

RAPID REPORT

Differential stimulation of myofibrillar and sarcoplasmic protein synthesis with protein ingestion at rest and after resistance exercise

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We aimed to determine whether there is a differential stimulation of the contractile myofibrillar and the cellular sarcoplasmic proteins after ingestion of protein and how this is affected by resistance exercise. Fasted (FAST) muscle protein synthesis was measured in seven healthy young men with a primed constant infusion of L-[ring-¹³C₆]phenylalanine. Participants then performed an intense bout of unilateral resistance exercise followed by the consumption of 25 g of whey protein to maximally stimulate protein synthesis. In the rested (FED) leg myofibrillar (MYO) protein synthesis was elevated ($P < 0.01$) above FAST at 3 h (~163%) but not at 1 and 5 h ($P > 0.05$). In contrast, MYO protein synthesis in the exercised (FED-EX) leg was stimulated above FAST at 1, 3 and 5 h (~100, 216, and 229%, respectively; $P < 0.01$) with the increase at 5 h being greater than FED ($P < 0.01$). Thus, the synthesis of muscle contractile proteins is stimulated by both feeding and resistance exercise early (1 h) but has a greater duration and amplitude after resistance exercise. Sarcoplasmic (SARC) protein synthesis was similarly elevated ($P < 0.01$) above FAST by ~104% at 3 h in both FED and FED-EX suggesting SARC protein synthesis is stimulated by feeding but that this response is not augmented by resistance exercise. In conclusion, myofibrillar and sarcoplasmic protein synthesis are similarly, but transiently, stimulated with protein feeding. In contrast, resistance exercise rapidly stimulates and sustains the synthesis of only the myofibrillar protein fraction after protein ingestion. These data highlight the importance of measuring the synthetic response of specific muscle protein fractions when examining the effects of exercise and nutrition.

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The ability of amino acids to increase the synthesis of mixed muscle proteins during the transition from a fasted to fed state has been well characterized (Rennie *et al.* 1982; Bohe *et al.* 2001, 2003; Paddon-Jones *et al.* 2004; Fujita *et al.* 2007). However, less is known about the stimulatory effect of dietary amino acids on the synthesis of specific muscle protein fractions. For example, myofibrillar protein synthesis responds to dietary amino acids in a dose-related manner but becomes refractory to persistent hyperaminoacidaemia (Bohe *et al.* 2001, 2003; Cuthbertson *et al.* 2005). Sarcoplasmic protein synthesis is also responsive to dietary amino acids but may be less sensitive to nutrient availability than myofibrillar proteins (Mittendorfer *et al.* 2005; Cuthbertson *et al.* 2005). Over-

all, we still do not know the amplitude or duration of the enhanced synthetic response after a discrete meal (i.e. protein bolus).

Resistance exercise has been shown to increase the synthesis of mixed muscle proteins (that must include myofibrillar proteins) and feeding potentiates this response (Borsheim *et al.* 2002; Tipton *et al.* 2003; Wilkinson *et al.* 2007; Dreyer *et al.* 2008; Moore *et al.* 2009); presumably, this is the basis for an expansion of the muscle protein pool that, over time, leads to hypertrophy (Tipton *et al.* 2003; Hartman *et al.* 2007; Wilkinson *et al.* 2007). Most acute studies have reported the anabolic effect of nutrients after exercise as a stimulation of mixed muscle protein fraction (Tipton *et al.* 2003; Wilkinson

et al. 2007; Dreyer *et al.* 2008; Drummond *et al.* 2008). We, and others, have reported that resistance exercise stimulates myofibrillar protein synthesis in the fasted state (Kim *et al.* 2005) and in the presence of a constant supply of amino acids (Louis *et al.* 2003; Moore *et al.* 2005; Cuthbertson *et al.* 2006; Wilkinson *et al.* 2008). What we remain uncertain about are the latency, amplitude, and duration of acute changes in the synthesis of myofibrillar proteins in response to bolus protein ingestion after resistance exercise.

