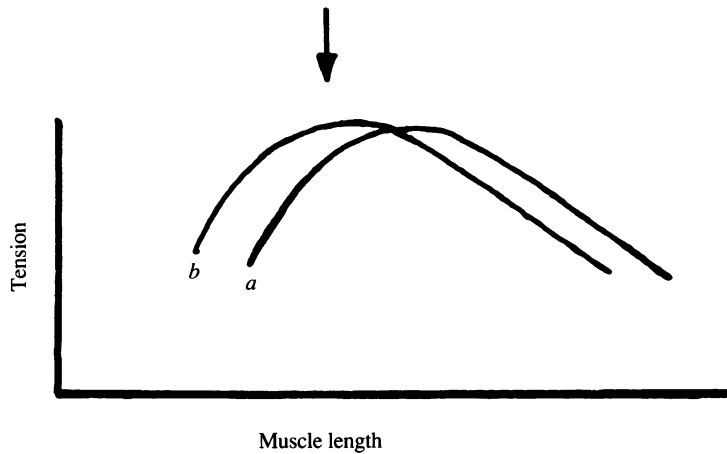


Fig. 6. The possible effect of training on muscle fibre length and the consequences for the length-tension relationship. Training with the muscle in a shortened position (arrow) may lead to a decrease in the numbers of sarcomeres in each muscle fibre and a shift in the length-tension relationship with an apparent and specific improvement in strength at the training length. *a*, before training; *b*, after training.

strength this would be reflected in a similar percentage increase in the force sustained at all velocities. This, however, has not always been the case. One of the earliest studies of isokinetic training was by Moffroid & Whipple (1970), who trained subjects at slow or fast velocities. The slow-velocity group showed the greatest changes at the lower velocities of testing, whereas the fast group showed uniform increases throughout the full range of test velocities and this was confirmed by Coyle, Feiring, Rotkis, Cote, Roby, Lee & Wilmore (1981). Caiozzo, Perrine & Edgerton (1981) and Kaneshi & Miyashita (1983) demonstrated increases in strength which were greatest at or near the training velocities (Fig. 7 *A* and *B*). Lesmes, Costill, Coyle & Fink (1978) found significant increases in strength at or below the training velocity.

The data supporting the concept of velocity specificity in training are far from conclusive. Considerable caution must be observed in interpreting data obtained with many commercial isokinetic testing machines since the force-velocity relationships obtained are generally very flat and linear, bearing little resemblance to those obtained with isolated muscles (Close, 1972) or with muscles *in situ* (Wilkie, 1950). Training would appear to introduce bumps into the force-velocity curves (Fig. 7) which, again, is quite at variance with conventional explanations for the shape of the relationship. The reason for these discrepancies is, most likely, deficiencies in the frequency response of the torque-recording systems which can introduce considerable artifacts into the records (see Thomas *et al.* 1987 for discussion). In addition, while some studies record torque at specific joint angles, others record peak torque from a damped signal, which could mask the true peak values.

It is important to review the possible mechanisms that would lead to a velocity-specific effect of training leaving aside, for a moment, possible neural adaptations.

A change in muscle fibre length will change the maximum velocity of shortening of a muscle. There is no reason to think that this does occur as a result of training at high or low velocity but the possibility could be checked by examining the length-tension relationship of muscle showing a velocity-specific effect. During rapid shortening slow muscle fibres may be unloaded and generate little or no force. If force *per se* is the stimulus

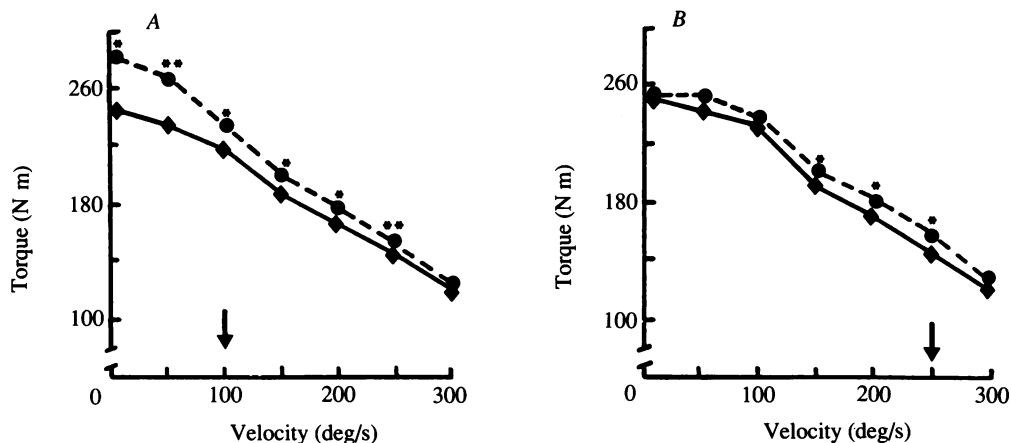


Fig. 7. The specificity of training at different velocities. Legs were trained at either a high (*A*) or a low velocity (*B*) (arrow shows the training velocity) and then tested over the whole range. Continuous line, before training; dashed line after training. * indicates significant differences as a result of training. Redrawn from Caiozzo, Perrine & Edgerton (1981).

