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ORIGINAL ARTICLE

**Darren G. Candow · Philip D. Chilibeck**
**Darren G. Burke · K. Shawn Davison · Truis Smith-Palmer**

# Effect of glutamine supplementation combined with resistance training in young adults

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**Abstract** The purpose of this study was to assess the effect of oral glutamine supplementation combined with resistance training in young adults. A group of 31 subjects, aged 18–24 years, were randomly allocated to groups (double blind) to receive either glutamine (0.9 g·kg lean tissue mass$^{-1}$·day$^{-1}$; $n = 17$) or a placebo (0.9 g maltodextrin·kg lean tissue mass$^{-1}$·day$^{-1}$; $n = 14$) during 6 weeks of total body resistance training. Exercises were performed for four to five sets of 6–12 repetitions at intensities ranging from 60% to 90% 1 repetition maximum (1 RM). Before and after training, measurements were taken of 1 RM squat and bench press strength, peak knee extension torque (using an isokinetic dynamometer), lean tissue mass (dual energy X-ray absorptiometry) and muscle protein degradation (urinary 3-methylhistidine by high performance liquid chromatography). Repeated measures ANOVA showed that strength, torque, lean tissue mass and 3-methylhistidine increased with training ($P < 0.05$), with no significant difference between groups. Both groups increased their 1 RM squat by approximately 30% and 1 RM bench press by approximately 14%. The glutamine group showed increases of 6% for knee extension torque, 2% for lean tissue mass and 41% for urinary levels of 3-methylhistidine. The placebo group increased knee extension torque by 5%, lean tissue mass by 1.7% and 3-methylhistidine by 56%. We conclude that glutamine supplementation during resistance training has no significant effect on muscle performance, body composition or muscle protein degradation in young healthy adults.

**Keywords** Strength · Exercise · Muscle · Men · Women

D.G. Candow · P.D. Chilibeck (✉) · D.G. Burke · K.S. Davison
College of Kinesiology, 105 Gymnasium Place,
University of Saskatchewan, Saskatoon,
Saskatchewan, Canada, S7 N 5C2
E-mail: chilibec@duke.usask.ca
Tel.: +1-306-9666469
Fax: +1-306-9666464

D.G. Burke
Department of Human Kinetics,
St. Francis Xavier University, Antigonish,
Nova Scotia, Canada, B2G 2W5

T. Smith-Palmer
Department of Chemistry,
St. Francis Xavier University, Antigonish,
Nova Scotia, Canada, B2G 2W5

## Introduction

Glutamine, the most versatile and abundant amino acid in plasma and skeletal muscle, accounts for more than half the total intramuscular free amino acid pool (for reviews see Lacey and Wilmore 1990; Curthoys and Watford 1995). During periods of physiological stress such as illness which induce hypercatabolic states, the body's endogenous rate of glutamine synthesis falls below the concentrations required for homeostasis (for reviews see Wagenmakers 1998; Antonio and Street 1999). Thus, glutamine is considered to be a "conditionally" essential amino acid (Lacey and Wilmore 1990).

Glutamine supplementation has become increasingly popular in recent years among athletes because it has been shown to have a beneficial effect on muscle glycogen resynthesis (Varnier et al. 1995; Bowtell et al. 1999), and immune system response (Castell et al. 1996; Castell and Newsholme 1998) following endurance exercise. Following a physiological stress such as surgery, glutamine supplementation maintains muscle glutamine content and either increases muscle protein synthesis or prevents protein catabolism (Hammarqvist et al. 1990; Petersson et al. 1994). Glutamine supplementation appears to increase intramuscular glycogen concentrations following prolonged endurance exercise by acting as a substrate for gluconeogenesis in the liver (Varnier et al. 1995; Bowtell et al. 1999). This increase in muscle glycogen could potentially attenuate amino acid release from skeletal muscle during extended exercise which may reduce muscle protein degradation. Following intense exercise of long duration (such as long-distance

running or rowing), glutamine supplementation has been shown to decrease the incidence of infections (indicative of over-training) and improve the response of cells of the immune system (Castell et al. 1996; Castell and Newsholme 1998). Glutamine supplementation therefore has the potential to prevent the negative effects induced by overtraining, allowing an athlete to maintain training at a greater frequency and intensity. Finally, in animal models glutamine supplementation has been shown to decrease protein degradation and increase protein synthesis (MacLennan et al. 1987, 1988), and in humans prevent a decrease in protein synthesis following surgery (Hammarqvist et al. 1990; Petersson et al. 1994) and in healthy individuals increase protein synthesis (Hankard et al. 1996). These effects may be mediated by an increase in growth hormone after ingestion of glutamine (Welbourne 1995).

