Myofibrillar disruption following acute concentric and eccentric resistance exercise in strength-trained men

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Abstract: We have previously quantified the extent of myofibrillar disruption which occurs following an acute bout of resistance exercise in untrained men, however the response of well-trained subjects is not known. We therefore recruited six strength-trained men, who ceased training for 5 days and then performed 8 sets of 8 uni-lateral repetitions, using a load equivalent to 80% of their concentric (Con) 1-repetition maximum. One arm performed only Con actions by lifting the weight and the other arm performed only eccentric actions (Ecc) by lowering it. Needle biopsy samples were obtained from biceps brachii of each arm ~21 h following exercise, and at baseline (i.e., after 5 days without training), and subsequently analyzed using electron microscopy to quantify myofibrillar disruption. A greater ($P \leq 0.05$) proportion of disrupted fibres was found in the Ecc arm (45 ± 11%) compared with baseline values (4 ± 2%), whereas fibre disruption in the Con arm (27 ± 4%) was not different ($P > 0.05$) from baseline values. The proportion of disrupted fibres and the magnitude of disruption (quantified by sarcomere counting) was considerably less severe than previously observed in untrained subjects after an identical exercise bout. Mixed muscle protein synthesis, assessed from ~21–29 h post-exercise, was not different between the Con- and Ecc-exercised arms. We conclude that the Ecc phase of resistance exercise is most disruptive to skeletal muscle and that training attenuates the severity of this effect. Moreover, it appears that fibre disruption induced by habitual weightlifting exercise is essentially repaired after 5 days of inactivity in trained men.

Key words: muscle damage, muscle injury, protein synthesis, hypertrophy, leucine.

Résumé : Dans une étude antérieure, nous avons mesuré l’ampleur de la rupture myofibrillaire qui se produit après un exercice aigu contre résistance chez des hommes non entraînés ; toutefois, on ne connaît pas la réponse des sujets bien entraînés. Par conséquent, nous avons recruté six hommes s’entraînant contre résistance, qui ont cessé de s’entraîner pendant 5 jours, pour ensuite effectuer 8 séries de 8 répétitions unilatérales avec une charge équivalant à 80 % de leur répétition maximale concentrique (Con). Un bras a effectué uniquement des mouvements Con en levant le poids, alors que l’autre bras a effectué uniquement des mouvements excentriques (Exc) en l’abaissant. Des échantillons ont été prélevés par biopsie à l’aiguille du biceps brachial de chaque bras environ 21 h après l’exercice, et en conditions basales (c.-à-d. après 5 jours sans entraînement), puis ont été analysés par microscopie électronique pour mesurer l’ampleur de la rupture myofibrillaire. Une plus grande proportion ($P \leq 0.05$) de fibres endommagées a été observée dans le bras Exc (45 ± 11 %) qu’en conditions basales (4 ± 2 %), tandis que l’ampleur de la rupture fibillaire dans le bras Con (27 ± 4 %) n’a pas différé ($P > 0.5$) de celle observée en conditions basales. La proportion de fibres endommagées et l’ampleur de la rupture (mesurée par le comptage des sarcomères) ont été significativement moins importantes que ce qui avait été observé chez des sujets non entraînés après un exercice de même type. La synthèse protéique musculaire, évaluée environ 21–29 h après l’exercice, n’a pas différé entre les bras Con et Exc. Nous concluons que la phase Exc de l’exercice contre résistance est plus dommageable pour le muscle squelettique et que l’entraînement atténue l’intensité de cet effet. De plus, il semble que la rupture fibrillaire provoquée par un entraînement régulier avec poids et haltères soit totalement réparée aprè 5 jours d’inactivité chez les hommes entraînés.

Mots clés : dommage musculaire, lésion musculaire, synthèse protéique, hypertrophie, leucine.

