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Protein supplementation before and after exercise does not further augment skeletal muscle hypertrophy after resistance training in elderly men

L. B. Verdijk, R. A. Jonkers, B. G. Gleeson, M. Beelen, K. Meijer, H. H. Savelberg, W. K. Wodzig, P. Dendale and L. J. van Loon
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Coingestion of Carbohydrate and Protein Hydrolysate Stimulates Muscle Protein Synthesis during Exercise in Young Men, with No Further Increase during Subsequent Overnight Recovery

M. Beelen, M. Tieland, A. P. Gijsen, H. Vandereydt, A. K. Kies, H. Kuipers, W. H. M. Saris, R. Koopman and L. J. C. van Loon
J. Nutr., November 1, 2008; 138 (11): 2198-2204.
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Protein coingestion stimulates muscle protein synthesis during resistance-type exercise

M. Beelen, R. Koopman, A. P. Gijsen, H. Vandereydt, A. K. Kies, H. Kuipers, W. H. M. Saris and L. J. C. van Loon
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Effect of glucose supplement timing on protein metabolism after resistance training

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Roy, B. D., M. A. Tarnopolsky, J. D. MacDougall, J. Fowles, and K. E. Yarasheski. Effect of glucose supplement timing on protein metabolism after resistance training. *J. Appl. Physiol.* 82(6): 1882–1888, 1997.—We determined the effect of the timing of glucose supplementation on fractional muscle protein synthetic rate (FSR), urinary urea excretion, and whole body and myofibrillar protein degradation after resistance exercise. Eight healthy men performed unilateral knee extensor exercise (8 sets/~10 repetitions/~85% of 1 single maximal repetition). They received a carbohydrate (CHO) supplement (1 g/kg) or placebo (Pl) immediately ($t = 0$ h) and 1 h ($t = +1$ h) postexercise. FSR was determined for exercised (Ex) and control (Con) limbs by incremental L-[1-¹³C]leucine enrichment into the vastus lateralis over ~10 h postexercise. Insulin was greater ($P < 0.01$) at 0.5, 0.75, 1.25, 1.5, 1.75, and 2 h, and glucose was greater ($P < 0.05$) at 0.5 and 0.75 h for CHO compared with Pl condition. FSR was 36.1% greater in the CHO/Ex leg than in the CHO/Con leg ($P =$ not significant) and 6.3% greater in the Pl/Ex leg than in the Pl/Con leg ($P =$ not significant). 3-Methylhistidine excretion was lower in the CHO (110.43 ± 3.62 $\mu\text{mol/g}$ creatinine) than Pl condition (120.14 ± 5.82 , $P < 0.05$) as was urinary urea nitrogen (8.60 ± 0.66 vs. 12.28 ± 1.84 g/g creatinine, $P < 0.05$). This suggests that CHO supplementation (1 g/kg) immediately and 1 h after resistance exercise can decrease myofibrillar protein breakdown and urinary urea excretion, resulting in a more positive body protein balance.

muscle protein synthesis; protein degradation; 3-methylhistidine; urea nitrogen; and insulin

MUSCLE GROWTH in adult humans results from muscle fiber hypertrophy (11). Hypertrophy is the result of an increased net muscle protein balance [i.e., fractional muscle protein synthetic rate (FSR) – muscle protein degradation rate (MPD)]. Both FSR (4, 6, 30) and MPD (4) can be stimulated by heavy-resistance exercise in humans. It is also known that amino acid transport is increased after resistance exercise (3). Further understanding of the factors influencing net protein balance may allow the ability to maximize FSR and minimize MPD, thus maximizing the rate and amount of muscle hypertrophy.

Research in the area of resistance training and its effects on FSR and MPD is limited. Recent work from independent laboratories has shown that FSR was elevated after a bout of resistance training in humans (4, 6). Net protein balance, although more positive, was still negative after resistance exercise in the fasted state (4). In addition, it has been demonstrated that in the fed state, strength-trained individuals have a net positive whole body protein balance and an elevated

whole body protein synthesis rate (WBPS) and amino acid flux compared with sedentary individuals (23). These inconsistencies may relate to the availability of amino acids and energy status during the hyperinsulinemic state (2).