for hypertrophy then high-velocity contractions might only provide a stimulus for growth of the fast motor units, possibly explaining the specificity of high-velocity training. However, the converse is not true as during high-force, low-velocity contractions, all motor units would be expected to be active and receiving a training stimulus.

Neural adaptation

It is possible that in the untrained state subjects are not able, by voluntary effort, to fully activate muscle groups at certain speeds or with the limb held in certain positions. There is no doubt that during rapid movements it is difficult for the subject to be sure that the effort is truly maximal and contractions certainly feel very unusual when holding an arm or leg in a fully flexed or extended position. Training may help the subject to fully activate the muscle over the full range of movement and speeds but there is little direct evidence that this is the explanation of length or velocity specificity and it might be more rewarding to consider the muscle-based explanations discussed above.

Task specificity may be accounted for by an improvement in co-ordination of the different muscle groups that are involved in activities such as learning to ride a bicycle or write with the non-dominant hand. The neural pathways involved in such learning are clearly complex, may exist at several levels in the central nervous system and involve various sensory inputs from skin, joints, eyes and the vestibular system. Although many training tasks may appear to require minimal skill this is only a relative judgement. Looking at subjects making large contractions of almost any muscle it is immediately obvious that they are also contracting many different muscles to stabilize the particular limb and the rest of the body. Contraction of abdominal and chest wall muscles seems to be a universal and necessary response to making a large effort and is part of the skill that has to be acquired.

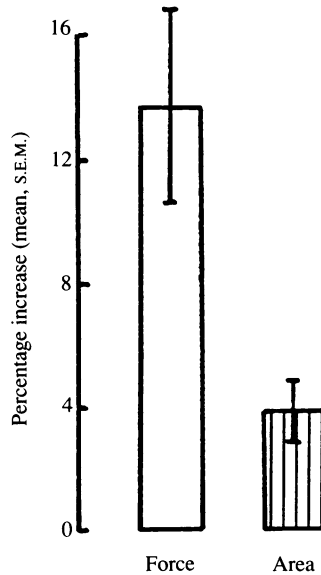


Fig. 8. Changes in muscle strength and size as a result of training. Quadriceps cross-sectional area and strength were measured before and after 12 weeks of training. Note that the increase in strength is greater than the extent of muscle hypertrophy. Data from Jones & Rutherford (1986).

CHANGES IN MUSCLE STRENGTH AND SIZE DURING TRAINING

Short-term training studies have demonstrated increases in strength that are greater than increases in muscle size (Ikai & Fukunaga, 1970; Moritani & deVries, 1979; Young, Stokes, Round & Edwards, 1983; Jones & Rutherford, 1987; Fig. 8). Once again the explanations for this phenomenon have centred on 'neural adaptations' although there are also several peripheral mechanisms which could result in an increased force per unit area.

Neural mechanisms

It has been claimed that, prior to training, muscle cannot be maximally activated by voluntary activity. In the first 6–8 weeks of training before changes in muscle size become apparent, it is suggested that activation, and therefore strength, increases as a result of altered neural drive. Evidence to support this view has come from surface EMG recordings where an increase of approximately 10% has been demonstrated in the maximum integrated EMG as a result of training (Moritani & deVries, 1979; Komi, 1986). There is also contradictory evidence of either no change or a decrease in maximum activation with training (Komi & Buskirk, 1972; Thorstensson, Karlsson, Viitasalo, Luhtanen & Komi, 1976*b*).

There are two main ways in which neural adaptation could result in an increase in the maximum voluntary isometric strength.

(1) Large fast motor units are recruited only at the higher forces and it is possible that during maximal voluntary contractions there are some units that are never recruited in the untrained state. Training is therefore seen as a way of facilitating the recruitment of these large and fast motor units.