The purpose of this study was to examine the acute changes in muscle protein synthesis at rest after ingestion of a bolus of dietary protein. Additionally, the synthetic response of both the myofibrillar and non-myofibrillar muscle protein fraction was measured to determine the relative stimulation after protein ingestion both at rest and after resistance exercise. We hypothesized that resistance exercise would potentiate the feeding-induced stimulation of protein synthesis primarily in the contractile myofibrillar protein fraction.

Methods

Subjects

Seven healthy recreationally active males (26 ± 3 years; 84.8 ± 4.5 kg; 1.76 ± 0.05 m; means \pm S.E.M.) volunteered to participate in the study. Participants reported engaging in recreational lower body resistance exercise no more than once per week for the prior 3 months. Participants were informed about the experimental procedure to be used as well as the purpose of the study and all potential risks prior to obtaining written consent. All participants were deemed healthy based on their response to a routine medical screening questionnaire. The study carried approval by the local Research Ethics Board of McMaster University and Hamilton Health Sciences and conformed to all standards for the use of human subjects in research as outlined in the 1963 *Declaration of Helsinki* on the use of human subjects in research.

General design

To define the changes in myofibrillar and non-myofibrillar (sarcoplasmic) protein synthesis a primed constant infusion of L-[ring- $^{13}\text{C}_6$]phenylalanine was used to measure basal muscle protein synthesis prior to performing an acute bout of unilateral leg resistance exercise. By using a within-subject design, whereby each individual served as his own rested control, we are better able to attribute the acute changes in protein synthesis to the respective feeding and/or exercise stimuli rather than to interindividual response variation. After exercise, participants consumed a drink containing 25 g of whey

protein to maximally stimulate muscle protein synthesis in both the rested and exercised legs (Cuthbertson *et al.* 2005; Moore *et al.* 2009) and changes in protein synthesis were measured over the subsequent 5 h of recovery.

Infusion protocol

Participants reported to the laboratory at least 1 week prior to the infusion trial to undergo a muscle biopsy procedure for the measurement of baseline protein enrichment. A single biopsy was taken from the vastus lateralis of a randomly selected thigh using a Bergström needle modified for manual suction under local anaesthesia (2% xylocaine). All muscle biopsy samples taken throughout the study were blotted and freed of any visible fat and connective tissue, frozen (within ~ 20 s of excision) in liquid nitrogen, and stored at -80°C until further analysis. On the morning of the trial, participants reported to the laboratory at 07.00 h after an overnight fast and having refrained from any strenuous physical activity for the previous 3 days. A catheter was inserted in the antecubital vein of one arm for blood sampling. After a baseline blood sample was drawn, a second catheter was inserted in the contralateral arm for the application of a primed constant infusion of L-[ring- $^{13}\text{C}_6$]phenylalanine (prime: $2 \mu\text{mol kg}^{-1}$; $0.05 \mu\text{mol kg}^{-1} \text{min}^{-1}$). The participants rested comfortably on a bed throughout the infusion. Blood samples were drawn every 0.5–1 h (Fig. 1) and processed as previously described (Moore *et al.* 2009). At 3 h after the start of the infusion, a single muscle biopsy was taken from the contralateral leg from the baseline biopsy to measure fasted (FAST) rates of protein synthesis. Participants then performed an intense bout of unilateral leg resistance exercise consisting of five sets each of leg press and knee extension using a predetermined weight that elicited voluntary failure in 8–10 repetitions. The exercise bout was completed in 24 ± 1 min. Upon completion of the exercise, a blood sample was drawn and then participants consumed a drink containing 25 g of whey protein to maximize muscle protein synthesis in the rested (FED) and exercised (FED-EX) legs. To minimize disturbances in isotopic equilibrium, drinks were enriched to 6% with tracer according to a measured phenylalanine content of $\sim 3.5\%$ in whey protein. Bilateral muscle biopsies were obtained from separate incisions (~ 5 cm apart) at 1, 3 and 5 h after protein ingestion to characterize the acute changes in protein synthesis in FED and FED-EX. To minimize the number of biopsies, muscle samples were not taken from each leg immediately upon completion of the exercise bout but rather only 1 h after exercise cessation and the subsequent protein ingestion. Therefore, since an additional ~ 24 min of tracer incorporation occurred during the exercise bout at a fasted (FED) rate that would be further suppressed with exercise