It appears that, when insulin is combined with increased amino acid delivery, FSR and WBPS are increased (2). The importance of insulin in suppressing or attenuating the increase in MPD after exercise may be of particular importance in the postexercise period (4).

Studies to date have not addressed the potential interaction of resistance exercise and insulin/nutritional state on leucine turnover/protein balance. Because insulin may cause a decrease in MPD, and a possible increase in FSR, and resistance exercise is known to increase FSR, it is possible that insulin could decrease MPD and increase FSR simultaneously after a bout of resistance exercise. If the latter occurs in combination with the increase in FSR due to the exercise (4, 6), the net protein balance would be even more positive, thus resulting in a greater net accretion of myofibrillar protein. The consumption of a carbohydrate (CHO) supplement is a simple method of increasing insulin concentrations after exercise (9, 32).

We hypothesized that a CHO supplement consumed immediately after resistance exercise would result in 1) decreased urinary 3-methylhistidine (3-MH) excretion (a marker of MPD), 2) increased muscle [¹³C]leucine incorporation rate (increased FSR), and 3) decreased urinary urea nitrogen excretion (net positive protein balance).

METHODS

Subjects. Eight healthy young (aged 20–25 yr) men who had been participating in a resistance training program for at least 1 yr before the investigation (≥ 2 times/wk) were recruited as subjects (Table 1). The experimental procedures, possible risks, and benefits were explained to each volunteer before written consent was obtained. The study was approved by the McMaster University Human Ethics Committee.

Design. Each subject participated in a placebo-controlled randomized double-blind trial with a postexercise CHO supplement and a placebo (Nutrasweet; Pl) trial. They performed unilateral knee extensor exercise such that the muscles of the nonexercised limb served as a control [exercise (Ex) and rest (Con) leg]. One week before the two trials, the subjects' single maximal repetition (1 RM) strength was determined for knee extension and leg press, and their body density was determined by hydrostatic weighing. In addition, subjects completed 4-day diet records, which were analyzed by using a nutritional analysis software package (Nutritionist III, First Data Bank, San Bruno, CA). From this, a dietary checklist was created for each subject. The diets were isoenergetic,

Table 1. *Subjects' descriptive data*

Subject No.	Age, yr	Mass, kg	Height, cm	%Body Fat	Energy, kcal/day	%CHO	%Fat	%Pro
1	24	73.5	176	16.8	2990	68	22	10
2	27	77	175	11.2	2863	63	25	12
3	21	76.3	174	15.6	2674	64	23	12
4	19	79.6	172	18.1	2533	65	24	12
5	21	73.8	182	21.4	3176	62	26	12
6	18	63	167	12.7	3091	61	27	13
7	21	87.4	187	17.8	2593	69	20	11
8	21	69.2	173	18.4	2056	74	16	10
Mean \pm SD	21.5 \pm 2.8	75.0 \pm 7.2	175.8 \pm 6.2	16.5 \pm 3.3	2747 \pm 363.9	65.8 \pm 4.3	22.9 \pm 3.6	11.5 \pm 1.1

CHO, carbohydrate; Pro, protein.

isonitrogenous, flesh free, and were controlled for the 3 days before each trial (dietary checklist). On the day of the trial, all food was supplied in a prepackaged form and consumed in three distributed meals. For the CHO trial, the subjects received the drink (1 g/kg glucose) immediately and 1 h after the exercise bout and consumed placebo (Nutrasweet) with breakfast. In the PI trial, the subjects consumed a drink (2 mg/kg glucose) with their breakfast and the placebo post-Ex at the same times as CHO. Daily energy and nitrogen consumption was the same for the two trials.

The subjects refrained from any resistance exercise with the legs for 3 days before each trial and any form of exercise for the 2 days before each trial. They consumed meals at 0700/1100/1400 and then reported to the laboratory at 1700 ($t = -120$ min). A 20-gauge catheter was inserted into an antecubital vein for tracer infusion, as described by Tarnopolsky et al. (23, 24). A second catheter was placed into a contralateral dorsal hand vein for "arterialized" blood sampling (hot box at $65 \pm 5^\circ\text{C}$) (16). A primed (0.972 ± 0.03 mg/kg) constant (0.977 ± 0.02 mg \cdot kg $^{-1}$ \cdot h $^{-1}$) infusion of L-[1- ^{13}C]leucine (Masstrace, Somerville, MA) was used to determine mixed muscle protein FSR as previously described (6, 23, 30). The infusate was passed through an antibacterial filter (0.2 μm ; Acrodisc) immediately before infusion. At ~ 1830 ($t = -30$ min), subjects performed the prescribed weight training bout [4 sets each of unilateral leg press and knee extension, 85% of 1 maximal repetition (RM), 8–10 repetitions]. The 90 min between the start of the infusion and the onset of exercise was to ensure that an isotopic plateau