In contrast to the above positive findings concerning the ingestion of glutamine, others have found no beneficial effect of orally administered glutamine on glycogen resynthesis following glycogen-depleting exercise (van Hall et al. 2000). Also, a number of studies have indicated that glutamine supplementation has no effect on cells of the immune system following intense exercise (Rohde et al. 1998a, c). Thus it is controversial whether glutamine supplementation has the potential for improving exercise performance and adaptations to training.

Given that strength training results in glycogen depletion (Roy and Tarnopolsky 1998) and an elevated protein turnover (MacDougall et al. 1995), it has been suggested that glutamine supplementation may be of benefit to strength training (Antonio and Street 1999). The purpose of this study was to investigate the effect of oral glutamine supplementation when combined with total body resistance training on muscle performance, lean tissue mass and muscle protein degradation in young, healthy adults.

We hypothesized that when combined with strength training, glutamine supplementation would enhance lean tissue mass and muscle strength and attenuate myofibrillar protein degradation when compared to strength training alone.

## Methods

### Subjects

A group of 40 subjects (21 men and 19 women), aged 18–24 years, were initially recruited for the study. Subjects were recruited from an active population which was participating in resistance training two to four times a week. Individuals were free from other ergogenic aids for at least 6 weeks prior to the initial tests to eliminate any effects from other supplementation. For example, creatine monohydrate has been shown to elicit enhancements in muscle performance for up to 4 weeks post-supplementation (Vandenberghe et al. 1997). The study was approved by the Ethics Review Board of the University of Saskatchewan, Saskatoon, Saskatchewan, Canada and the experiments described in this paper complied with the current laws of Canada. The subjects were informed of the risks and purposes of the study before their written consents were obtained.

### Experiment protocol

The study used a double-blind repeated measures design in which every subject participated in strength training and was allocated randomly to a glutamine or placebo treatment condition. All subjects were required to come into the laboratory on two occasions at the beginning of the 6 week study; once for familiarization with the resistance equipment and the Biodex isokinetic dynamometer and on a second occasion for initial data collection. Subjects returned to the laboratory after 6 weeks of training for post-treatment tests. Prior to the laboratory tests, all participants were instructed to refrain from resistance training for 24 h. They were also instructed to collect urine samples at least 24 h before the tests after a 72 h period during which a meat-free diet (no fish, poultry, beef, pork or processed meat products) was consumed. The purpose of this meat-free diet was to ensure valid baseline levels for urinary 3-methylhistidine (Lukaski et al. 1981) excretion, which was used to assess myofibrillar protein degradation (see below). Subjects were randomly assigned to receive either glutamine powder (0.9 g·kg lean tissue mass$^{-1}$·day$^{-1}$) or placebo (maltodextrin) powder (0.9g·kg lean tissue mass$^{-1}$·day$^{-1}$). The dose of 0.9 g·kg lean tissue mass$^{-1}$·day$^{-1}$ was chosen because it approximated the upper limit that has been shown to be safe without being toxic (Lowe et al. 1990) and is greater than the doses shown to be effective for promoting glycogen resynthesis (Bowtell et al. 1999) and sustaining protein synthesis following surgery (Petersson et al. 1994). Doses were based on lean tissue mass because glutamine is found primarily in skeletal muscle where it is the most abundant amino acid. The dependent variables measured were (1) strength, (2) peak torque, (3) lean tissue mass, and (4) urinary 3-methylhistidine excretion. In addition, the individuals completed dietary records for 3 days prior to each test trial to assess nutrient differences between groups. During the initial and post-treatment test day participants were assessed on two strength measurements of one-repetition maximum (1 RM) squat and bench press. Peak torque was assessed using three sets of one maximal knee extension of the dominant leg on a Biodex isokinetic dynamometer. Lean tissue mass was assessed using dual energy X-ray absorptiometry (DEXA) and muscle protein degradation was assessed from 3-methylhistidine excretion using high performance liquid chromatography (HPLC).