(FED-EX), the reported protein synthetic rates measured at 1 h (i.e. ~84 min of incorporation) represent slight underestimations of the true rates of protein synthesis in FED (~10%) and FED-EX (~25%). These average estimates were calculated by comparing our measured rate to an estimated rate of protein synthesis, as determined by extrapolating the basal rate of protein synthesis during exercise in FED and a 30% reduction during exercise in FED-EX (Dreyer *et al.* 2006). However, we have presented the uncorrected data as we did not directly measure the additional tracer incorporation occurring during the brief exercise time. In addition, we speculate that the relatively brief incorporation time during exercise is unlikely to significantly affect the rates of protein synthesis when expressed over 3 and 5 h.

Analyses

Blood amino acid concentrations were analysed by HPLC as previously described (Moore *et al.* 2005). Blood glucose concentration was measured using a commercially available spectrophotometric kit (Standbio Laboratory, Boerne, TX, USA) and plasma insulin concentration was determined using a standard radioimmuno assay kit (Diagnostics Products Corporation, Los Angeles, CA, USA).

Intracellular proteins were isolated from a piece of wet muscle (~30 mg) by hand-homogenizing on ice using a Teflon pestle in a standard 20 mM Tris (pH 7.2) Western blotting buffer (10 μ l mg⁻¹) (Glover *et al.* 2008). The sample was spun for 10 min at 1500 g at 4°C to separate the myofibrillar and collagen proteins. The resultant homogenate was removed and the sarcoplasmic proteins were precipitated by addition of 1 ml of 1 M perchloric acid (PCA). The myofibrillar and collagen pellet was washed once with buffer and the myofibrillar proteins were solubilized by adding 1.5 ml of 0.3 M NaOH and heating to 37°C for 30 min with vortex mixing

every 10 min. Samples were centrifuged for 10 min at 4000 g at 4°C and the supernatant containing the myofibrillar fraction was collected. Myofibrillar protein was precipitated by adding 1 ml of 1 M PCA and spinning for 10 min at 4000 g at 4°C. The sarcoplasmic and myofibrillar proteins were washed twice with 70% EtOH before lyophilization. Amino acids were liberated from myofibrillar and sarcoplasmic proteins by adding 1.5 ml of 6 M HCl and heating to 110°C for 24 h. Free amino acids were purified using cation exchange chromatography (Dowex 50WX8-200 resin; Sigma-Aldrich Ltd) and converted to their *N*-acetyl-*n*-propyl ester derivatives for analysis by gas chromatography combustion-isotope ratio mass spectrometry (GC-C-IRMS: Hewlett Packard 6890; IRMS model Delta Plus XP, Thermo Finnigan, Waltham, MA, USA). Derivatized amino acids were separated on a 30 m DB-1701 column (temperature programme: 110°C for 2 min; 20°C min⁻¹ ramp to 210°C; 5°C min⁻¹ ramp to 280°C; hold for 5 min) prior to combustion.

Intracellular amino acids were isolated from a separate piece of muscle (~20 mg) by homogenizing on ice with 500 μ l of acetonitrile. Samples were centrifuged at 10000 g at 20°C for 10 min and the supernatant containing the intracellular amino acids was collected and dried under nitrogen at 70°C. Amino acids were converted to their heptafluorobutyric (HFB) derivatives before analysis by GC-MS (models 6890 GC and 5973 MS; Hewlett-Packard, Palo Alto, CA, USA). Intracellular phenylalanine enrichment was determined using electron-impact ionization by monitoring ions 316 and 322 (*m* + 0 and *m* + 6, respectively).