had been achieved (23). Immediately after exercise (~ 1900 ; $t = 0$), a blood sample was drawn, a muscle sample was taken from the vastus lateralis muscle of each leg (post-Ex $_0$, post-Con $_0$) by using a suction-modified Bergstöm biopsy needle (Stille), and the glucose (1 g/kg) or Nutrasweet drink was consumed. Blood samples (4 ml) were collected every 15 min for the next hour and immediately centrifuged and stored at -50°C . At ~ 2030 ($t = +1$ h) a second CHO (1 g/kg) or PI drink was administered. Blood samples were again collected every 15 min for the next 1.5 h and again at ~ 0400 , 0430, and 0500 the next morning. Final biopsy specimens were taken at ~ 0500 (post-Ex $_{10}$, post-Con $_{10}$; ~ 10 -h incorporation time). The subjects also collected all urine excreted during the 24-h period (~ 12 h pre-Ex, ~ 12 h post-Ex) for subsequent creatinine, 3-MH, and urea nitrogen determination. Sample collection began in the morning (0600) of the trial (first urination not collected) and continued through to the following morning (0600). Diets were isoenergetic and isonitrogenous during this collection period. The subjects did not leave the laboratory until the final urine sample was collected.

Analysis. Visible fat and connective tissue were removed from the muscle samples, which were then quenched in liquid nitrogen and subsequently stored at -70°C . The L-[^{13}C]leucine enrichment in mixed muscle protein was determined by using gas chromatography/combustion/isotope ratio mass spectrometry (GC/C/IRMS) as described by Yarasheski et al. (29). Blood was analyzed for plasma glucose [kit 315, coefficient of variation (CV) = 3.9%, Sigma Diagnostics, St. Louis MO] and insulin concentration (radioimmunoassay, CV = 2.9%, Diagnostic Products, Los Angeles, CA). Plasma α -[^{13}C] ketoisocaproic acid (α -KIC) was prepared as the trimethylsilyl-quinoxalinol derivative. Its isotopic enrichment was determined with the use of electron-impact ionization capillary gas chromatography/mass spectrometry (GC/MS) by using selected ion monitoring of mass-to-charge ratio 233/232. Urinary urea nitrogen and creatinine excretion were determined from aliquots of the 24-h urine collections by using colorimetric methods as described by Tarnopolsky et al. (23) (kits 640 and 555, CV = 4.7 and $<1\%$, respectively, Sigma Diagnostics). 3-MH concentration was determined by using an automated amino acid analyzer and was normalized to the 24-h urinary creatinine excretion (Beckman Instruments, Palo Alto, CA).

Calculations. Muscle FSR was calculated according to the equation

$$\text{FSR} = (\text{Le}_m \times 100) / (\text{K}_{\text{ep}} \times t)$$

where muscle protein FSR is measured in percentage per hour, t is the incorporation time (in h) between muscle samples taken from the same leg, Le_m is the increment in ^{13}C abundance in leucine from mixed muscle protein obtained

Table 2. *Infusion data*

Subject No.	Trial No.	Time, h	Concn, mg/ml	Rate, mg/h	Rate, mg \cdot kg $^{-1}$ \cdot h $^{-1}$
1	1	11.08	7.75	84.35	1.12
1	2	13.87	5.54	68.08	0.86
2	1	11.33	6.63	70.34	0.95
2	2	12.53	6.71	64.76	0.89
3	1	11.35	7.08	75.18	0.98
3	2	11.17	7.17	77.40	1.01
4	1	11.76	7.30	75.31	0.94
4	2	11.28	6.65	71.24	0.90
5	1	11.3	7.23	77.72	1.05
5	2	11.8	7.13	73.20	1.00
6	1	11.38	6.36	67.63	1.07
6	2	12.3	6.49	64.75	1.03
7	1	11.52	7.32	76.86	0.88
7	2	12.83	7.90	80.46	0.92
8	1	11.5	6.69	70.75	1.03
8	2	11.52	6.63	69.49	1.00
Mean \pm SE		11.78 \pm 0.19	6.91 \pm 0.14	72.97 \pm 1.4	0.977 \pm 0.019