(2) The pattern of electrical stimulation of the motor units may change. Electrical

stimulation of human muscles, either through the motor nerve or peripheral branches, requires frequencies of around 50–100 Hz to generate maximum tetanic force but measurements of the natural motor unit firing frequencies suggest that 20 Hz is close to the physiological maximum. This difference may be explained by the results of Rack & Westbury (1969) and Lind & Petrofsky (1978) who showed that asynchronous stimulation at a relatively low frequency of portions of a divided nerve generated more tension than synchronous stimulation at the same frequency. The explanation of this phenomenon probably concerns the amount of internal work performed by the muscle against series elastic components. With synchronous stimulation at low frequencies all motor units will be activated together and as tension is developed they will stretch the series elastic elements. In doing this the muscle will be moving and shortening and will consequently develop less than the maximal isometric force. With asynchronous stimulation the series elements will be stretched by the first portion of the muscle to contract but then remain stretched as the second and subsequent portions contract isometrically and generate full force. Komi (1986), however, has summarized the evidence that in trained muscles there is an increase in synchronization. It is not clear how this change would give rise to an increased isometric strength but it could be functionally important when it is necessary to rapidly develop maximum force such as during jumping or throwing.

The two possibilities described above would be expected to have some consequences for recorded EMG activity. Recruitment of previously inactive motor units, their use at higher firing frequencies or increasing synchronization would increase the maximum integrated EMG activity. Desynchronization of motor unit activity would be expected to lead to a decrease in the integrated EMG signal as a consequence of the individual motor unit potentials overlapping and cancelling out when recorded from surface electrodes.

There are considerable technical problems associated with monitoring EMG activity over a prolonged training period. The EMG signal picked up from surface electrodes is critically dependent on their position on the skin relative to the underlying muscle and on the impedance of the skin and underlying tissues. With care it is probably possible to relocate the electrodes with reasonable accuracy and most investigators go to some lengths to prepare the skin and reduce skin resistance to a low and constant value. What is not possible to control is the subcutaneous and intramuscular fat content that could well vary over the course of a prolonged training study.

A cross-over training effect has been observed in several studies where training one limb has resulted in strength increases in the contralateral untrained limb. Proponents of the neural hypothesis put this forward as support for a central adaptation affecting both limbs. Cross-over strength increases ranging from 10 to 30% have been demonstrated (Darcus & Salter, 1955; Komi, Viitasalo, Rauramara & Vihko, 1978; Moritani & deVries, 1979) whilst other groups have found no evidence for such an effect (Kruse & Mathews, 1955; Young *et al.* 1983; Jones & Rutherford, 1987; Davies, Parker, Rutherford & Jones, 1988). Young *et al.* (1983) suggested that the contradictory findings may be explained by the activity in the untrained limb during the training sessions. If support is offered to the other limb the subject may unknowingly contract the 'untrained' muscle thereby subjecting it to a training stimulus.

A different approach to the question of whether a voluntary contraction is fully activated is to stimulate the voluntarily contracting muscle to see whether any additional force can be obtained (Merton, 1954; Belanger & McComas, 1981; Bellemare & Bigland-Ritchie, 1984; Gandevia & McKenzie, 1985; Rutherford, Jones & Newham, 1986*b*; Fig. 9). The general conclusion from this type of investigation is that most normal subjects can fully

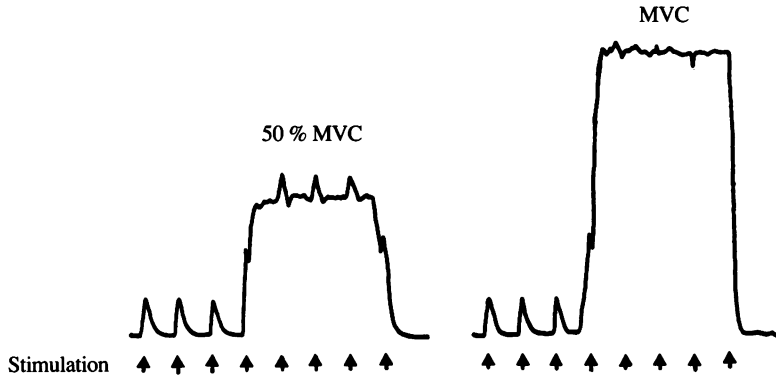


Fig. 9. Superimposed electrical stimulation on voluntary contractions. Muscle was stimulated with single twitches (arrows) at one a second while the subject made either a submaximal (left) or maximal (right) contraction. Note that during the maximal voluntary contraction (MVC) there is no additional force on stimulation.

activate most muscle groups without any training. In our own training experiments we routinely test subjects before, during and after training and have only found one subject who had any inhibition prior to training (Jones & Rutherford, 1987). It is difficult to imagine how demonstrably fully activated muscle can be further activated to produce greater force.

