Short-Term Training: When Do Repeated Bouts of Resistance Exercise Become Training?

Stuart M. Phillips

Catalog Data

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Abstract/Résumé

Chronic resistance training induces increases in muscle fibre cross-sectional area (CSA), otherwise known as hypertrophy. This is due to an increased volume percentage of myofibrillar proteins within a given fibre. The exact time-course for muscle fibre hypertrophy is not well-documented but appears to require at least 6–7 weeks of regular resistive training at reasonably high intensity before increases in fibre CSA are deemed significant. Proposed training-induced changes in neural drive are hypothesized to increase strength due to increased synchrony of motor unit firing, reduced antagonist muscle activity, and/or a reduction in any bilateral strength deficit. Nonetheless, increases in muscle protein synthesis were observed following an isolated bout of resistance exercise. In addition, muscle balance was positive, following resistance exercise when amino acids were infused/ingested. This showed that protein accretion occurred during the postexercise period. The implications of this hypothesis for training-induced increases in strength are discussed.

Au cours d’un entraînement prolongé à la force, la surface de section transversale (CSA) des fibres musculaires augmente ; c’est le processus d’hypertrophie. L’augmentation de la

Stuart Phillips is with the Dept. of Kinesiology at McMaster University, Hamilton, Ontario L8S 4K1.
CSA par l’entraînement est due à l’augmentation du pourcentage de protéines myofibrillaires dans une fibre donnée. La durée exacte de la période d’hypertrophie n’est pas connue mais il faut environ 6 à 7 semaines d’entraînement standard (trois fois par semaine) par des exercices dont l’intensité est d’au moins 70% du maximum avant d’observer un 10 à 15% d’amélioration dite significative. Au cours des premières phases d’un programme d’entraînement à la force, l’augmentation de la force est principalement due à une meilleure activation des fibres musculaires. Parmi les mécanismes hypothétiques de modification de l’activation agissant seul ou en combinaison, mentionnons la meilleure synchronisation des unités motrices, la réduction de l’activité des antagonistes et la réduction potentielle du déséquilibre bilatéral. À la suite d’une seule séance d’exercices, on note une augmentation de la synthèse des protéines. En outre, grâce à la récente élaboration de modèles et de techniques d’estimation précise du catabolisme protéique chez les humains, le bilan protéique (synthèse moins dégradation) dans le muscle est positif si des acides aminés sont fournis (par injection ou par ingestion) au cours de l’entraînement. Un bilan protéique positif à la suite de séances de musculation est une indication d’accrétion, probablement au niveau des protéines productrices de tension. L’addition de nouvelles protéines myofibrillaires n’a pas fait l’objet d’observation directe mais la possibilité existe. Les conséquences pratiques de cette hypothèse pour l’amélioration de la force par l’entraînement sont analysées.

Introduction

Increased resistive loading of human skeletal muscle brings about numerous adaptations within and external to the muscle, including increased force generating capacity, changes in myosin isoforms, increased fibre cross-sectional area (CSA), changes in ionic pump (i.e., Na⁺- K⁺-ATPase and sarcoplasmic reticulum Ca²⁺-ATPase) activity, and adaptations in the neuromuscular system. One of the most commonly studied adaptations to resistance exercise is the increase in skeletal muscle fibre CSA—fibre hypertrophy. Underlying the training-induced hypertrophy is an increase in the rate of skeletal muscle protein synthesis that chronically exceeds the rate of skeletal muscle protein breakdown. While this caveat may seem self-evident, it was only recently that techniques were developed to measure the rate of skeletal muscle protein breakdown and apply the techniques to humans (Biolo et al., 1995; Phillips et al., 1997; Phillips et al., 1999). The fact that the rate of breakdown of skeletal muscle can be measured is of extreme importance since skeletal muscle proteins are constantly being synthesized and degraded, and it’s the balance between these processes that determines protein accretion or loss (Biolo et al., 1995; Phillips et al., 1997). Normally the process of skeletal muscle protein synthesis (MPS) is stimulated during the fed state due to the postprandial increase in insulin but predominantly to the increased provision of amino acids (Svanberg et al., 1997; Svanberg et al., 1996). At the same time while MPS is stimulated, in the fed state, muscle protein breakdown (MPB) is reduced, but MPB increases with time as the postprandial period (up to 4–5 hr post-feeding) becomes the post-absorptive (>5 hr post-feeding) period, depending on meal size (Svanberg et al., 1999). Hence, accretion of protein (i.e., positive muscle protein balance) can only occur when a person is in the fed state.
Hypertrophy