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Introduction

The high mechanical tension generated in skeletal muscle during heavy resistance exercise is believed to be the primary stimulus for muscle hypertrophy (MacDougall 1986; McDonagh and Davies 1984), but may also cause tissue injury (Ebbeling and Clarkson 1990; Stauber 1989). Muscle damage following resistance exercise has most often been assessed using indirect markers such as changes in serum enzyme concentrations, force production, or muscle soreness ratings (Clarkson et al. 1986; Ebbeling and Clarkson 1990). However, we (Gibala et al. 1995) and others (Staron et al. 1992; Roth et al. 1999) have provided direct evidence of myofibrillar disruption following weightlifting exercise in humans. As previously observed following other types of strenuous exercise (Fridén et al. 1981, 1983), the most common structural element affected was the Z-disk, which appeared widened, distorted, or smeared, and frequently interrupted the normal transverse alignment of sarcomeres (Staron et al. 1992; Gibala et al. 1995; Roth et al. 1999). We have further shown that the eccentric (Ecc) phase of weightlifting results in greater fibre disruption than the concentric (Con) phase (Gibala et al. 1995). This observation is consistent with the findings of a bench-stepping study conducted by Newham et al. (1986), which to our knowledge is the only other direct comparison of ultrastructural disruption following these two types of muscle action in humans. The greater fibre disruption may be attributable to higher absolute tensions generated in active fibres, since electromyographical recordings have indicated that for a given absolute load there is less muscle activation during an Ecc as compared to Con muscle action (Komi and Buskirk 1972; Gibala et al. 1995).

It appears that trained muscles are less susceptible to exercise-induced muscle damage (Ebbeling and Clarkson 1989) and that even a single bout of resistance exercise can exert a protective effect on the muscle, such that strength loss, muscle soreness, and muscle-specific enzyme release are markedly attenuated following a subsequent exercise bout (Ebbeling and Clarkson 1990; Clarkson et al. 1992). Again however, most studies have relied on indirect markers of tissue injury (Vincent and Vincent 1997), and few data are available regarding changes in the myofibrillar fine structure of skeletal muscle following resistance exercise in trained subjects. Staron et al. (1992) and Roth et al. (1999) observed evidence of fibre disruption in previously untrained subjects after 8–9 weeks of leg strength training. The relative proportion of damaged fibres reported in those studies (Staron et al. 1992; Roth et al. 1999) was considerably lower than what we observed in untrained subjects (Gibala et al. 1995), however this comparison is confounded somewhat by the fact that we examined the biceps brachii and the other two studies examined the vastus lateralis. Moreover, to our knowledge, no study has systematically investigated myofibrillar disruption following an acute bout of resistance exercise in subjects who have trained regularly for a number of years.

The primary purpose of the present study, therefore, was to determine whether myofibrillar disruption occurs following an acute bout of heavy resistance exercise in well-trained individuals and further to separately examine the Con and Ecc phases of exercise in these subjects. The general methods employed, and the specific type, intensity, duration, and volume of the exercise bout, were similar to that utilized in our previous study of untrained individuals (Gibala et al. 1995). Although cross-sectional in nature, we believed this would permit a more satisfactory comparison between trained and untrained individuals for the reason stated above. As in our previous study (Gibala et al. 1995), fibre disruption was quantified using electron microscopy and a rating scale designed to assess the severity of disruption.

In addition, we have previously demonstrated that mixed muscle protein synthetic rate (MPS) is elevated in biceps brachii following a single bout of heavy resistance exercise (Chesley et al. 1992) and appears to peak approximately 24 h following the training session (MacDougall et al. 1995). The design of the present study presented us with an excellent opportunity to examine the potential effects of myofibrillar disruption on MPS. If, as in our previous study (Gibala et al. 1995), the Ecc-exercised muscles were to suffer the greatest damage, one might hypothesize that they should also demonstrate a higher protein synthesis rate following the exercise. Consequently, a second objective was to examine the effect of contraction type on MPS following exercise.

Materials and methods

Subjects

Six healthy men, with a mean age, height, and weight of 22.1 ± 1.1 y, 175.8 ± 4.3 cm, and 78.8 ± 4.6 kg, respectively, volunteered for the study. The subjects were recreational bodybuilders from the McMaster University community who had been engaged in a regular program of resistance training (≥3 times per wk) for 3.4 ± 0.9 y. Subjects were specifically engaged in training of the elbow flexor muscles either once or twice per week, and each session consisted of 6–10 sets of various arm curl exercises at intensities ≥80% of their one repetition maximum (1 RM). The subjects were advised of the purposes of the study and associated risks and all gave written informed consent. The experimental protocol was approved by the Human Ethics Committee of McMaster University.