### Supplementation

The glutamine and placebo (maltodextrin) were identical in energy content, colour, taste and texture. The supplements were provided to each participant in bundles of eight bags sufficient for an 8 day cycle. Each bag contained the participant's supplement for 1 day. The subjects were instructed to consume the supplement twice daily, firstly immediately following a workout to promote amino acid uptake and protein synthesis (Tipton et al. 1999) and secondly before going to bed. On rest days, participants were instructed that they could consume the supplement as desired during the day but that the ingestion before going to bed had to be adhered to. Supplements were to be consumed every day for the total 6 weeks and could be mixed in water or juice. Subjects were instructed to mix the glutamine in the water or juice just prior to consumption to avoid degradation of the glutamine. Compliance with the supplementation protocol was monitored by verbal communication with subjects and by having subjects return empty bags when picking up an additional supplement.

### Strength

To measure the 1 RM squat, a squat rack and an Olympic barbell were used. Each subject positioned his/her feet approximately shoulder width apart inside the squat rack and in front of a full body mirror. The position of the placement of subjects' feet was recorded using a grid taped to the floor to allow identical placement during the tests after the 6 weeks of training. Subjects were instructed to lower the squat bar until an internal angle at the knees

of 90° was achieved before returning to the upright position. A warm-up consisted of the modified hurdler's stretch held twice by each leg for 20 s followed by ten repetitions of squat exercise using a weight determined by each subject as an appropriate warm-up weight. For the bench press, the subjects were positioned on the bench with both feet flat on the floor. The subjects were not allowed to lift their buttocks off the bench or arch their backs during a lift. A complete repetition went from the top straight-arm position, down until the bar touched the chest, and then ended with the bar returning to the top straight-arm position. A warm-up consisted of 20 push-ups, two static stretches of the chest musculature against a wall, held for 8 s each, and ten repetitions with a comfortable starting weight as determined by each subject.

Following the warm-ups, for both squat and bench press testing, subjects selected a weight with which they felt they could complete three repetitions. At this weight, they only performed one repetition. Subjects then selected a weight they felt would be their 1 RM. If successful, the weight was increased by 2.3–4.5 kg for subsequent attempts, until his/her 1 RM was reached. The 1 RM was usually reached in four to six sets, including the warm-up set. There was a 3 min rest between sets and two assistants changed the weight on the bar between sets. The reproducibility of the squat and bench press was assessed by testing 1 RM strength on 12 subjects at two occasions, 1 week apart. Coefficients of variation were 5.97% for squat and 1.89% for bench press 1 RM.

### Torque

Torque was measured for the right knee extensor muscles, using an isokinetic dynamometer (Biodex System 3, Biodex Medical Systems Inc., Shirley, NY). The dynamometer was set in the concentric mode for knee extension, at an angular velocity of 60° a second. The range of motion consisted of movement from 90–170° of knee flexion (internal angle). The subjects sat against a back support, producing an angle of 85° of hip flexion. Stabilizing belts were placed over the lap, across the chest, and across the distal one-third of the leg thigh tested. The rotational axis of the dynamometer was positioned to be coaxial with the knee axis (lateral condyle) during the tests. The subjects were initially given several submaximal and several maximal practice trials for familiarization with the equipment. For the actual tests, one repetition of knee extension at maximal effort was repeated three times with 1 min rest between sets. The highest peak torque obtained during the three repetitions was recorded. Torque measurements were corrected for the effects of gravity on the lower leg and the dynamometer's resistance pad. The torque output on the dynamometer was checked weekly with a calibration weight throughout the study duration. Reproducibility was assessed by testing maximal knee extension on 8 subjects, 1 week apart. The coefficient of variation for knee extension peak torque was 2.43%.