Muscle fibre hypertrophy is defined as an increase in muscle fibre CSA. There are likely multiple signals that could trigger hypertrophy; however, one possibility is an increase in protein density within the muscle fibre, which acts as a stimulus for increasing fibre size. One important point to make is that, in theory, density (i.e., mass/volume) of myofibrils could increase within a fibre without an increase in CSA (Figure 1). However, protein packing within the muscle fibre is relatively precise since myofibrils make up ~80% of the fibre space. Hence, there would not be much room for myofibrillar protein density to increase, without interfering with filament interaction. There is evidence that myofilament lattice spacing decreases with models of inactivity, in which fibre areas, and specific tension, are reduced (Widrick et al., 1999). This author is unaware, however, of an exercise-induced increase in myofilament lattice spacing.

An unsettled issue is whether resistance exercise is a stimulus for increasing the synthesis of all cellular proteins or whether it is selective for only the force-producing myofibrillar proteins. A number of studies have reported that resistance exercise results in an increase in mixed MPS (Biolo et al., 1995; Biolo et al., 1997; Chesley et al., 1992; Phillips et al., 1997; Phillips et al., 1999; Yarasheski et al., 1999; Yarasheski et al., 1993); however, it is not clear whether the exercise-induced increase in MPS is due to increased synthetic rates of all (i.e., myofibrillar and nonmyofibrillar) proteins or exclusively myofibrillar. Some studies have shown

- Myofibril

![Diagram](Image)

Figure 1. Proposed scheme of addition of force-generating myofibrillar proteins that occurs early in a resistance training program. Each dot (*) with a muscle fibre represents a new myofibril. The time taken for the transformation from pretraining to time 1, if such a transformation occurs at all, is unknown. From pretraining to time 2 appears to take at least 6 weeks (see text for details).
that resistance training-induced hypertrophy reduces muscle fibre mitochondrial volume percentage, due to the fact that an increasing proportion of fibre volume was occupied by myofibrillar protein (Alway et al., 1988; Wang et al., 1993). At the same time, sarcoplasmic reticulum and cytoplasmic proteins appear to increase in proportion to the increase in myofibrillar proteins (Alway et al., 1988). The obvious implication of these findings is that resistance exercise is a greater stimulus for myofibrillar versus mitochondrial protein (Alway et al., 1988; MacDougall et al., 1979). Recent observations (Chilibeck et al., 1999) appear to have confirmed this earlier work (Alway et al., 1988; MacDougall et al., 1979). However, findings from other investigators have shown that resistance training resulted in no change in the relative fibre volume occupied by myofibrils or mitochondria, despite an increase in fibre CSA (Hather et al., 1991; Wang et al., 1993). In contrast to studies reporting a training-induced reduction in muscle mitochondrial volume density (Alway et al., 1988; Chilibeck et al., 1999; MacDougall et al., 1979) are reports that resistance training induces proportional changes in myofibrillar and mitochondrial proteins (Hather et al., 1991; Wang et al., 1993). Varying methodology used in quantifying mitochondrial density/oxidative capacity, initial training status of subjects, and the type of resistive training employed likely contributed to the divergent findings (Alway et al., 1988; Chilibeck et al., 1999; Hather et al., 1991; MacDougall et al., 1979; Wang et al., 1993). A conclusion regarding how resistance training affects mitochondrial protein volume density, therefore, would remain speculative.