Very few training studies have been carried out in which measurements of tetanic force have been made mainly because of the difficulty and discomfort associated with supramaximal tetanic stimulation through the motor nerve of human muscle. The results from the few studies that have been done are contradictory. McDonagh, Hayward & Davies (1983) found that 5 weeks of maximum isometric contractions of the biceps brachii increased voluntary force by 20% with no change in tetanic force at 40 Hz. The same group found that 8 weeks of isometric training of the first dorsal interosseous (FDI) caused a 33% increase in voluntary force with no change in tetanic force (Dooley, McDonagh & White, 1983). They concluded that increased neural drive was responsible for the voluntary strength increases. However, in the first of these studies on the biceps the initial tetanic forces were much lower than the voluntary force which would suggest that either (i) the whole muscle was not being stimulated or (ii) other muscle groups were contributing to the voluntary contraction. In the second study on the FDI, values for tetanic force were not reported. In a similar study Duchateau & Hainaut (1984) found that 3 months of isometric or dynamic training of the adductor pollicis did increase tetanic tension by 20 and 11% respectively, values very similar to those found by others for voluntary force increases.

Peripheral mechanisms

Alternative explanations for the discrepancy between changes in muscle strength and size following training involve peripheral changes in the composition of the muscle which result in higher specific tensions. A number of possible mechanisms are discussed below and it will become apparent that the two major problems in this area are (i) methodological difficulties in studying muscle composition in humans and (ii) the lack of understanding of the factors that determine muscle strength in the untrained state. Only about 50% of the variation in quadriceps strength between people can be explained by variations in muscle cross-sectional area (Chapman, Grindrod & Jones, 1984; Rutherford, 1986; Fig. 10). Other

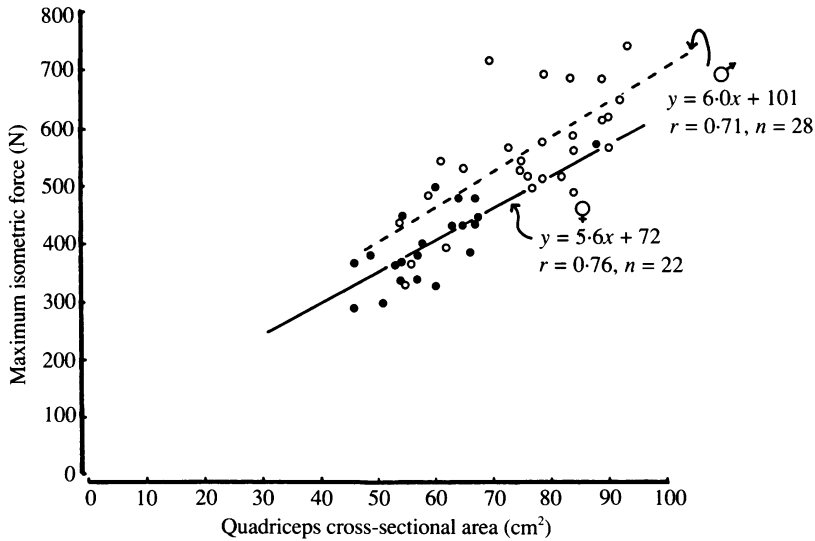


Fig. 10. The relationship between muscle cross-sectional area and strength. Quadriceps muscle cross-sectional area was measured by CT scanning and is compared with the maximum isometric voluntary contraction force in young male (○) and female (●) subjects. Data from Rutherford (1986).

factors which may affect strength include the lever system through which strength is measured (McCullough, Maughan, Watson & Weir, 1984), fibre type composition (Young, 1984; Grindrod, Round & Rutherford, 1987), fibre architecture (Alexander & Vernon, 1975) and the packing of contractile material (Penman, 1970). It is conceivable that the last three could be modified by a period of strength training.

Fibre type composition. There is some evidence from both human and animal work that type 2 fibres are intrinsically stronger than type 1. Studies in human muscle have suggested as much as a twofold difference in the two fibre types (Komi, Rusko, Vas & Vihko, 1977; Tesch & Karlsson, 1978; Young, 1984; Grindrod *et al.* 1987), but there are also contradictory reports showing no effect of fibre type on strength (Thorstensson *et al.* 1976*b*; Clarkson, Kroll & McBride, 1980; Maughan & Nimmo, 1984). The results from animal work are also equally contradictory, with support for intrinsically stronger type 2 fibres coming from several groups (Bárány & Close, 1971; Burke & Tsairis, 1973; Kean, Lewis & McGarrick, 1974; Edjtehardi & Lewis, 1979). If type 2 fibres do have a greater intrinsic strength than type 1, then either preferential type 2 hypertrophy or an increased type 2 frequency after training would lead to a greater increase in muscle strength compared to cross-sectional area in a mixed muscle. Although elite power athletes have been found to have larger type 2 fibres (MacDougall, Sale, Elder & Sutton, 1982; Tesch & Karlsson, 1982) the evidence for this occurring in short-term training studies is contradictory (Thorstensson *et al.* 1976*b*; MacDougall, Elder, Sale, Moroz & Sutton, 1980; Young *et al.* 1983; Rutherford, 1986). Where selective hypertrophy has been found, the differences have been too small to account for the disparity between changes in strength and size of the whole muscle. Neither is there any evidence for fibre type conversion after strength training regimes (for review see Edstrom & Grimby, 1986). Some caution is needed in drawing conclusions from the human biopsy work as there is a large individual variation