### Body composition

Bone mineral-free lean tissue mass was assessed using DEXA at the beginning of the study and following 6 weeks of supplementation and training. Whole-body DEXA scans were performed using a Hologic QDR-2000 in array mode and analysed (excluding the head region) using system software version 7.01. The same technician analysed all DEXA scans. Reproducibility was determined on 10 subjects on two separate occasions. The coefficient of variation for lean tissue mass was 0.54%.

### The 3-methylhistidine analyses

For the measurement of 3-methylhistidine, an index of myofibrillar protein degradation, urine was collected from each individual at two stages, prior to and immediately following 6 weeks of resistance training, in each case following a 72 h meat-free diet. It has been determined that meat consumption affects 3-methylhistidine levels and that at least 3 days of a meat-free diet are required to return urine concentrations of 3-methylhistidine to baseline levels (Lukaski et al. 1981). The designated urine collection procedure was to discard the product of the first urination upon waking in the morning and then collect all samples for the following 24 h, including the first one upon waking up the next morning. Urine samples were brought to the researcher where the individual's urine volume and identification number were recorded. Aliquots of each urine sample were drawn off from the 24 h collection and stored at –20 C until analysed. The 3-methylhistidine was assessed using HPLC (Varian 5000) by the methods of Long et al. (1975) and the amount in a 24 h sample of urine expressed relative to lean tissue mass ($\mu$mol kg$^{-1}$).

### Dietary assessment

Dietary intake was recorded 3 days prior to each test, but on days separate from those of the meat-free diet, to assess whether there were differences in energy intake and macronutrient composition between groups. Participants used a 3 day food booklet to record what they ate for 2 weekdays and 1 weekend day. They were instructed to record all food items and portion sizes consumed for the 3 designated days. The Fuel 2.1 dietary analysis program (Nutrition Software, Logiform Nutrition Sport) was used to analyse the food records. Each item was entered and the program provided the total energy consumption on average over the 3 days as well as the amounts of energy from carbohydrates, proteins, and fats individually.

### Resistance training programme

All subjects followed the same supervised, periodized, free-weight resistance-training programme for 6 weeks. The programme consisted of four to five sets of six to ten repetitions at 60%–90% 1 RM. This programme was designed to induce muscle hypertrophy. We have previously used similar programmes successfully to achieve muscle hypertrophy (Chilibeck et al. 1999; Burke et al. 2001). Weight training started on the first day of supplementation and consisted of a 3 day split routine involving whole body musculature. Day 1 involved chest and triceps musculature and included the following exercises in order: bench press, incline bench press, flat bench dumb-bell flys, incline dumb-bell flys, cable triceps extensions, rope reverse triceps extensions, and French curls. Day 2 involved back and biceps musculature and included the following exercises in order: chin-ups, low row, *lat* pull-downs, alternate dumb-bell row, standing EZ-curls, preacher curls, and alternate dumb-bell curls. Day 3 involved leg, shoulder, and abdominal musculature and included the following exercises in order: squats, leg extensions, hamstring curls, standing calve raises, military dumb-bell press, upright row, shrugs, deltoid flys, and abdominal crunches. Day 4 was a rest day. This 4 day cycle was repeated throughout the duration of the study. Average training volume per session was determined for each subject as resistance×sets×repetitions.

### Statistics

A 2 (placebo vs glutamine)×2 (men vs women)×2 (before vs after training) ANOVA with repeated measures on the 3rd factor was used to determine whether there were any differences between the glutamine and placebo groups over time for the dependent variables of strength, torque, lean tissue mass, 3-methylhistidine excretion and diet (energy and macronutrient contents). An independent sample Student's *t*-test was used to determine whether there was a difference in average training volume between glutamine and placebo groups and to determine whether there were differences in baseline measurements between groups. Statistical significance was set at $P < 0.05$. Statistical analyses were carried out using SPSS version 10.02 for Windows (SPSS Inc., Chicago, Ill).

## Results

Of the 40 subjects originally recruited, 31 subjects completed the study. Of the original participants 7 (3 from the glutamine and 4 from the placebo group) were excluded from data analysis due to lack of compliance with the resistance training programme. Compliance required that each subject should not miss more than 1 exercise day per cycle.

