Glutamine: A Potentially Useful Supplement for Athletes

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Abstract/Résumé

The role of glutamine as a possible ergogenic aid has not been posited in the scientific literature. Although there is an abundance of clinical evidence supporting the need for exogenous glutamine in the maintenance of muscle protein mass and immune system function in critically ill patients, little work has been done that examines the potential utility of glutamine for athletes engaged in heavy exercise training. This brief review will describe a number of studies on the effects of glutamine supplementation on muscle protein mass, immune system function, and glucose regulation. Based on the available clinical evidence, we would speculate that glutamine has potential utility as a dietary supplement for athletes engaged in heavy exercise training.

Le rôle ergogène de la glutamine n’a pas été établi dans la littérature scientifique. Malgré la quantité importante d’observations cliniques appuyant la thèse en faveur d’un supplément de glutamine exogène pour le maintien de la masse protéique des muscles et le bon fonctionnement du système immunitaire chez des patients, il y a peu d’études sur l’utilité potentielle de la glutamine chez des athlètes à l’entraînement intense. Cette brève revue dresse le bilan d’études sur les effets d’un supplément de glutamine sur la masse protéique des muscles, la fonction du système immunitaire et la régulation du glucose. À la lumière des observations cliniques, il semble qu’un supplément de glutamine puisse aider les athlètes à l’entraînement intense.

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Introduction

Glutamine is the most abundant amino acid in plasma as well as skeletal muscle (Lacey and Wilmore, 1990) and accounts for more than 60% of the total intramuscular free amino acid pool (Rowbottom et al., 1996). Because skeletal muscle represents such a large mass of tissue, it is quantitatively the most important site of glutamine synthesis despite the fact that glutamine synthetase activity is relatively low per unit mass in skeletal muscle (Curthoys and Watford, 1995). However, other investigators have suggested that adipose tissue may represent a site of glutamine synthesis similar in magnitude to skeletal muscle (Frayn et al., 1991). In addition, the lungs, liver, and brain are other sites of glutamine synthesis.

Glutamine is utilized primarily as fuel by tissues such as the small intestine (i.e., enterocytes), immune system (neutrophils, thymocytes, lymphocytes, and macrophages), and hair follicles (Curi et al., 1997, Curthoys and Watford, 1995; Rowbottom et al., 1996). The gastrointestinal tract alone accounts for 40% of the total glutamine utilized by the body (Windmueller and Spaeth, 1974). In the liver, glutamine is used for glucose and urea synthesis whereas the brain utilizes glutamine as a precursor for neurotransmitter substances (Curthoys and Watford, 1995). The kidneys normally use very little glutamine except in times of metabolic acidosis (i.e., kidneys utilize glutamine to support renal ammoniagenesis) (Fine, 1982; Squires and Brosnan, 1983). In endotoxin-treated rats, it has been estimated that intramuscular stores of glutamine would be depleted in approximately 7 hours if de novo formation of glutamine did not occur in skeletal muscle (Austgen et al., 1992a; Rowbottom et al., 1996).

There is evidence that glutamine is important for the maintenance of skeletal muscle protein levels, immune system function, and glucose/glycogen metabolism. This has led to the reclassification of glutamine as a "conditionally essential" amino acid (Hall et al., 1996; Lacey and Wilmore, 1990). That is, under certain conditions of metabolic stress, the body’s need for glutamine exceeds its ability to synthesize glutamine endogenously; therefore, the provision of glutamine is important and has been shown to result in better nitrogen balance and preservation of skeletal muscle.

This review examines those functions of glutamine which could theoretically have an impact on athletic training and performance. For example, the role of glutamine in modulating the immune system as well as its antiproteolytic effect might have interesting implications for athletes engaged in intensive exercise training. The existing data on glutamine use by athletes is scant. However, an abundance of clinical data suggests there exists a theoretical basis for the use of glutamine as an ergogenic aid. This review will highlight our current understanding of glutamine and its effects on the immune response, regulation of glucose metabolism, and its anticitobolic action in skeletal muscle. This body of evidence has accumulated primarily within the last decade.

Facts About Glutamine

SKELETAL MUSCLE AND GLUTAMINE METABOLISM

Glutamine is synthesized from glutamate by the action of glutamine synthetase (Newsholme and Leech, 1983). Glutamate is formed from alpha-ketoglutarate, an intermediate of the Krebs cycle, and ammonia:
Glutamine  •  3

\[ \text{NH}_4^+ + \text{alpha-ketoglutarate} + \text{NADPH} + H^+ \xrightarrow{\text{glutamate dehydrogenase}} \text{glutamate} + \text{NADP}^+ + \text{H}_2\text{O} \]

\[ \text{ATP} + \text{NH}_4^+ + \text{glutamate} \xrightarrow{\text{glutamine synthetase}} \text{ADP} + \text{Pi} + \text{glutamine} + H^+ \]

Skeletal muscle, and possibly adipose tissue, is quantitatively the most important source of glutamine (Frayn et al., 1991). The ability of skeletal muscle to control the conversion of amino acids into alanine and glutamine represents part of a complex physiological mechanism whereby alanine is available for hepatic glucose synthesis and glutamine is available for the regulation of acid/base balance and as fuel for various cells.