The time necessary for muscle hypertrophy to occur is not well-defined and likely is dependent upon the initial training status of the individual (Alway et al., 1992), the type of training program (i.e., high repetition, low load; low repetition, high load; eccentrically or concentrically biased; Akima et al., 1999; Conley et al., 1997; Hisaeda et al., 1996), nutritional factors (Biolo et al., 1997), and genetically determined variables affecting hormonal responses, local growth factor production, and membrane permeability to selected ions. Most studies that have examined the time course of fibre hypertrophy have, however, used the muscle biopsy technique and quantified fibre size using histochemical staining (Goreham et al., 1999; Green et al., 1999; Hortobagyi et al., 1996; MacDougall et al., 1979; McCall et al., 1996; Staron et al., 1994). From these (Green et al., 1999; Hortobagyi et al., 1996; MacDougall et al., 1979; McCall et al., 1996; Staron et al., 1994; Zgoreham et al., 1999) and other studies, it appears that at least 6–7 weeks of training are necessary to induce a measurable increase in muscle fibre hypertrophy, at least when 10 subjects or less are being trained. Other methods that have been used to assess the change in muscle CSA include magnetic resonance imaging (MRI; Akima et al., 1999; Conley et al., 1997; Higbie et al., 1996; Narici et al., 1996; Walker et al., 1998), although relative changes in muscle CSA measured using MRI do not necessarily show good correspondence with relative changes in fibre CSA measured from biopsies (Narici et al., 1996). The lack of agreement between changes in MRI-measured CSA of whole muscle and individual fibre CSA from biopsies might be explained by the fact that MRI images contain connective tissue, the content of which may change with training, which does not affect estimates of histochemically determined fibre CSA. Alternatively, the variability in muscle fibre CSA is high enough that a single muscle biopsy may not be reliable in estimating an average muscle fibre CSA.
Strength Increases

There is no doubt that resistive training increases strength, measured either as a dynamic voluntary single-repetition maximum or as an increase in maximal voluntary isometric or dynamic torque (Conley et al., 1997; Goreham et al., 1999; Green et al., 1999; Higbie et al., 1996; Hortobagyi et al., 1996; MacDougall et al., 1979; McCall et al., 1996). A variety of studies have shown that when strength, expressed as a maximal voluntary contraction (MVC), is expressed relative to muscle CSA, most differences in force generating capacity are no longer evident (Berg et al., 1997; Welle et al., 1996); however, there is a tremendous variability in MVC/CSA in humans, and MVC/CSA can be increased with the appropriate training regime (Berg et al., 1997; Welle et al., 1996). That MVC/CSA (which differs from the true definition of specific tension, defined as maximum tension or Po/fibre CSA) can increase with training has been cited as good evidence, since CSA has not changed, that strength is not a property that is completely intrinsic to the muscle fibre, but that there are neural factors that can change the muscle’s ability to generate force and that the neural system is trainable. Hence, neural drive is not thought to be the same between trained and untrained individuals (Akima et al., 1999; Enoka, 1998; Sale, 1992).

Prevailing theory is that much, or even all, of the early gains in strength seen at the initiation of a resistance training program are due to neural adaptations (Akima et al., 1999; Chilibeck et al., 1998; Enoka, 1998; Hakkinen et al., 1998; Sale, 1992). These neural training-induced adaptations might include an improved synchronicity in motor unit firing, reduced bilateral deficit (if the lift is bilateral in nature), and potentially reduced antagonist muscle activity (Enoka, 1998; Sale, 1992). However, a crux of the argument that strength training mediates an increased neuromuscular drive is that changes in neither muscle nor muscle fibre CSA can be observed during a short period of training in which increases in strength have been observed to occur (Akima et al., 1999; Chilibeck et al., 1998; Enoka, 1998; Hakkinen et al., 1998; Sale, 1992). The problem with this stance is that the methodology for estimating muscle and muscle fibre CSA may not be sensitive enough to detect small yet significant increases. Further, it may be that increases in myofibrillar protein volume density within a fibre could increase before increases in fibre diameter could be observed (Figure 1). Consequently, while it may take weeks to realize the repeated addition of these proteins as a measurable increase in muscle fibre CSA (Goreham et al., 1999; Green et al., 1999; Hortobagyi et al., 1996; McCall et al., 1996; MacDougall et al., 1979; Staron et al., 1994), it may be that relevant, from a strength gain perspective, increases in myofibrillar protein content could occur earlier than previously realized. The proposed hypothesis is that these newly added myofibrillar proteins would not contribute to an increase in muscle fibre CSA; this idea is shown schematically in Figure 1.