There were 2 participants (1 from each group) who withdrew due to medical reasons. A male participant in the glutamine group could not perform 1 RM bench press after training because of a shoulder injury sustained a day prior to the testing. Therefore, of the 40 original participants, 30 were used for 1 RM bench press data analysis, while 31 (9 men and 8 women in the glutamine group and 8 men and 6 women in the placebo group) were used for data analysis of squat strength, torque, and lean tissue mass. There were 21 subjects (6 men and 4 women in the glutamine group and 6 men and 5 women in the placebo group) who were able to provide urine samples prior to and after the training programme for analysis of 3-methylhistidine. Baseline characteristics of subjects who completed the study are shown in Table 1. There were no significant differences between the glutamine and placebo groups for any of the baseline measurements.

Changes in all measurements over the training programme were similar between the men and women (i.e. there were no sex×time interactions). For clarity, only comparisons between glutamine and placebo groups with the sexes combined are presented.

There were significant time main effects for all the strength measurements, peak torque, lean tissue mass, and urinary 3-methylhistidine levels ($P < 0.05$), with no differences between glutamine and placebo groups at any time. Glutamine and placebo groups increased 1 RM squat by 31% and 30%, respectively (Fig. 1). For bench press 1 RM, the glutamine group had a relative increase of 14%, while the placebo group increased by 13% (Fig. 2). For knee extension peak torque, the glutamine group increased by 6%, while the placebo group increased by 5% (Fig. 3). The glutamine group increased lean tissue mass by 2.0%, while the placebo group increased by 1.7% (Fig. 4). Urinary levels of 3-methylhistidine were increased by 41% and 56% for glutamine and placebo groups, respectively (Fig. 5).

There were no differences in average training volume between glutamine and placebo groups. The glutamine group had a mean (SEM) average volume of 2,367 (237.8) kg per training session while the placebo group had an average volume of 2,389 (283.9) kg per session. Dietary intake did not differ significantly between groups and did not differ significantly over the course of the training (Table 2).

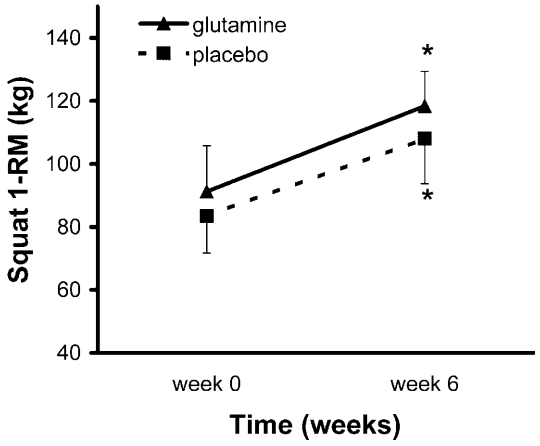


**Fig. 1** Mean one repetition maximum (1 RM) squat for glutamine ($n = 17$) and placebo ($n = 14$) groups at week 0 and week 6 of resistance training. Error bars are ±SEM. *$P < 0.05$ compared to week 0

## Discussion

To our knowledge, this is the first study to investigate the effect of oral glutamine supplementation during strength training. Glutamine supplementation has been shown to enhance glycogen resynthesis following endurance-type exercise (Varnier et al. 1995; Bowtell et al. 1999), decreases protein degradation when infused into animals (MacLennan et al. 1988) and increases protein synthesis when infused into animals (MacLennan et al. 1987) or humans (Hankard et al. 1996). Based on the fact that resistance training results in glycogen depletion (Tesch et al. 1986; Robergs et al. 1991; Roy and Tarnopolsky 1998) and elevated protein turnover (Biolo et al. 1995; MacDougall et al. 1995; Phillips et al. 1997), it has been speculated that adaptation to resistance training may be enhanced by glutamine supplementation (Antonio and Street 1999). We hypothesized that strength, torque and lean tissue mass would increase more in our glutamine group than in the placebo group, and that myofibrillar protein degradation would be