Calculations

The fractional synthetic rates (FSR) of myofibrillar and sarcoplasmic protein were calculated using the standard precursor-product method:

$$\text{FSR (\% h}^{-1}\text{)} = \Delta E_p / E_m \times 1/t \times 100$$

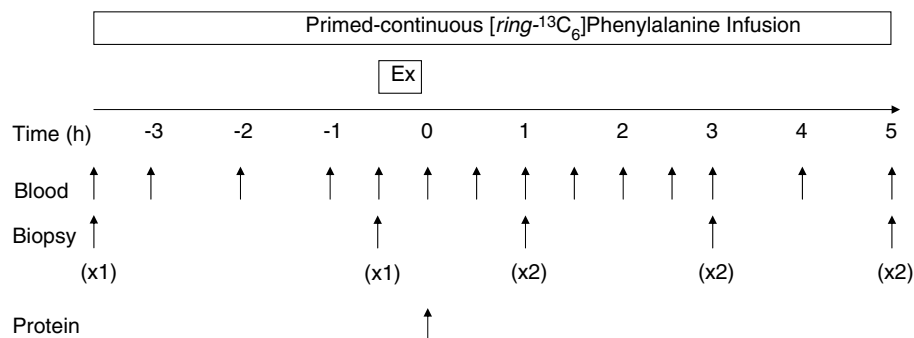


Figure 1. Schematic diagram of the experimental protocol

Single muscle biopsies (1 ×) were taken from alternate thighs to characterize fasted protein synthesis after which a unilateral bout of resistance exercise (EX) was performed. After a drink containing 25 g of whey protein (Protein) was ingested at 0 h, bilateral muscle biopsies (2 ×) were taken from the rested (FED) and exercised (FED-EX) legs.

where ΔE_p is the change in bound protein enrichment between two biopsy samples, E_m is the average enrichment of intracellular phenylalanine across the two biopsy samples, and t is the time between biopsies. Specifically, fasted FSR was calculated from the change in bound protein enrichment between the baseline biopsy and the biopsy taken at 3 h after the start of the infusion using the intracellular phenylalanine enrichment at 3 h. FSR at 1 h was calculated from the change in bound protein enrichment from the fasted biopsy to the 1 h biopsy after feeding in both the FED or FED-EX conditions, respectively. The change in bound protein enrichment between subsequent biopsies within each condition was used to calculate FSR in FED and FED-EX at 1–3 and 3–5 h (i.e. FSR expressed over 2 h intervals). Additionally, FSR in FED and FED-EX was also calculated over 3 and 5 h intervals using the change in bound protein enrichment between the fasted biopsy and biopsies at 3 and 5 h, respectively, to allow for better comparisons between the

present results and those of other studies that typically measure protein synthesis over these longer time frames.

Statistics

Because we used a within-subject study design, myofibrillar and sarcoplasmic protein synthesis were first analysed using a one-factor (1×7 ; FSR) repeated measures ANOVA with a Holm–Sidak *post hoc* test for comparison to control (Fast) to determine whether changes in protein synthesis were elevated above fasted. Myofibrillar and sarcoplasmic protein synthesis were then analysed using a two-factor (2×3 ; condition, time) repeated measures ANOVA to determine significant differences between conditions and across time (i.e. over 2 h intervals). Additionally, myofibrillar and sarcoplasmic protein synthesis expressed over 3 and 5 h were analysed using a one-factor (condition) ANOVA. Differences in the relative stimulation of myofibrillar and sarcoplasmic protein synthesis between conditions were determined by Student's paired *t* test. Blood glucose, plasma insulin, and blood amino acid concentrations were analysed using a one-factor (time) repeated measures ANOVA. Unless otherwise stated, Tukey's *post hoc* test was performed to determine differences between means for all significant main effects and interactions. For all analyses, differences were considered significant at $P < 0.05$. Values are expressed as means \pm S.E.M.