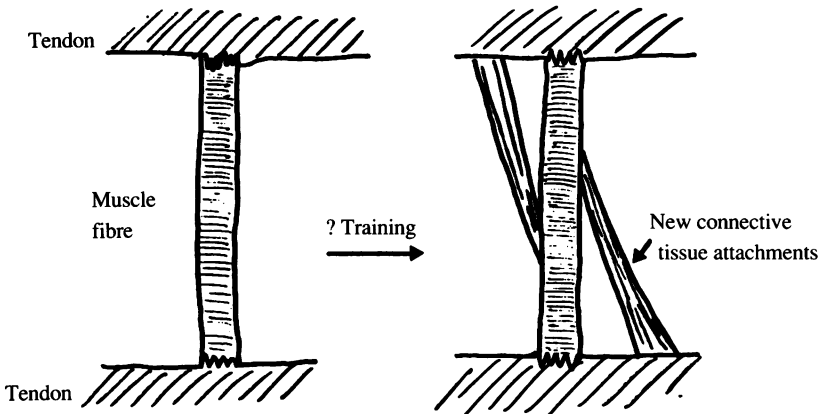


Fig. 11. Possible effects of training on connective tissue attachments. Left, before training a single muscle fibre is attached to the tendons only at the ends, while after training (right) additional connective tissue attachments are made between the tendon and intermediate points on the fibre such as to increase isometric force but reduce the effective length of the fibre.

in fibre hypertrophy resulting from the same training regime. In our experience changes in fibre area can range from -13 to $+47\%$, a finding confirmed by other investigators (Young *et al.* 1983; MacDougall, 1986).

The angle of muscle fibre insertion. Individual muscles vary in the arrangement of the fibres between tendons, the simplest architecture being where the fibres lie parallel to the line of action of the muscle (parallel muscle). Many muscles, including the four portions of the quadriceps, are pennate with the fibres inserting into the tendons at an angle to the line of action of the muscle. In this latter situation the cross-sectional area measured at right angles to the limb (anatomical cross-sectional area) underestimates the true cross-section of the fibres themselves (physiological cross-sectional area) (Edgerton, Roy & Apor, 1986). A change in this angle of insertion may alter the force measured between the ends of a muscle. For the same length and anatomical cross-section of muscle an increased angle of pennation can result in more contractile material being attached to a larger area of tendon (Alexander & Vernon, 1975; Gollnick, Timson, Moore & Riedy, 1981). The relationship between the force resolved in the tendon, the angle of pennation and the amount of contractile material, is complex, Alexander & Vernon (1975) calculated that this force was proportional to the sine of twice the angle of pennation. This model predicts that up to an angle of pennation of 45 deg the force will increase. In the human quadriceps the angle of pennation has been estimated to be about 13 – 18 deg (Alexander & Vernon, 1975). If, as a result of training, there was a simultaneous increase in both the angle at which fibres attach to the tendon and their cross-sectional area, the overall change in muscle cross-sectional area would be smaller than in fibre area (Rutherford, 1986). At present it is not possible to measure accurately the angle of pennation *in vivo* but an indirect way of investigating this problem would be to study the changes occurring in a parallel muscle after training. In a parallel-fibre muscle the physiological cross-sectional area can be estimated with reasonable confidence from the anatomical cross-sectional area. The constituent groups of the forearm flexors are mainly parallel and the results of studies training these muscle have given both qualitatively and quantitatively similar results to that

of the quadriceps (Ikai & Fukanaga, 1970; Davies *et al.* 1988) which would suggest that a change in fibre architecture is not the major cause of the increased force-generating capacity after training.

Contractile material packing. An increase in force per unit area could be explained by an increased packing of the contractile material. This could involve either a closer packing of the myofilaments or myofibrils or a loss of fat and connective tissue from between the fibres. Increased myofilament (Helander, 1961) and myofibril protein density (Gordon, Kowalski & Fritts, 1967) has been found after periods of heavy exercise in animal muscle, although variations in muscle density are reported to be very small in untrained animal muscle (Mendez & Keys, 1960). The results from human studies are contradictory; MacDougall, Sale, Moroz, Elder, Sutton & Howald (1979) found that high-resistance training produced an increase in the number and size of myofibrils but this was accompanied by a significant fibre hypertrophy such that the myofibril density remained the same. Conversely in a limited study of three subjects, Penman (1970) found that weight training resulted in a 50% increase in myosin filament density without a change in limb girth. There was a simultaneous increase in muscle strength of 40% which he attributed to the increased myosin packing. Several studies have now found an increase in the radiological density of muscle after training as measured from computerized tomography (CT) scans (Horber, Scheidegger, Grunig & Frey, 1985; Jones & Rutherford, 1987). This could occur for a number of reasons: a decrease in the fat content of the muscle, an increase in the packing of the contractile elements or an increase in the connective tissue content. A consequence of the first two possibilities would be an increase in the force per unit area.