Concn, concentration.

between the muscle samples removed from each leg, and K_{ep} is the mean plasma α - ^{13}C]KIC enrichment for $t = 2.5$ -, ~ 10 -, and ~ 10.5 -h blood samples (corrected for natural abundance of α - ^{13}C]KIC in the $t = 0$ -h blood sample). Leucine flux (\dot{Q}) was calculated by using the reciprocal pool model (8), at isotopic plateau

$$\dot{Q} = i[(E_i/E_p) - 1]$$

where $i = L$ - ^{13}C]leucine infusion rate ($\mu\text{mol} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$), E_i is enrichment of the infused leucine, E_p is enrichment of the plasma α - ^{13}C]KIC (atom percent excess), and the term “ -1 ” corrects for the contribution of the infused isotope to \dot{Q} . The rate of whole body protein degradation (WBPD) was estimated from \dot{Q} based on the equation

$$\dot{Q} = B + I$$

where B is the rate of appearance of endogenous leucine, and I is the dietary leucine intake. Because the subjects had no dietary intake of leucine during the infusion, $I = 0$; thus $B = \dot{Q}$.

Statistical analysis. Muscle and blood data were analyzed by using repeated-measures analysis of variance (time \times treatment; GB-STAT version 5.30, Dynamic Microsystems). When a significant interaction occurred, Tukey's post hoc analysis was used to locate the pairwise differences. Area under the curve (insulin, glucose) was calculated with a custom-made software package. Urine and area under the curve data were analyzed by using paired t -tests. $P < 0.05$ was selected as being indicative of statistical significance. Values are expressed as means \pm SE.

RESULTS

There were no differences in plasma insulin concentrations at the beginning ($t = -1.5$ h) and end of the infusion ($t = \sim 10$ h; CV = 2.9%). Plasma insulin concentrations were significantly higher for the CHO compared with the PI condition at the +0.5-, +0.75-, +1.25-, +1.5-, +1.75-, and +2.0-h time points ($P < 0.01$) (Fig. 1). The area under the insulin curve over the first 2.5 h was ~ 4 times greater for the CHO condition compared with PI [$65.2 \pm 12.1 \mu\text{IU} \cdot \text{h}^{-1} \cdot \text{ml}^{-1}$ for CHO and $15.2 \pm 2.1 \mu\text{IU} \cdot \text{h}^{-1} \cdot \text{ml}^{-1}$ for PI ($P < 0.01$)] (Fig. 2).

Plasma glucose concentrations were not significantly different between CHO and PI before the beginning of ($t = -1.5$ h) and at the end of the isotope infusion (Fig. 3). At the completion of exercise, plasma glucose levels were greater than baseline values for both the CHO and PI conditions [6.26 ± 0.40 to 6.73 ± 0.54 mmol/l for CHO and 5.70 ± 0.55 to 6.50 ± 0.25 mmol/l for PI ($P < 0.05$)]. Plasma glucose concentration was also significantly higher ($P < 0.01$) at +0.5 and +0.75 h in the CHO condition compared with PI. The area under the curve for glucose in the first 2.5 h was significantly greater for CHO compared with PI ($P < 0.01$; CHO = 7.21 ± 0.43 mmol \cdot h $^{-1} \cdot$ l $^{-1}$ and PI = 5.88 ± 0.16 mmol \cdot h $^{-1} \cdot$ l $^{-1}$) (Fig. 4).

Twenty-four-hour urinary creatinine excretion was not significantly different between the two conditions ($n = 7$; 1.76 ± 0.15 g/24 h for CHO and 1.70 ± 0.09 g/24 h for PI). Because these values were not significantly different, the remainder of the urinary results were expressed relative to the creatinine values. 3-MH excretion was significantly lower for the CHO condition vs.