Results

Blood glucose and plasma insulin

Fasted blood glucose was 4.2 ± 0.3 mM and remained stable for the duration of the infusion trial. Fasted plasma insulin concentration was 4.5 ± 0.4 μ IU ml^{-1} and increased by $\sim 190\%$ 1 h after protein ingestion but returned to basal values by 2 h.

Blood amino acid concentration

Blood EAA and leucine concentrations were stable during the fasted condition. After protein ingestion, blood EAA concentration increased immediately at 0.5 h and reached a peak at 1 h after which it declined to basal levels by 3 h (Fig. 2). Blood leucine concentration showed a similar pattern to that of EAA (Fig. 2).

Intracellular precursor labelling

Fasted intracellular phenylalanine enrichment was 3.9 ± 0.3 APE. Intracellular phenylalanine enrichment was stable across time for both FED (4.8 ± 0.3 , 5.4 ± 0.3 and 4.8 ± 0.3 APE at 1, 3, and 5 h, respectively; $P > 0.05$) and

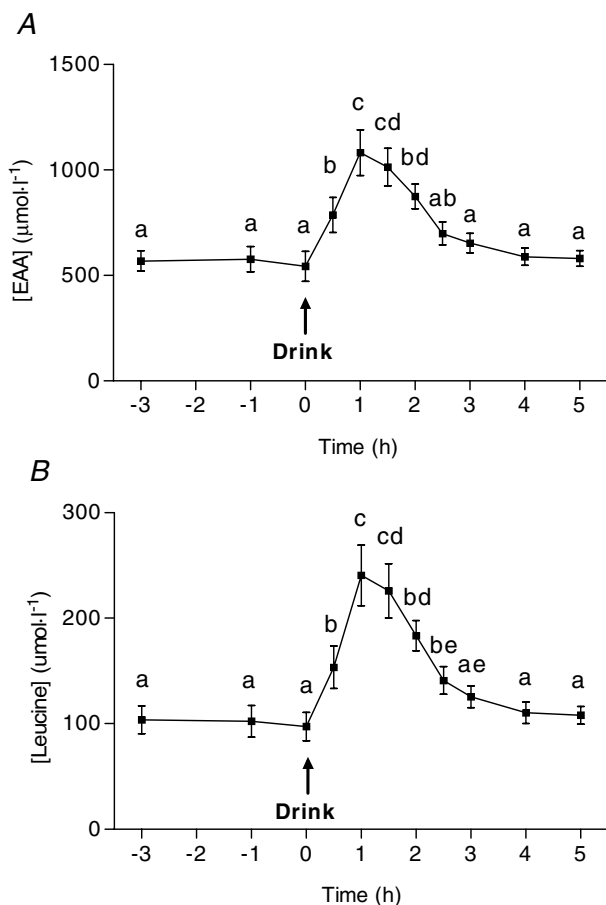


Figure 2. Blood essential amino acid (EAA; top) and leucine (bottom) concentration after ingestion of 25 g of protein (Drink) Values are means \pm S.E.M. ($n = 7$). EAA are sum of His, Ile, Leu, Lys, Met, Phe, Thr, Val (note: Cys not measured). Means with different letters are significantly different from each other, $P < 0.05$.