Indirect evidence for tighter fibre packing is provided by the finding of greater increases in fibre area compared to the muscle anatomical cross-sectional area after training (McDonagh & Davies, 1984; Rutherford, 1986; Frontera, Meredith, O'Reilly, Knuttgen & Evans, 1988). On average the increases in muscle area are about 5–10% (Dons, Bollerup, Bonde-Petersen & Hancke, 1979; Young *et al.* 1983; Lüthi *et al.* 1986; Jones & Rutherford, 1987; Cerretelli *et al.* 1989) whereas increases in fibre area are closer to 15–20% (MacDougall, Sale, Moroz, Elder, Sutton & Howald, 1979; MacDougall *et al.* 1980; Rutherford, 1986; Frontera *et al.* 1988). However, as discussed above, this discrepancy could be explained by a rearrangement of the fibre architecture.

Connective tissue attachments. It is generally assumed that tension is transmitted longitudinally in a muscle fibre through serial sarcomeres so that the force is proportional only to the cross-sectional area and is independent of the length. If, however, attachments were made between the tendons and intermediate sarcomeres this would increase the force generated per unit cross-section of muscle (Fig. 11). Mammalian muscle fibres are enveloped in a connective tissue matrix which could play some role in transmitting tension to the tendons (Street, 1983). Work-induced hypertrophy is known to increase collagen synthesis in animal muscle (Schiaffino, Bornioli & Aliosi, 1972; Goldberg, Etlinger, Goldspink & Jablecki, 1975) and the increased radiological density found after training in humans (Horber *et al.* 1984; Jones & Rutherford, 1987) could result from an increased connective tissue content (Grindrod *et al.* 1982).

Consequences for power output. Changes in the muscle architecture or in the connective tissue attachments to the muscle fibres that result in a greater isometric force will also reduce the effective length of the muscle fibres. The change might be an absolute loss of fibre length with change in pennation or, where connective tissue attachments are made at points

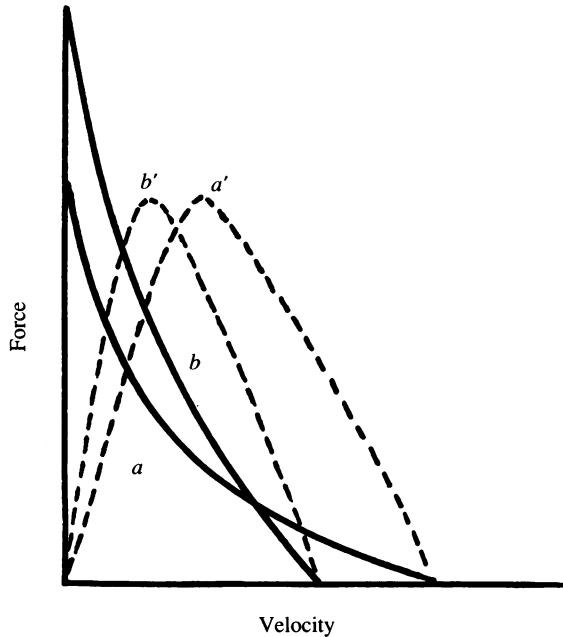


Fig. 12. The consequences of increased connective tissue (see Fig. 11) for muscle strength and power output. *a*, before training; *b*, after training. Dashed lines labelled *a'* and *b'* are the power outputs corresponding to curves *a* and *b*. The putative effect of training would be to increase isometric strength but decrease maximum shortening velocity giving rise to an increased power at lower velocities but reduced power at high velocities of shortening.

along the fibre (e.g. Fig. 11) there could be a reduction in the functional muscle length. In either case, the loss of length would be expected to result in a reduction of the maximum velocity of shortening because there would be fewer sarcomeres in series along the line of action of the muscle. The combination of an increased isometric force and decreased velocity of shortening would result in variable changes in power output depending upon the velocity at which the measurement was made. At low velocities the trained muscle would be expected to be more powerful, although the percentage increase would not be as great as for the isometric force alone, while at higher velocities of shortening the trained muscle would be less powerful (Fig. 12). The velocity at which the maximum power output was achieved would be expected to decrease if there was a shortening of the effective muscle length. There is little experimental evidence to test these ideas, but many of the experiments concerning velocity specificity of training tend to show greater gains in strength when testing at lower velocities (Lesmes *et al.* 1978; Caiozzo *et al.* 1981; and see Fig. 7). Training of the quadriceps, which resulted in significant gains in isometric strength, have been found not to result in increased power output when subjects were tested on a cycle ergometer (Rutherford *et al.* 1986*a*). However, in a training study of the adductor pollicis of the hand, Duchateau & Hainaut (1984) found that the maximum velocity of shortening of the trained muscle was unchanged although the training study was only on one subject with five points to define the force-velocity curve.