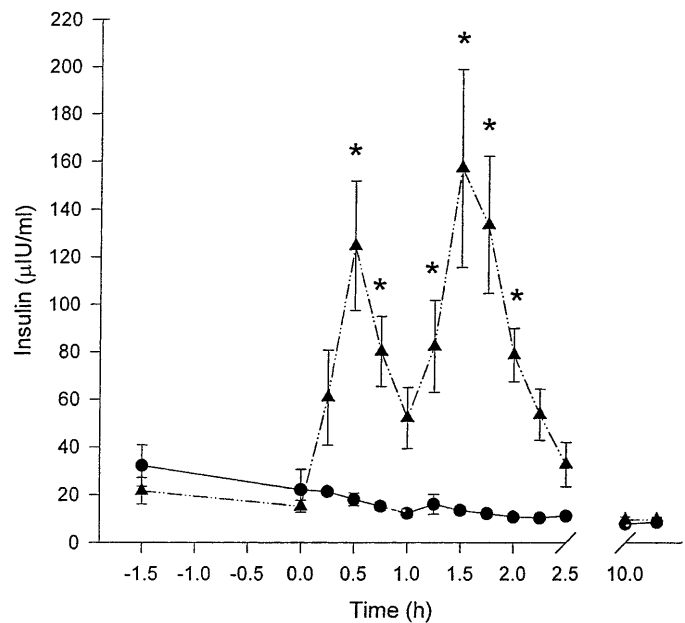


Fig. 1. Plasma insulin values for both carbohydrate (CHO; ▲) and placebo (PI; ●) with respect to time. Values are means \pm SE; $n = 8$. * $P < 0.01$ between 2 conditions.

PI ($P < 0.05$; $n = 7$; 110.43 ± 3.62 and 120.14 ± 5.82 $\mu\text{mol/g}$ creatinine, respectively) (Fig. 5A). A similar difference was observed for urinary urea nitrogen ($P < 0.05$; $n = 7$; CHO = 8.60 ± 0.66 g/g creatinine and PI = 12.28 ± 1.84 g/g creatinine) (Fig. 5B).

Plasma α - ^{13}C]KIC enrichment at each sampling point is shown in Fig. 6 ($n = 7$). Isotopic equilibrium was achieved for each individual subject [CV = $< 10\%$, slope = not significant (NS)] and maintained for the duration of the infusion, as expected from previous work (24).

Compared with the control leg muscle, FSR in the exercised vastus lateralis muscle was elevated by 36.1% in the CHO condition and by 6.3% in the PI condition (NS; $n = 6$) (Fig. 7).

No significant difference was observed for whole body leucine flux between the two conditions ($n = 7$; 115.37 ± 5.65 $\mu\text{mol} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ for CHO and 113.07 ± 4.05 $\mu\text{mol} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ for PI).

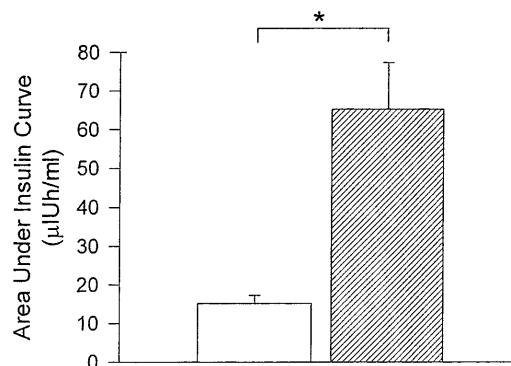


Fig. 2. Area under insulin curve for CHO (hatched bar) and PI (open bar). Values are means \pm SE for 1st 2.5 h postexercise; $n = 8$. * $P < 0.01$ between 2 conditions.