Table 1. Myofibrillar and sarcoplasmic protein synthesis after feeding at rest (FED) and resistance exercise (FED-EX) expressed over 3 and 5 h

	3 h			5 h	
	Fasted	FED	FED-EX	FED	FED-EX
Myofibrillar					
Rate (% h ⁻¹)	0.025 ± 0.002*	0.051 ± 0.003*†	0.066 ± 0.003†‡	0.049 ± 0.003*†	0.070 ± 0.003†‡
Change (%)	—	112 ± 19	179 ± 36‡§	103 ± 22	204 ± 48‡§
Sarcoplasmic					
Rate (% h ⁻¹)	0.052 ± 0.004	0.086 ± 0.009†	0.084 ± 0.006†	0.074 ± 0.004†	0.077 ± 0.005†
Change (%)	—	71 ± 20	66 ± 15	44 ± 8	51 ± 9

Values are means ± s.e.m. (*n* = 7) for myofibrillar and sarcoplasmic protein synthesis expressed over 3 and 5 h in absolute rate (Rate; % h⁻¹) and relative increase above fasted (Change; %). *Different from sarcoplasmic at same time, *P* < 0.01. †Different from fasted, *P* < 0.01. ‡Different from FED at same time, *P* < 0.01. §Different from sarcoplasmic at same time, *P* < 0.01.

FED-EX (5.8 ± 0.1, 5.7 ± 0.3 and 5.1 ± 0.4 APE at 1, 3 and 5 h, respectively; *P* > 0.05). More importantly, the slope of intracellular phenylalanine enrichment by time curve for both FED and FED-EX was not different from zero (*P* = 0.9 and 0.3, respectively) further confirming all measurements were made at isotopic plateau, the requisite condition for application of the steady-state precursor product equation.

Muscle protein synthesis

In FED, myofibrillar protein synthesis was elevated ~2.6-fold above the fasted state (FAST) only at 1–3 h (*P* < 0.01; Fig. 3). In contrast, FED-EX increased myofibrillar protein synthesis above FAST at 1 h (*P* < 0.05) and was further elevated at 1–3 and 3–5 h (*P* < 0.01) with the increase at 3–5 h being greater than FED (*P* < 0.01). In contrast, sarcoplasmic protein synthesis was similarly elevated (*P* < 0.01) above FAST at 1–3 h in both FED

and FED-EX (~2.2 and 1.9-fold, respectively) with no difference between the conditions (*P* > 0.05; Fig. 3). In FAST, the rate of incorporation of labelled phenylalanine into sarcoplasmic proteins was greater (*P* < 0.01) than myofibrillar (Table 1). There was an expected graded response of feeding and resistance exercise (FAST < FED < FED-EX; all *P* < 0.01) when the rate of myofibrillar protein synthesis was expressed over 3 and 5 h (Table 1); however, the difference between FED and FED-EX was greater over 5 h compared to 3 h (0.020 ± 0.003 versus 0.012 ± 0.004% h⁻¹, respectively; *P* < 0.05, paired *t* test). In addition, although both were greater than FAST (*P* < 0.01), there was no difference (both *P* > 0.05) between FED and FED-EX when sarcoplasmic protein synthesis was expressed over 3 or 5 h (Table 1). Additionally, there was no difference in the increase above fasted of myofibrillar and sarcoplasmic protein synthesis after feeding at rest (FED) with respect to the increase in peak rate (118 ± 19 and 121 ± 33%, respectively; *P* = 0.8;