THE STIMULUS FOR INCREASE IN STRENGTH

A great deal has been written about the merits of different training protocols and it is of obvious importance to establish the most effective means of increasing strength (Atha, 1981; McDonagh & Davies, 1984). Unfortunately in many studies it is difficult to disentangle learning effects and possible neural adaptation from increases in strength and hypertrophy of individual muscles. Nevertheless there is a general consensus that high forces have to be employed before any new muscle growth is obtained. It is not clear whether it is the high force *per se* that is the stimulus for change or simply that it is a means of ensuring that all motor units are recruited and subjected to a training stimulus.

All growth requires the remodelling of existing structures, i.e. an increased turnover involving both synthesis and degradation, but an increase in tissue size implies an excess of protein synthesis over protein degradation (Waterlow, Garlick & Millward, 1978). There are two aspects to protein turnover: one is a maintenance of some basal tissue size for which turnover needs to respond to short-term changes such as circulating amino acids and glucose, and hormones such as insulin. Control at this level is probably at the translational level with fluctuations in the 'RNA activity'. The second aspect of control occurs over a longer time scale. The laying down of more tissue is probably dependent upon increased ribosomal content which, in turn, may require division of satellite cells and incorporation of one of the daughter nuclei into the muscle fibre. In this way the nuclear material increases and the DNA unit size remains constant.

The possible stimuli for muscle hypertrophy can be divided into three categories:

Hormonal stimuli

Exercise may result in endocrine, paracrine or autocrine responses that stimulate muscle growth. It is unlikely that endocrine changes are the major stimulus since hypertrophy is often limited to a single muscle group on one side of the body. Nevertheless a certain concentration, or pattern of release, of hormones such as insulin, growth hormone or testosterone, may have a permissive action acting in conjunction with local changes associated with the working muscles. Localized changes in paracrine hormones such as insulin-like growth factor-1 (IGF1) may be very important in regulating tissue growth but it has yet to be determined whether there is any mechanism coupling the production of growth factors to mechanical activity of the muscle.

Metabolic stimuli

Most people training with weights instinctively feel that 'it has to hurt to do any good'. The hurting referred to is the burning sensation associated with metabolic changes in the working muscles. Despite this common sentiment there is no clear indication as to whether metabolic depletion is a prerequisite for change. Heavy exercise will result in large metabolic fluxes in the tissue with the accumulation of high concentrations of H^+ , inorganic phosphate and creatine, together with smaller quantities of other substances such as ADP, NH_3 and inosine, and it is possible that one or more of these metabolites could stimulate muscle growth. However, these metabolite changes are associated with fatiguing rather than strengthening exercises and it is more likely that they would be the stimulus for mitochondrial proliferation and capillary growth, associated with increased endurance, rather than increase in muscle size and strength. In cases where there may be impaired oxygen supply to the muscle, and metabolites are likely to be in a chronically depleted state, such as with peripheral vascular disease, the muscular adaptations are to increase the

mitochondrial content rather than to increase fibre size. Highly trained endurance athletes do not have muscles that are especially strong.

Training exercises in which muscles are stretched (eccentric exercise or negative work) can generate forces in the active muscles that are considerably greater than the more conventional exercise in which muscles shorten as weights are lifted. In addition, because in eccentric exercise work is absorbed rather than generated by the muscle, the metabolic costs are far less than during concentric work (Abbot, Bigland & Ritchie, 1952; Curtin & Davies, 1972; Bigland-Ritchie & Woods, 1976). A comparison of training using eccentric or concentric contractions should therefore provide a means of testing whether the stimulus for muscle growth is high force or metabolite changes. There has been one report that eccentric exercise is the best training stimulus (Komi & Buskirk, 1972) but, perversely, the majority of studies which have used this approach have not shown a difference between the two types of exercise (Rasch, 1974; Jones & Rutherford, 1987) and the relative importance of force and metabolic change remains unresolved.

Mechanical factors

It is well established that muscles held in shortened or lengthened positions rapidly change their overall length so that the sarcomere lengths are restored to the optimum for force generation (Goldspink *et al.* 1974; Williams & Goldspink, 1978). This is achieved by addition or removal of sarcomeres from the ends of the fibres. In addition to changing in length, muscle also appears to hypertrophy in response to stretch (Laurent, Sparrow & Millward, 1978).