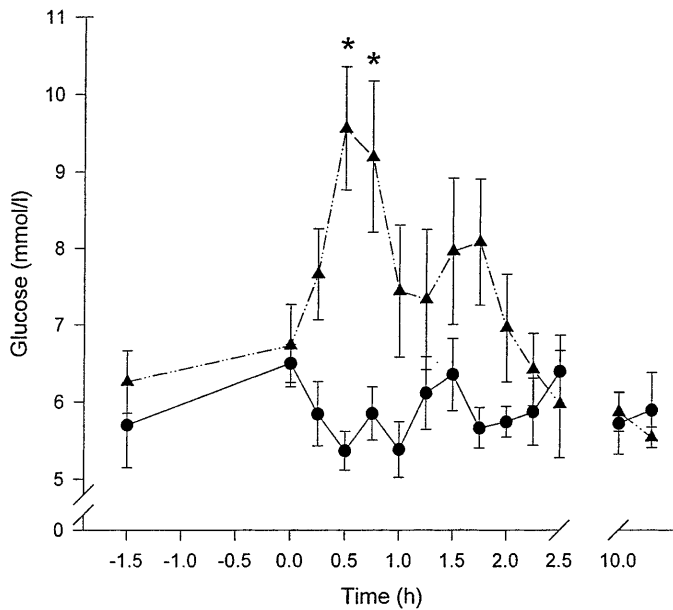


Fig. 3. Plasma glucose values for both CHO (▲) and PI (●) with respect to time. Values are means \pm SE; $n = 8$. * $P < 0.05$ between 2 conditions.

DISCUSSION

The purpose of this investigation was to determine the effect of glucose supplementation timing when given immediately after a bout of resistance exercise on FSR, MPD, WBPD, and urinary urea excretion. A glucose supplement of 1 mg/kg (immediately and +1 h postexercise) resulted in a significant increase in plasma glucose and insulin concentrations as seen by others (9, 32). This was associated with less urinary 3-MH and urea nitrogen excretion, with no difference in vastus lateralis FSR or WBPD. The net effect was anabolic and would result in a more positive net muscle protein balance.

Most of the work in the area of insulin and its effects on protein turnover has involved the use of insulin and glucose infusions (3, 13, 14, 17). The present study is the first report in humans of the influence of oral glucose supplementation on post-resistance-exercise protein metabolism and has practical implications for

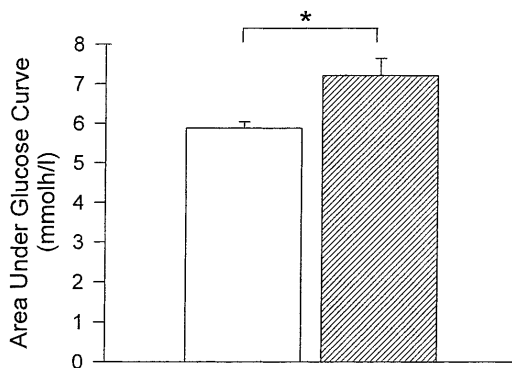


Fig. 4. Area under glucose curve for CHO (hatched bar) and PI (open bar). Values are means \pm SE for 1st 2.5 h postexercise; $n = 8$. * $P < 0.01$ between 2 conditions.

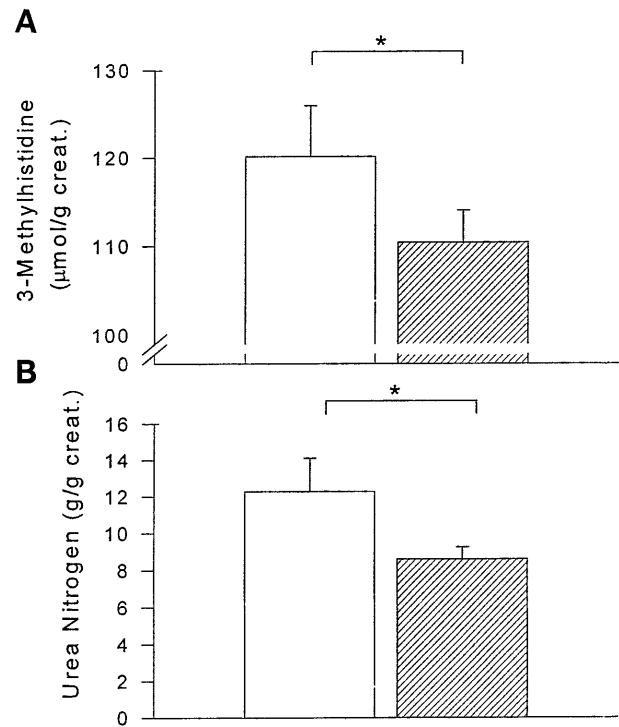


Fig. 5. Urine results (24 h). A: 3-methylhistidine for both PI (open bar) and CHO (hatched bar). B: urinary urea nitrogen for both PI and CHO. Values are means \pm SE; $n = 8$. creat, Creatinine. * $P < 0.01$ between 2 conditions.

athletes and persons performing therapeutic exercise. The positive effects of supplementation on protein metabolism were achieved from a simple redistribution of the timing of the subject's habitual caloric intake.