Pre-experimental procedures

Approximately 14 d prior to the actual experiment, subjects refrained from training their elbow flexor muscles for 5 d and then reported to the laboratory in order to determine their 1 RM. The exercise performed was a unilateral arm curl movement with the dominant arm, using a standard weight training dumbbell while seated on an incline bench. Loads were increased in multiples of 2 kg and a 3 min rest interval was allowed between attempts. The criteria used to indicate that 1 RM load was achieved were: (i) the ability to just complete a strict repetition, and (ii) the inability to perform a complete repetition with a heavier load following a successful 1 RM attempt.

Experimental protocol

The protocol was designed to separately examine the effect of an acute bout Con or Ecc weightlifting exercise on myofibrillar disruption and MPS. Subjects reported to the laboratory during the early evening, after having refrained from resistance training for 5 d. They performed a series of unilateral arm curl exercises on an incline bench with a standard weight training dumbbell, as utilized for the 1 RM determination. In order to separately examine the Con and Ecc phases of the exercise, subjects lifted the weight with one arm and lowered it with the other as previously described (Gibala et al. 1995). Arm designation was randomly assigned and
counterbalanced so that 3 subjects performed the Con phase with their dominant arm and 3 with their non-dominant arm. Subjects performed 8 sets of 8 repetitions using a resistance equivalent to 80% of their previously determined Con arm 1 RM, with a 3 min recovery period between sets. The force-time characteristics of the Con and Ecc actions were kept constant by instructing subjects to perform each phase of the exercise in 2.0 s and by providing visual feedback of oscilloscope recordings from electrogoniometers at each elbow. In instances where subjects were unable to complete 8 lifts with the Con arm, the same number of Ecc actions were performed with the opposite arm. Subjects therefore performed the same absolute amount of work with each arm.

Following the exercise bout, subjects were instructed to consume their habitual diet, refrain from strenuous physical activity, and report back to the laboratory during the early afternoon of the following day. Upon arrival at the laboratory, they consumed a standardized 1200 kcal meal formulated from 70% carbohydrate, 14% fat, and 16% protein. Two hours following the meal (~19 h following the exercise bout), a 20-Ga catheter was inserted into a hand vein for blood sampling and a second catheter was positioned in a contralateral forearm vein for infusion of L-[1,2-13C2] leucine (Tracer Technologies, Somerville, Mass.). A priming dose of L-[1,2-13C2] leucine (1 mg·kg−1·body wt) was administered followed by a constant infusion of 1 mg·kg−1·hr−1 for 10 h using a Harvard syringe pump. During the 10 h infusion period subjects remained by a constant infusion of 1 mg·kg−1·hr−1 for 10 h using a Harvard syringe pump. During the 10 h infusion period subjects remained

A fibre that contained >10 focal areas was also given a moderate rating. A fibre that contained >10 continuous sarcomeres and >10 adjacent myofibrils was defined as extreme. A fibre that determined isodense areas was also given an extreme rating. Refer to Gibala et al. (1995) for sample micrographs of the three different ratings. For all categories, the extent of disruption was expressed as a percentage of fibres examined. All analyses were conducted in a blind fashion such that the investigator was aware of the subject’s identity but not the sampling condition.

Determination of MPS

Enrichment of plasma [1,2-13C2]-α-KIC (atom % excess) was determined by electron impact ionization gas chromatography–mass spectrometry (Hewlett-Packard 5980A-MSD) as previously described (Tamopskys et al. 1991). Enrichment of muscle [1,2-13C2]-leucine (mole % excess) was determined by capillary gas chromatography–combustion isotope ratio mass spectrometry as described by Yarasheski et al. (1992). MPS was calculated for each muscle sample according to the method of Nair et al. (1988) using plasma [1,2-13C2]-α-KIC as the precursor pool enrichment as previously described (MacDougall et al. 1992). Data are expressed as %·hr−1, where the incorporation time is the time between biopsies for each subject. Biopsies were taken from both arms 1.5 h after the priming dose and again 10 h after the priming dose so that the mid point of the measurement procedure would occur ~24–25 h after completion of the resistance training bout.