There are a number of ways in which muscle turnover might be affected by mechanical stress.

(i) High force might cause micro-damage to the sarcomere structure and thereby provide a stimulus for repair and compensatory growth. Goldspink (1971) has suggested that high forces lead to disruption of the Z discs causing the myofibril to split, the fragments then growing back to full size. This is an attractive visual model and dislocations of Z lines can be seen in electron micrograph photographs especially after exercise involving high-force eccentric contractions (Fridén, Sjöström & Ekblom, 1983; Newham, McPhail, Mills & Edwards, 1983).

(ii) Using isolated preparations of rat diaphragm or rabbit skeletal muscle, Reeds & Palmer (1985; for a review of their work see Reeds, Palmer & Wahle, 1987) have shown that mechanical stimulation (repeated stretching of the resting muscle) causes an increase in both protein synthesis and degradation. The authors have also convincingly shown that this is associated with increased prostaglandin production, $\text{PGF}_{2\alpha}$ being associated with increased synthesis and PGE_2 with degradation which is in keeping with the results of Rodermann & Goldberg (1982). The working hypothesis is therefore that mechanical stress causes activation of phospholipase activity and the liberation of arachidonic acid from muscle membranes which then serves as the precursor for prostaglandin synthesis.

(iii) The collagen connective tissue network formed by fibroblasts is an integral part of muscle and must grow and change with the muscle fibres. The connective tissue matrix also provides the connection between the force-generating components and the tendons and, as such, will be subject to mechanical stress. The mRNAs for IGF1 have been localized to fibroblasts in fetal muscle (Han, D'Ercole & Lund, 1987) and the presence of IGF1 has been reported in regenerating muscle fibres and satellite cells after injury (Jennische, Skottner & Hansson, 1987). These observations suggest a possible paracrine action, with IGF1 being synthesized in the fibroblasts but exerting an anabolic effect on the muscle

fibres. If fibroblasts in the connective tissue were to produce IGF1 in response to mechanical stress this might be a way of regulating muscle growth.

There are two problems with the mechanisms described above: the first is that they all imply some form of damage probably caused by high forces, yet eccentric exercise has been shown not to result in any particular benefit as far as strength is concerned (Jones & Rutherford, 1987). The second is that they probably relate to changes in the existing muscle fibre, i.e. maintenance protein turnover rather than providing a mechanism whereby extra nuclei can be incorporated into the muscle fibre. Muscles that have become atrophic as a result of injury and immobilization probably have a greater potential for growth and strength gain than normal uninjured muscles (Imms, Hackett, Prestidge & Fox, 1977), nevertheless the recovery is often incomplete in the absence of specific weight training (Imms & MacDonald, 1978; Imms, 1980). The relatively slow recovery contrasts with the rapid recovery of normal strength following experimental muscle damage. In the latter case damaged fibres degenerate and subsequently regenerate from satellite cells to a mature muscle fibres in 3–4 weeks. The new fibres seem to have a greater potential for growth since everyday activity appears to be a sufficient stimulus for full recovery of strength (Jones, Newham, Round & Tolfree, 1986; Newham, Jones & Clarkson, 1988). During recovery from immobilization-induced atrophy, growth occurs in mature fibres and these may require a greater stimulus before they return to their original size.

SUMMARY

The picture of training that emerges is of a process that can be divided into a number of phases. In the first phase there is a rapid improvement in the ability to perform the training exercise such as lifting weights which is the result of a learning process in which the correct sequence of muscle contractions is laid down as a motor pattern in the central nervous system. This phase is associated with little or no increase in the size or strength of individual muscles. The learning process appears to be very specific in that lifting weights makes better weight lifters but not better sprinters. The second phase is an increase in the strength of individual muscles which occurs without a matching increase in the anatomical cross-section. The mechanism for this is not clear but could be a result of increased neural activation or some change in the fibre arrangement or connective tissue content. The third phase starts at a point where scientific studies usually end, at about 12 weeks when non-athletic subjects are beginning to tire of the repeated training and testing. After this point, if training continues, there is probably a slow but steady increase in both size and strength of the exercised muscles. The stimulus for these changes remains enigmatic but almost certainly involves high forces in the muscle, probably to induce some form of damage that promotes division of satellite cells and their incorporation into existing muscle fibres. Our information on the effect of long-term training comes primarily from observations on elite athletes whose physique may well be the result of genetic endowment or the use or abuse of drugs. For the athlete or patient hoping to increase muscle size by weight training the best combination of intensity, frequency and type of exercise still remains a matter of individual choice rather than a scientific certainty.

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