The administration of a CHO drink led to a significant decrease in urinary 3-MH excretion over the day of the study. We interpreted this as a reduction in MPD. This finding is supportive of some (13, 18) but not all (3, 14) previous studies of the effect of elevated insulin on MPD. An advantage of 24-h urinary 3-MH excretion over the arteriovenous balance technique is the length of time over which the determination occurs. The

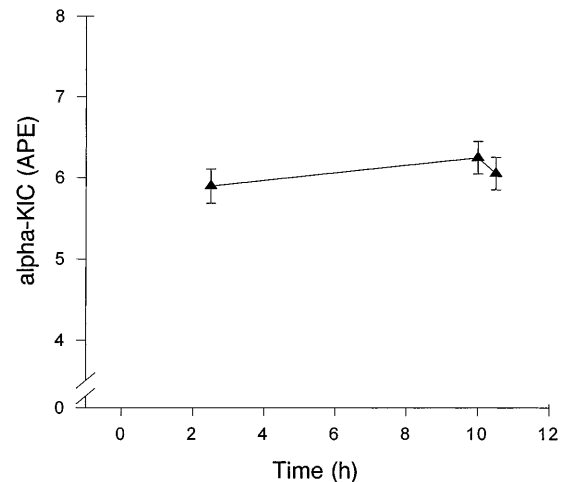


Fig. 6. Plasma ketoisocaproic acid (α -KIC) enrichment across time. Values are means \pm SE; $n = 6$. APE, atom % excess.

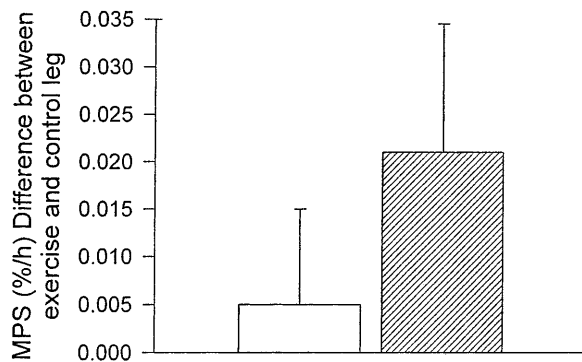


Fig. 7. Muscle protein synthetic rate (MPS; difference between exercise and control leg) for both PI (open bar) and CHO (hatched bar). Values are means \pm SE; $n = 8$.

longer collection duration for the urinary excretion method is advantageous in assessing MPD over the entire postexercise recovery period and thus is useful in determining the impact of an intervention on WBPD. Others have also considered 3-MH to be indicative of MPD (5, 27, 31).

Concerns about the validity of 3-MH excretion as an indicator of MPD relate primarily to the contribution of nonmuscle sources (skin/splanchnic/dietary) to the 3-MH pool (20). Two other sources of error in using 3-MH determinations are a lack of dietary controls and failure to account for interindividual differences in the ratio of nonmyofibrillar/myofibrillar contributions to total urinary 3-MH excretion (10, 20, 27). In the present study, the subjects were on a controlled flesh-free diet for 3 days before each trial and a prepackaged flesh-free diet on the day of each trial (10). Furthermore, we used a crossover repeated-measures design and, therefore, the interindividual variation in contribution from skeletal muscle vs. non-skeletal muscle protein to urinary 3-MH would be constant.

It has been demonstrated in humans with infection that the contribution of splanchnic sources to the 3-MH pool is relatively small during periods of extensive catabolism (21, 22). On the other hand, under periods of hyperinsulinemia and hyperaminoacidemia there appears to be a reduction in nonmyofibrillar protein breakdown in resting humans (14). However, in the postexercise state, when protein breakdown is increased (4), it is not known whether insulin attenuates this in myofibrillar proteins.

In summary, the provision of a glucose supplement immediately post-resistance exercise decreases urinary 3-MH excretion. Without arteriovenous differences measurements, the source of this reduction cannot be ascertained with certainty.