Although the liver can oxidize most of the 20 amino acids, skeletal muscle has shown that it can only oxidize 6 amino acids, that is, the branched-chain amino acids, aspartate, asparagine, and glutamate (Wagenmakers, 1998). This is significant, not just for oxidation but also for the ability of skeletal muscle to convert these amino acids into glutamine and alanine. A small amount of glutamine is utilized for biosynthesis of purines, pyrimidines, glucosamine, and other amino acids. However, enterocytes, various cells of the immune system (e.g., thymocytes, neutrophils, lymphocytes, and macrophages) use glutamine as a primary fuel source. In this case, glutamine enters the Krebs cycle via its conversion to alpha-ketoglutarate where it is oxidized. In addition, glutamine serves as a gluconeogenic precursor in the liver and for ureagenesis while the kidney metabolizes glutamine into ammonium ions that are eventually excreted (Figure 1).

ENHANCED PROTEIN SYNTHESIS/DECREASED PROTEIN DEGRADATION

Because skeletal muscle accounts for most of the protein pool in the body, the regulation of protein metabolism in skeletal muscle is important for whole-body protein homeostasis. Glutamine serves an important role in the regulation of muscle protein levels. MacLennan et al. (1987) used an isolated perfused rat hindquarter model to examine the effect of glutamine on muscle metabolism. They found that increasing the concentration of glutamine significantly increased intracellular glutamine and protein synthesis in the absence of insulin. An in vitro study showed that glutamine augments protein synthesis in myotubes that are under heat-stressed conditions; however, there was no effect on myotubes under normal conditions (Zhou and Thompson, 1997).

In addition, glutamine has an antiproteolytic effect on the noncontractile protein component of rat skeletal muscle (MacLennan et al., 1988). In a human study comparing the effects of glutamine versus glycine, it was found that the enteral infusion of glutamine increased protein synthesis (Hankard et al., 1996). On the other hand, an isonitrogenous amount of glycine did not affect protein synthesis but only slightly decreased proteolysis.

Work by Haussinger et al. (1994) posits that the hydration state of cells is a critical factor that may influence metabolic processes within a cell. An increase in cellular volume or hydration status acts as an anabolic signal while a decrease in cellular volume acts as a catabolic signal (Haussinger et al., 1993; Waldegger et al., 1997). In a study using an isolated rat skeletal muscle preparation, Parry-Billings et al. (1991) found that changes in the osmolarity of the surrounding medium
Figure 1. Role of skeletal muscle in regulating glutamine metabolism. Branched-chain amino acids, glutamate, aspartate, and asparagine derived from muscle proteolysis contribute to the de novo formation of glutamine. Most of this glutamine is oxidized by the gastrointestinal tract and cells of the immune system. Glutamine can also transport ammonia to the kidneys for excretion and to the liver to be excreted as urea.

affected the rates of glutamine and alanine release from skeletal muscle. Moreover, glutamine may exert an anticatabolic effect by mediating increases in cellular volume (Vom Dahl and Haussinger, 1996).

It is known that the depletion of intramuscular glutamine is associated with increased muscle catabolism; therefore, it is important that these stores be maintained in order to maintain skeletal muscle size. For instance, glucocorticoids accelerate the release of muscle glutamine during times of illness (Muhlbacher et al., 1984). Falduto et al. (1992) showed that glucocorticoid administration for 4 days reduced plantaris and quadriceps muscle mass to 90% of control values. This was accompanied by a threefold increase in glutamine synthetase mRNA and enzyme activity in the deep quadriceps (fast-twitch red) muscle. Exercise training
resulted in a protective effect on skeletal muscle mass as well as a reduction in the effects of glucocorticoid treatment on glutamine synthetase mRNA and enzyme activity (Falduto et al., 1992).

The infusion of the dipeptide, alanyl-glutamine, further alleviates muscle atrophy and glutamine synthetase production in rats given hydrocortisone 21-acetate (Hickson et al., 1996). The mechanisms by which glutamine ameliorates glucocorticoid-induced muscle atrophy is not associated with changes in plasma levels of insulin-like growth factor I or insulin-like growth factor binding proteins (Hickson et al., 1997). However, at the molecular level, glutamine prevents the downregulation of myosin heavy-chain synthesis seen in glucocorticoid-induced muscle atrophy (Hickson et al., 1995).