Statistical analyses

Ultrastructural data were analyzed using a one-factor (1 × 3: condition) repeated measures analysis of variance (ANOVA) to examine the total number of disrupted fibres in the baseline, Con, and Ecc samples; and a two-factor (3 × 3: arm × classification) repeated measures ANOVA to examine the severity of fibre disruption in each sample. Plasma enrichment data were analyzed using a one factor (1 × 6; time) repeated measures ANOVA, and MPS data were analyzed using a one-factor (1 × 2; arm) repeated measures ANOVA in order to compare MPS between the Con and Ecc arms. Although the F-test is considered quite robust, due to the relatively small number of subjects a Greenhouse-Geisser adjustment was made to the degrees of freedom in order to correct for the potential violation of the assumption of sphericity. For all analyses, statistical significance was accepted as P < 0.05, and significant interactions and main effects were further analyzed using a Tukey post hoc test. Values are expressed as mean ± SE.

Results

Myofibrillar disruption

The extent of fibre disruption observed at baseline and following exercise in the Con and Ecc arm samples is illustrated in Fig. 1. The total percentage of disrupted fibres in the Ecc arm (45 ± 11%) was greater (P < 0.05) compared with baseline values (4 ± 2%), but not different (P > 0.05) compared with the Con arm (27 ± 4%). Fibre disruption in the Con arm was not significantly different compared with baseline values (P > 0.05). The severity of fibre disruption, indicated by the number of fibres with focal, moderate, and extreme disruption, is illustrated in Fig. 2. The extent of fibre disruption following exercise was primarily restricted to focal and moderate areas, and less than 3% of the fibres examined in either arm showed evidence of extreme disruption. Nonetheless, there were no significant interactions between ratings of fibre disruption.
Discussion

An important, novel finding from the present study is that the lowering (Ecc) phase of an acute bout of heavy resistance exercise results in significant myofibrillar disruption in well-trained individuals. Staron et al. (1992) and Roth et al. (1999) previously reported evidence of myofibrillar disruption in subjects who had trained for 8–9 weeks, but the present results show that significant fibre disruption also occurs in subjects who have trained regularly for a number of years. The overall magnitude and severity of damage, however, was considerably lower than previously observed in an untrained group of subjects following a similar exercise bout (Gibala et al. 1995). Consistent with the findings of that study, our current results indicate that the Ecc phase of weightlifting is most disruptive to skeletal muscle. However, the proportion of fibres which showed evidence of disruption in the present group of trained subjects was approximately half of that observed following a similar Ecc-exercise bout in untrained individuals (Fig. 4). Moreover, in our previous study ~40% of fibres examined from the Ecc arm following exercise showed extreme disruption, the most severe category of damage. In the present group of trained subjects, most areas of disruption were focal or moderate in nature (Fig. 2), and only ~3% of the fibres examined in either arm were classified as extreme. While a proper statistical comparison cannot be made between trained and untrained subjects, the combined results from our two studies suggest that the muscles of resistance-trained individuals may be more resilient to ultrastructural disruption following an acute bout of heavy resistance exercise. These data therefore provide direct morphological evidence in support of previous studies which have reached a similar conclusion based on indirect markers of muscle injury (Ebbeling and Clarkson 1990; Clarkson et al. 1992). Significant fibre disruption nonetheless occurred in these well-trained subjects, however, and this was particularly associated with the Ecc phase of the movement. These observations are consistent with the hypothesis that high specific tensions generated during weightlifting exercise, especially during the Ecc phase, may cause mechanical damage to the contractile apparatus of the fibre (Waterman-Storer 1991; Fridén and Lieber 1992).