The decrease in 3-MH excretion was accompanied by significantly lower urinary urea nitrogen excretion, which suggested a reduction in amino acid transamination and oxidative deamination because urinary urea excretion is determined by the concentration of urea in the plasma and the glomerular filtration rate (7). We assumed that the glomerular filtration rate for each subject was similar between trials because dietary energy, protein, fluid intake, and exercise were identi-

cal for each condition. Furthermore, there were no differences in creatinine excretion, and both urea and 3-MH were expressed relative to this. Thus, assuming that sweat and fecal loss did not differ between the two trials (19, 25), whole body nitrogen balance would be more positive for the CHO condition.

Differences in WBPD were not observed between conditions. It appears the CHO treatment did not provide enough of a reduction in MPD to influence WBPD. However, FSR contributes $\sim 25\%$ to WBPS (15), and one could estimate that only $\sim 7\%$ of the total muscle mass was active during the exercise. Therefore, changes in MPD may have contributed too little to influence WBPD due to a dilutional effect. A similar protocol with the use of a whole body exercise stimulus (vs. single leg) may have shown an effect of MPD on WBPD. Alternatively, protein degradation in non-skeletal muscle tissue (i.e., splanchnic) may have changed in an opposite direction and attenuated the influence of MPD on WBPD (28).

Furthermore, the WBPD measurements were taken from α - ^{13}C KIC collected immediately postexercise and near the termination of the infusion ($t = +10$ h; fasted state). It is probable that for the CHO trial there was a reduction in WBPD during the period of hyperinsulinemia postexercise (~ 2 h) and for the PI postbreakfast (2 g/kg CHO) (14). WBPD measurements would have to have been taken for ~ 3 h after each of these time periods to determine whether WBPD was more sensitive to the effect of insulin in the postexercise period. The 3-MH data and the reduction in urea excretion suggested that this may have been the case.

We found that the rates of FSR remained unchanged in response to the administration of CHO. A trend was observed in that the CHO condition led to a nonstatistically significant 36% increase in the difference between the exercise leg and the rest leg (Fig. 7). A positive effect of insulin on FSR has been described by others (3, 17). One factor that may have attenuated an increase in FSR was the fact that the glucose supplement likely caused a decrease in plasma amino acid availability due to elevated insulin. It has been demonstrated that the positive effect of insulin on FSR is seen predominantly with concomitant hyperaminoacidemia (2, 3, 17). Another factor that may have attenuated a positive response from the postexercise glucose supplement was the fact that the insulin was only significantly increased for ~ 2 h after the supplementation, whereas the incorporation time was 10 h. Future studies should use methods that can determine FSR over a period of ~ 4 h (3, 4).

Unforeseen sampling errors led to a decrease in the sample size for the FSR analysis ($n = 6$). Therefore, a type II error may also explain the lack of significant increase. In addition, the exercise stimulus might not have been sufficient to stimulate an increase in FSR in the vastus lateralis. We have previously reported an increase in FSR by using a greater volume of training in a fusiform muscle (biceps brachii) after training (6). Because the vastus lateralis is a pennate muscle that contributes to both knee stabilization and extension,

the force/unit area may have been less than in our previous study using the biceps brachii. A study of female swimmers also found no effect of resistance exercise on FSR by using a muscle that is difficult to fully activate (posterior deltoid) (26). This same group, however, found an increase in FSR in the vastus lateralis by using an almost identical intensity and volume of resistance exercise (4). It should be noted, however, that the subjects in the latter study were untrained (4), and it is possible that this may partially explain the discrepant results. A third possibility is that our measurement of FSR over a 10-h period immediately after exercise may not have included the time points over which FSR is maximal. We know from previous studies that FSR appears to peak at ~24 h after exercise (12).

In summary, our results indicate that consumption of a 1 g/kg CHO supplement immediately and 1 h after completion of a resistance training bout significantly decreased myofibrillar protein breakdown and urinary urea nitrogen excretion, and slightly increased FSR, resulting in a more positive protein balance. This suggests that consumption of a glucose supplement after resistance exercise increases insulin concentration and thus may enhance muscle protein balance.

The authors thank Dr. E. V. Youglai and Marty Gibala for help and technical support.

This study was supported by a Sport Canada Applied Research Grant, the Natural Science and Engineering Research Council of Canada, and Division of Research Resources Grant RR-00954.

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Received 16 September 1996; accepted in final form 10 February 1997.

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