**Table 1** Mean (SEM) subject characteristics at baseline for glutamine and placebo groups. *1 RM* One repetition maximum

| | Mass (kg) | Lean tissue mass (kg) | Height (cm) | Squat 1 RM (kg) | Bench press 1 RM (kg) | Knee extension torque (N·m) |
|---|---|---|---|---|---|---|
| Glutamine (9 men, 8 women) | 72.7 (2.7) | 50.1 (2.9) | 175.7 (2.6) | 91.2 (14.6) | 56.1 (11.7) | 227.8 (23.6) |
| Placebo (8 men, 6 women) | 77.3 (4.1) | 49.4 (3.2) | 173.4 (2.4) | 83.5 (11.8) | 55.8 (16.1) | 208.3 (12.9) |

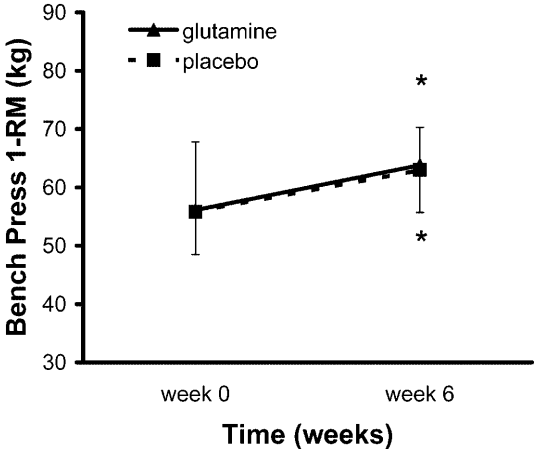


**Fig. 2** Mean one repetition maximum (1 RM) bench press for glutamine ($n=16$) and placebo ($n=14$) groups at week 0 and week 6 of resistance training. Error bars are ±SEM. *$P<0.05$ compared to week 0

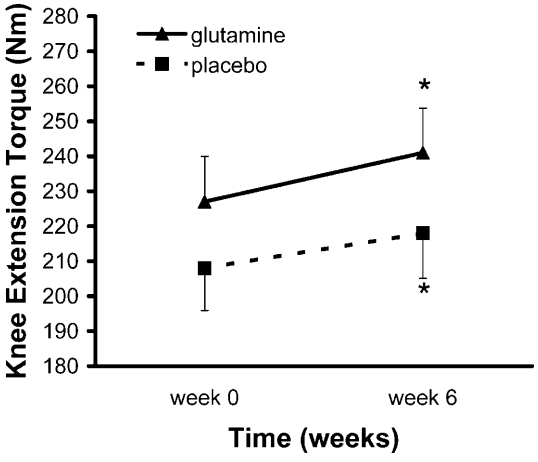


**Fig. 3** Mean knee extension torque for glutamine ($n=17$) and placebo ($n=14$) groups at week 0 and week 6 of resistance training. Error bars are ±SEM. *$P<0.05$ compared to week 0

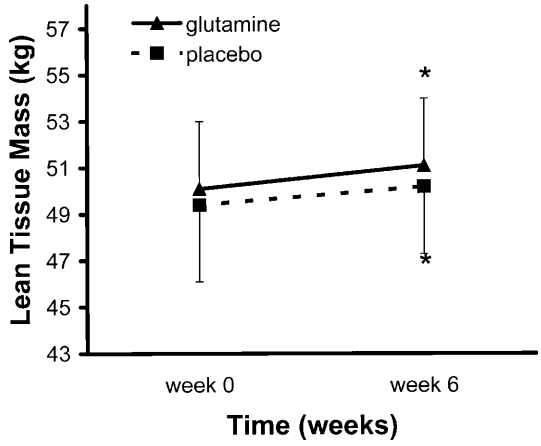


**Fig. 4** Mean lean tissue mass for glutamine ($n=17$) and placebo ($n=14$) groups at week 0 and week 6 of resistance training. Error bars are ±SEM. *$P<0.05$ compared to week 0