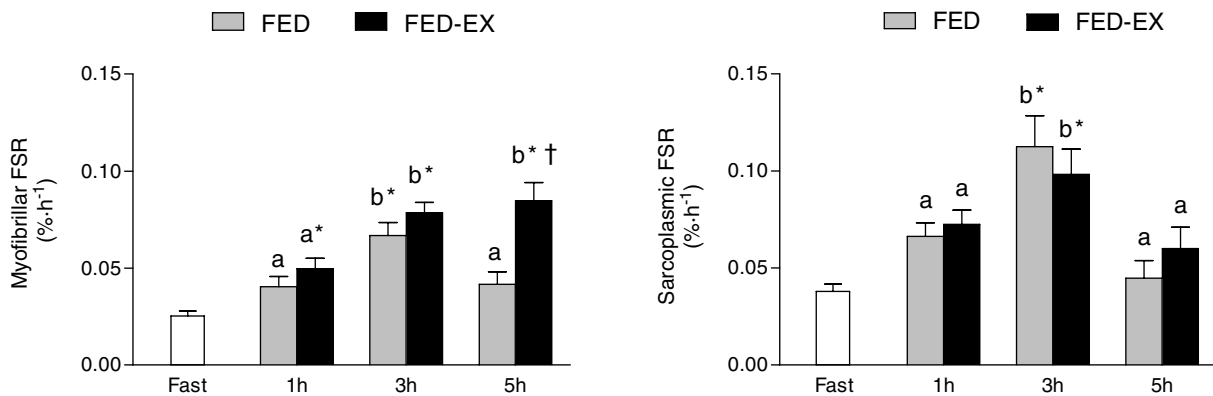


Figure 3. Myofibrillar (top) and sarcoplasmic (bottom) protein synthesis (FSR) in the fasted state (Fast) and after protein ingestion at rest (FED) and after resistance exercise (FED-EX)

Values are means ± s.e.m. (*n* = 7). *Significantly different from Fast, *P* < 0.01. †Significantly different from FED at same time point, *P* < 0.01. Times with different letters are significantly different from each other within each condition, *P* < 0.01.

paired *t* test) or when the rates were expressed over 3 h ($P=0.31$; paired *t* test) or 5 h ($P=0.15$; paired *t* test) (Table 1).

Discussion

Here we report for the first time changes in myofibrillar and non-myofibrillar (sarcoplasmic) protein synthesis after ingestion of a bolus dose of protein. The novel findings of the study were that myofibrillar and sarcoplasmic protein synthesis are similarly, but transiently, enhanced with protein feeding with the stimulation peaking at 3 h and returning to basal levels by 5 h. Additionally, resistance exercise stimulates a rapid (i.e. within 1 h) increase in myofibrillar protein synthesis and sustains the synthetic response of only the myofibrillar protein fraction after discrete protein ingestion.

In response to feeding alone the amplitude and duration of myofibrillar and sarcoplasmic protein synthesis was similar. The acute time course for the stimulation of muscle protein synthesis in the present study is consistent with observed changes in whole body protein metabolism after ingestion of a discrete meal that would contribute to the repletion of fasted state losses in muscle protein balance (Boirie *et al.* 1997; Dangin *et al.* 2001). Our data are in contrast to previous studies reporting that sarcoplasmic protein synthesis is less sensitive to nutrient availability than myofibrillar protein (Mittendorfer *et al.* 2005; Cuthbertson *et al.* 2005). Previous studies measuring changes in both myofibrillar and sarcoplasmic protein synthesis with nutrients provided amino acids either orally, which only induced a fold change in blood EAA concentration, or via a sustained intravenous infusion (Cuthbertson *et al.* 2005; Mittendorfer *et al.* 2005). Accepting that muscle protein synthesis is regulated by acute changes in extracellular amino acid concentration (Bohe *et al.* 2003), it is possible that the 2-fold increase in EAA concentration after ingestion of a bolus of 25 g of protein in the present study enhanced the sensitivity of the sarcoplasmic protein fraction to nutrients as a result of a greater physiological increase in EAA concentration compared to previous studies. Nonetheless, we observed that myofibrillar and sarcoplasmic protein fractions respond in a qualitatively similar fashion with respect to the timing of peak rates and duration of the response suggesting the measurement of mixed muscle protein synthesis is an appropriate surrogate for determining the anabolic effect of a protein meal at rest.