Until recently, no reliable methodology existed to measure the breakdown rate of human muscle protein. A semiquantitative index of muscle myofibrillar protein degradation, 3-methylhistidine excretion, has been used but has severe limitations in interpretation due to the existence of far more labile pool, relative to skeletal muscle, of gut myofibrillar proteins (Rennie et al., 1983). Recent studies, however, have reported simultaneous measurements of both muscle protein synthesis and breakdown in human skeletal muscle following exercise, allowing the
calculation of muscle protein balance (Biolo et al., 1995; Biolo et al., 1997; Phillips et al., 1997; Phillips et al., 1999; Tipton et al., 1999). Resistance exercise by itself will increase the synthesis of muscle proteins and at the same time increase protein breakdown, but net balance (synthesis minus breakdown) becomes more positive (Biolo et al., 1995; Phillips et al., 1997; Phillips et al., 1999). However, resistance exercise and amino acids (protein) act synergistically to enhance muscle protein synthesis, and in the case of increased amino acid supply reduce an exercise-induced increase in protein breakdown (Biolo et al., 1997; Tipton et al., 1999).

The results from these studies also indicate that muscle protein balance becomes positive when amino acids are consumed during the post-exercise period, indicating a net accretion of protein occurring after only a single bout of exercise in untrained persons (Biolo et al., 1997; Tipton et al., 1999). These results, however, only show an increase in mixed muscle protein synthesis (Biolo et al., 1997; Tipton et al., 1999), which means that conclusions regarding the nature of the proteins that are being synthesized, myofibrillar or nonmyofibrillar, cannot be made. Since fibre hypertrophy and an increase in force generating capacity occurs following chronic resistance exercise, it seems that the postexercise increase in mixed MPS (Biolo et al., 1995; Biolo et al., 1997; Chesley et al., 1992; Phillips et al., 1997; Tipton et al., 1999; Yarasheski et al., 1993) must be due, at least in part, to an increased myofibrillar protein synthetic rate, since myofibrillar proteins make up ~70% of total skeletal muscle protein in humans (Welle et al., 1993).

Recent evidence suggests that the increase in postexercise mixed MPS is closely paralleled by the synthesis of actin and myosin (K.E. Yarasheski, personal communication), implying that a net gain of myofibrillar proteins is occurring following only a single bout of resistance exercise. How long it takes for these new myofibrillar proteins to be fully integrated and active in generating force within the muscle fibre is not known but theoretically could occur following a single “training” session. While speculative in nature, a rapid exercise-induced increase in new force-producing myofibrillar proteins appears to be a real possibility, which casts doubt on the idea that strength gains that occur early in a resistance training program are due exclusively to neural mechanisms (Akima et al., 1999; Chilibeck et al., 1998; Enoka, 1998; Hakkinen et al., 1998; Sale, 1992). In fact, the addition of new force-generating myofibrillar proteins without a change in fibre diameter may explain why training can induce a change in MVC/CSA, that is, a change in strength without changes in muscle and/or fibre CSA.

It is worth mentioning at this point that hypertrophy, brought about by dynamic training (leg press), does not necessarily result in an increase in isometric torque (Sale et al., 1992). Hence, hypertrophy cannot be the sole mechanism for gains in strength either. In fact, strength gains following the initiation of a strength training program in complete novices show that impressive gains in strength can occur very early (i.e., within 2 days; Chilibeck et al., 1998; Staron et al., 1994); this would appear to be far too early to be accounted for by increases in new myofibrillar proteins. Early gains in strength, however, would be highly dependent on whether strength was measured following isometric, eccentric, isokinetic, or more complex lifts such as a clean and jerk in which session-by-session gains in strength are apparent. There is almost no doubt that neural gains in strength are the predominant reason for early changes in strength during these types of resistance training programs.
Conclusion and Future Directions

Gains in strength following the initiation of a resistance training program in previously sedentary persons appear soon after the onset of training. While previous arguments have been made that increases in strength in this early phase of training are due almost exclusively to neural mechanisms, there is evidence that new force generating proteins could be added to the muscle fibre following only a single session of weightlifting. While concrete evidence to support this contention does not currently exist, future studies should focus on more sensitive methodology to measure rates of muscle myofibrillar protein synthesis and breakdown to see whether the proposed net gain of myofibrillar proteins (see Figure 1 and arguments presented above) actually occurs. Further, a close examination of the time course of how the relative myofibrillar volume percentage increases following resistance training needs to be performed (Figure 1). More sensitive measures such as electron microscopy should be used to more accurately assess whether myofibrillar lattice packing might adapt during the early phases of a resistance training program. Finally, measures from muscle fibre segments from human biopsies are now quite reproducible (Widrick et al., 1999); it would be of interest to see how the force properties (Po/CSA) might change early in a training protocol.

References


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