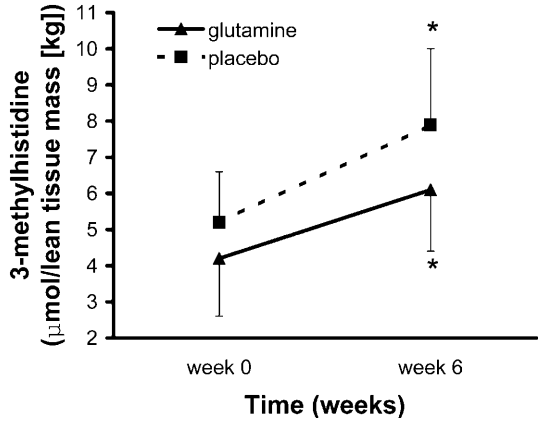


**Fig. 5** Mean urinary 3-methylhistidine levels for glutamine ($n=10$) and placebo ($n=11$) groups at week 0 and week 6 of resistance training. Error bars are ±SEM. *$P<0.05$ compared to week 0

attenuated in the glutamine group. However, the results of the present study showed no effect of glutamine on any of the dependent variables measured. This is significant given the fact that glutamine supplementation is currently very popular amongst individuals engaged in strength training (Kreider 1999).

The lack of effect of orally-administered glutamine on muscle function and lean tissue mass may be due to the consumption of glutamine by many other tissues before reaching the peripheral circulation and skeletal muscle. Glutamine is readily consumed by lymphocytes, macrophages, enterocytes and the mucosal epithelial cells of the small intestine where it is thought to serve as an energy source and nitrogen donor for nucleic acid synthesis (Ardawi 1988; Dechelotte et al. 1991). Other tissues that have rapidly replicating cells that use glutamine as an energy source include portions of the stomach, large intestine, spleen, and pancreas (for reviews see Windmueller 1982; Souba 1991). The kidneys use glutamine for the production of urinary ammonia during metabolic acidosis and the liver uses glutamine as a vehicle for the disposing of nitrogen, produced in catabolic reactions in peripheral tissues, by the formation of urea (Windmueller 1982; Souba 1991). It is estimated that approximately 50% of glutamine absorbed from the gut lumen is subsequently metabolized in the gut and liver (Darmaun et al. 1994). Nevertheless, at least a portion of the glutamine reaches the peripheral circulation and is therefore available to other tissues (Darmaun et al. 1994).

The failure of glutamine supplementation to enhance adaptations to resistance training may be due to the fact that resistance training is not *stressful* enough to benefit from glutamine supplementation. For example, glutamine has been shown to be beneficial for resynthesis of glycogen, by acting as a precursor for glycogen synthesis in liver and muscle following severe endurance type exercise, where muscle glycogen has been depleted by approximately 90% (Varnier et al. 1995; Bowtell et al. 1999). Glycogen is depleted during resistance exercise;

**Table 2** Mean (SEM) dietary variables for glutamine and placebo groups at week 0 (baseline) and at week 6 (post-training)

| | Placebo | | Glutamine | |
|---|---|---|---|---|
| | Week 0 | Week 6 | Week 0 | Week 6 |
| Kilocalories per day | 3,112 (195) | 2,910 (227) | 3,018 (195) | 3,023 (215) |
| % Carbohydrates | 54.6 (2.1) | 53.4 (1.5) | 51.9 (1.8) | 54.6 (1.7) |
| % Protein | 16.5 (1.2) | 18.9 (1.1) | 18.3 (1.1) | 21.5 (1.2) |
| % Fat | 28.9 (1.6) | 27.7 (13.2) | 29.8 (1.6) | 23.9 (2.3) |

however, a typical session of similar volume to that used in the present study results in a glycogen depletion of only 36% (Roy and Tarnopolsky 1998). This level of depletion may not be severe enough to benefit from glutamine supplementation. Whether glutamine supplementation enhances glycogen resynthesis following exercise is controversial, as recently it was demonstrated that oral glutamine supplementation had no effect on glycogen resynthesis following intense interval exercise that resulted in glycogen depletion (van Hall et al. 2000).