Resistance exercise induced a rapid (i.e. within 1 h) stimulation of myofibrillar protein synthesis that persisted for up to 5 h, which is consistent with the temporal changes measured in mixed muscle protein synthesis and the anabolic nature of the exercise stimulus (Dreyer *et al.* 2006; Drummond *et al.* 2008). The observation that the stimulation of myofibrillar protein synthesis demonstrates

a lag period of ~ 1 h in the fasted state could suggest that the immediate ingestion of dietary amino acids may have contributed to the rapid onset of myofibrillar protein synthesis after resistance exercise in the present study (Kumar *et al.* 2008). Interestingly, there was little additional stimulation in the amplitude of myofibrillar protein synthesis with feeding after exercise compared to feeding at rest suggesting that muscle contractile proteins are primarily responsive to physical stress/stretch after resistance exercise. These data would seem at odds with studies reporting a synergistic effect of exercise and nutrition on muscle protein synthesis (Biolo *et al.* 1997; Koopman *et al.* 2005; Tang *et al.* 2007; Dreyer *et al.* 2008; Moore *et al.* 2009). It is possible that the synergistic effect of exercise and nutrition previously reported in mixed muscle may be related to the amino acid-induced increase in sarcoplasmic protein synthesis, which in our hands is responsive only to nutrients after exercise. Notwithstanding, in light of recent work in the fasted state (Kumar *et al.* 2008), our data demonstrate it is the duration and not the amplitude for which the synergism of exercise and nutrition is observed in the myofibrillar protein fraction. Our decision to measure changes in protein synthesis over 2 h intervals likely afforded us the temporal resolution necessary to accurately measure changes in the peak rates of protein synthesis. For instance, when the present data are expressed over 3 and, especially, 5 h the expected increase in myofibrillar protein synthesis to ingestion of dietary amino acids after resistance exercise was observed (Table 1); these data suggest the timing of muscle biopsies, as has been highlighted by Bohe *et al.* (2001) at rest, should be considered in studies investigating the combined effects of exercise and nutrition. Nonetheless, the sustained synthesis of the myofibrillar protein fraction with protein feeding and resistance exercise is likely to represent an adaptive response to exercise within the contractile proteins that, over time, would ultimately translate into training-induced myofiber hypertrophy.

We found that resistance exercise had no additional stimulatory effect on the synthesis of the sarcoplasmic protein fraction after physiological feeding. This is in contrast to earlier work reporting an elevation in the synthesis of non-myofibrillar proteins after exercise (Louis *et al.* 2003; Miller *et al.* 2005; Cuthbertson *et al.* 2006; Wilkinson *et al.* 2008). Relative to the present bolus protein ingestion, it is possible that the previous studies may have provided an optimal, albeit non-physiological, anabolic environment for the synthesis of sarcoplasmic and mitochondrial proteins after exercise as a result of a feeding regime that induced a sustained hyperaminoacidaemia (Louis *et al.* 2003; Miller *et al.* 2005; Cuthbertson *et al.* 2006; Wilkinson *et al.* 2008). However, we cannot discount the possibility that a greater exercise volume (20 versus 10 sets to failure) (Louis *et al.* 2003) or

a more aerobic-based exercise stimulus (Miller *et al.* 2005; Cuthbertson *et al.* 2006) in previous studies may have induced adaptations typically associated with endurance exercise such as, for example, mitochondrial biogenesis, angiogenesis, and/or the synthesis of metabolic enzymes and metabolite transporters (Dubouchaud *et al.* 2000; Prior *et al.* 2004; Wilkinson *et al.* 2008), which could have been included in the non-myofibrillar protein fraction and therefore contributed to the acute postexercise increase in sarcoplasmic protein synthesis.

In summary, our data demonstrate that the synthesis of myofibrillar and sarcoplasmic protein fractions are similarly elevated after ingestion of a physiologically discrete protein bolus. Resistance exercise induces a rapid and sustained increase in the synthesis of myofibrillar proteins but fails to augment the synthetic response of the sarcoplasmic protein fraction after feeding. These data highlight the importance of measuring the response of specific protein fractions when investigating the combined effects of exercise and nutrition.

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