Although speculative, our data also allow for comment on the potential time course for repair of exercise-induced contractile protein damage in trained subjects, since a biopsy was also obtained from the biceps brachii of one arm after our subjects had refrained from training for 5 days. A period of 5 days was selected arbitrarily and our assumption was that any occurrence of disrupted fibres in this sample would be the same as that which existed 1 month earlier following 5 days without training. We consider this assumption justified since training of the elbow flexor muscle group for this month was mimicked as closely as possible to that in the month prior to the intervention. Moreover, the volume and intensity of a typical biceps training bout for our subjects was greater than the experimental testing session. That is, 8 sets of Con or Ecc repetitions at 80% 1 RM actually represented less total work than they were accustomed to performing (recall that subjects only performed one phase of the movement with each arm during the experiment). Subjects’ normal training routines consisted of 6–10 sets of arm curl exercises at intensities ≥80% 1 RM, such that each arm normally raised and lowered the weight. Thus, while we did not assess myofibrillar disruption following a typical training session (in order to limit the number of biopsies), it seems likely that the normal training-induced fibre disruption would have been at least as great as after the experimental training bout. Thus, our observation that only ~4% of fibres in these baseline samples showed evidence of disruption suggests that the intervening 5 days was an adequate period of time for repair of contractile protein following a
normal training session. This is consistent with data which indicate that in well-trained subjects, muscle strength is suppressed for several days following a typical high intensity training session but, with inactivity, reaches peak levels at approximately 4–5 days (Gibala et al. 1994). Moreover, it appears that the tissue repair process may be accelerated in trained individuals: in the present group of subjects, there was minimal fibre disruption after 5 days of recovery, whereas a previous study using untrained individuals reported evidence of degenerating and regenerating muscle fibres for up to 14 days following intense Ecc-resistance exercise (Jones et al. 1986). Direct comparisons between studies are difficult, however, due to differences in muscle(s) studied, exercise protocol employed, intensity and volume of contraction, timing of measurements post-exercise, and markers used to assess tissue damage.

The finding that MPS ~24 h following exercise was the same for both the Con and Ecc arms is consistent with recent observations made by Phillips and coworkers (1997), who measured mixed muscle protein synthesis following an isolated bout of Con or Ecc leg extensor exercise. Using an exercise intensity and volume similar to that employed in the present study (8 sets of 8 repetitions at 80% of the Con 1 RM), these authors reported no significant difference between contraction types for either fractional synthetic rate, fractional breakdown rate, or muscle protein net balance in a group of untrained subjects. Moreover, in that study measurements were obtained at the same time post-exercise as in our study (i.e., ~24 h), as well as 3 and 48 h following the bout. Our rationale for choosing the 24 h post-exercise time point was that in previous studies from our laboratory we observed that MPS was elevated in biceps brachii by 109% at 24 h following an acute bout of resistance exercise (Chesley et al. 1992), but returned to within 14% of the (unexercised) control arm by 36 h post-exercise (MacDougall et al. 1995). Unfortunately, we did not include baseline measurements of MPS in our study and therefore cannot determine the magnitude of the exercise-induced increase in MPS. It seems likely that such an increase would have occurred, however, based on previous investigations of changes in MPS after heavy resistance exercise in both trained (Phillips et al. 1999; MacDougall et al. 1995) and untrained individuals (Chesley et al. 1992). Regardless, the present results and those of Phillips et al. (1997) imply that the extent of MPS following heavy resistance exercise may be related more to the absolute load placed on the muscle rather than the type of contraction performed.

In summary, the present findings indicate that the Ecc phase of resistance exercise produces myofibrillar disruption in strength-trained individuals. To our knowledge this is the first morphological evidence of such an effect following an acute bout weightlifting exercise subjects who have trained for a number of years. The overall magnitude and extent of fibre disruption was considerably less severe than previously observed in untrained subjects following a similar exercise bout. The present results also suggest that repair of contractile protein is essentially complete within 5 days following an acute bout of resistance exercise in trained subjects. Finally, for a given absolute load, the type of muscle action performed during exercise does not appear to affect muscle protein synthetic rate ~24 h following the bout.

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