One of the proposed ergogenic effects of glutamine is to provide an energy source for cells involved in the immune system response. Exhausting endurance exercise has been shown to suppress the immune system. Whether glutamine supplementation improves the immune system response is controversial with some studies suggesting that glutamine supplementation improves immune system response and prevents infection or flu like symptoms following exercise (Castell et al. 1996, 1997; Castell and Newsholme 1998) and others demonstrating that glutamine supplementation has no effect on cells of the immune system (Rohde et al. 1998a, c). A number of authoritative reviews have made the argument that the evidence for an immune system enhancement with glutamine is not conclusive (Rohde et al. 1998b; Nieman and Pedersen 1999). Taking the findings from the positive studies into account, Antonio and Street (1999) proposed that glutamine may promote recovery between bouts of heavy resistance exercise and therefore promote muscle adaptation. However, there appears to be no evidence that heavy resistance exercise has a detrimental effect on the immune system (Rall et al. 1996; Flynn et al. 1999). Although these studies were of older, not younger, subjects, one could infer that resistance training may not place sufficient stress on the immune system to benefit from glutamine supplementation. This type of exercise may not be expected to induce even brief immunosuppression since a combination of intensity and long duration is needed for an effect on immune function (for review see Nieman and Pedersen 1999).

A third proposed ergogenic effect of glutamine is that it prevents protein degradation and therefore will enhance recovery following resistance training sessions (Antonio and Street 1999). This is largely based on the finding that glutamine infusion in rats prevents protein breakdown (MacLennan et al. 1988). Glutamine supplementation did not affect myofibrillar protein degradation as assessed by urinary 3-methylhistidine in the current study. There appeared to be a trend towards a greater myofibrillar breakdown in the placebo group (Fig. 5), but the difference was not significant. MacLennan et al. (1988) demonstrated that glutamine supplementation prevented a breakdown of soluble, but not myofibrillar protein in rats. Similarly, Hankard et al. (1996) failed to observe an alteration in rates of protein degradation, as assessed by the rate of leucine appearance in plasma, following glutamine infusion in human subjects.

One limitation of the present study may be the relatively short duration of training (6 weeks). We chose to use this programme because we have previously shown that other ergogenic aids (whey protein and creatine monohydrate) were effective for enhancing adaptations to an identical programme of the same duration (Burke et al. 2001). Thus, the findings with glutamine in the current study can be directly compared to the effectiveness of creatine monohydrate and whey protein for promoting adaptations during resistance training.

A second limitation may be the use of a mixture of male and female subjects. This most likely had minimal effect on our results however, as there were equal numbers of men and women in the glutamine and placebo groups and sex was not a factor in adaptations to the training programme. Men and women had similar relative increases in strength, torque and lean tissue mass. This is in agreement with other studies of short-term resistance training which have compared men and women (Cureton et al. 1988; Staron et al. 1994; O'Hagan et al. 1995; Abe et al. 2000).

The dose of glutamine used in the current study was rather large, approximating 45 g a day. It could be argued that excessive doses of any compound can have inhibitory effects, but this cannot be determined from the current study. There is concern for toxicity as some of the metabolic by-products of glutamine metabolism, specifically glutamate and ammonia, are potentially toxic in large amounts (Lowe et al. 1990; Souba 1991). None of our subjects reported negative side-effects while taking the glutamine supplement. Lowe et al. (1990) did not report increases in glutamate or ammonia with parenterally-administered glutamine at doses approximating 40 g a day in seven subjects and 57 g a day in two subjects. This may differ from orally-administered glutamine, as much of glutamine metabolism occurs in the gut (Windmueller 1982; Souba 1991); however, others have recently used higher oral doses than used in the current study and have not reported any negative

side-effects. Van Hall et al. (2000) orally administered approximately 66 g of glutamine a day and Rhode et al. (1998c) administered 0.9 g·kg body mass$^{-1}$, compared to our dose of 0.9 g·kg of lean tissue mass$^{-1}$ (approximately 45 g a day).

In conclusion, glutamine supplementation did not have a beneficial effect on the adaptations to strength training. While glutamine supplementation has been shown to have beneficial effects following endurance-type exercise, strength training may not be *stressful* enough to benefit from glutamine supplementation. Future research should look at the effect of glutamine supplementation during training protocols that provide a greater physiological stress, such as combined endurance and strength training. This type of programme has been shown to elicit a greater *stress response*, as measured by cortisol concentrations, than either type of training alone (Kraemer et al. 1995; Bell et al. 2000).

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