**GLUCOSE REGULATION**

In addition to its role in protein metabolism, glutamine has an integral role in glucose regulation. In humans who fasted for 18 to 42 hours, the contribution of protein degradation to glutamine formation increased while de novo synthesis declined (Hankard et al., 1997). In normal postabsorptive humans, glutamine was infused at a rate similar to its appearance in plasma after a protein meal was ingested. It was found that glutamine infusion resulted in a threefold increase in plasma glutamine and a sevenfold increase in glucose production from glutamine (Perriello et al., 1997). Further, gluconeogenesis (from glutamine) occurred without changes in plasma insulin and glucagon levels, providing evidence that glutamine itself can regulate gluconeogenesis.

Nurjhan et al. (1995) examined the contribution of alanine and glutamine to glucose formation in postabsorptive normal human volunteers. These investigators found that even though carbon transfer to glucose from glutamine and alanine were equivalent, the amount of glucose carbon that came from protein derived glutamine was 100% greater than from alanine. Varnier et al. (1995) studied the effects of glutamine, alanine + glycine, and saline infusion on glycogen accumulation in subjects who cycled for 90 minutes. At 2 hours postexercise, glutamine infusion resulted in a twofold greater concentration of muscle glycogen than did either saline or alanine + glycine infusion. In postabsorptive humans, it is likely that glutamine is more important than alanine for glucose formation derived from proteolysis. Furthermore, glutamine carbon can be directed to glycogen accumulation in skeletal muscle that had previously been glycogen depleted.

Glutamine supplementation of a high fat diet in mice that were genetically predisposed to obesity and hyperglycemia resulted in a reduction of body weight as well as a decrease in hyperglycemia and hyperinsulinemia (Opara et al., 1996). The mechanism for glutamine-induced weight reduction is not clear; however, it may be related to the glutamine's ability to ameliorate insulin resistance induced by high fat consumption. The administration of glutamine to lipid based total parenteral nutrition can prevent glucose intolerance and insulin resistance (Ballard et al., 1996).

**GLUTAMINE AND THE IMMUNE SYSTEM**

Severe stressors such as burns, surgery, sepsis, prolonged exercise, and athletic overtraining cause a significant reduction in skeletal muscle and plasma glutamine
concentration (Newsholme, 1994; Newsholme and Calder, 1997). Glutamine serves as an important fuel source for lymphocytes and macrophages. It is thought that during times of stress or illness, glutamine metabolism is increased in order to promote rapid cell division, antibody production, and protein synthesis (Ardawi and Newsholme, 1985). Caldwell et al. (1989) showed that glutamine is needed for the process of wound healing. Without adequate glutamine, lymphocyte proliferation diminishes, as does the synthesis of interleukin-1 by macrophages and interleukin-2 by lymphocytes (Newsholme and Calder, 1997).

In infected early weaned pigs, glutamine supplementation maintained skeletal muscle concentrations of glutamine and improved lymphocyte function while an isonitrogenous diet containing nonessential amino acids minus glutamine had little effect (Yoo et al., 1997). In patients with multiple organ failure, parenteral administration of glutamine improved the 6-month survival of these patients versus an isonitrogenous, isocaloric control (Griffiths, 1997). Thus, part of the impairment seen in the immune system post-trauma may be due to inadequate production or inadequate dietary intake of glutamine (Wallace and Keast, 1992).

In the athletic population, an imbalance due to excess exercise and inadequate recovery has led to what is termed the overtrained state or overtraining syndrome (Rowbottom et al., 1996). The overtrained state is characterized by recurrent infections, fatigue, impaired immune function, and reduced exercise performance.

The precise role of glutamine in the immunosuppressed state of overtrained athletes is not yet completely understood. Keast et al. (1995) induced overtraining by having subjects exercise twice a day for 10 days, followed by a 6-day recovery period. They found that plasma glutamine concentrations decreased in 4 of 5 subjects by the 6th day of overtraining, with all of them experiencing a marked decline by the 11th day. Two of these subjects had still not returned to normal plasma levels of glutamine after the 6th day of recovery.

Castell et al. (1996) examined athletes who had consumed glutamine versus a placebo immediately after and 2 hours after a marathon or ultramarathon running race. During the follow-up period 7 days postexercise, the glutamine group had a greater percentage of individuals who reported no infections, 81%, versus 49% for the placebo group. Questionnaires were administered to middle-distance runners, marathon runners, ultramarathon runners, and elite male rowers to determine the frequency of infection. The latter 3 groups had the highest infection rates (Castell and Newsholme, 1997). In fact, plasma glutamine levels were found to decrease by 20% one hour after a marathon race. There was marked elevation of white blood cells immediately after exhaustive exercise, followed by a decrease in lymphocyte count. The administration of oral glutamine resulted in an increase in T-helper : T-suppressor cell ratio and further decreased the incidence of subsequent infections. Hack et al. (1997) suggested that glutamine deficiency might impair the immune response due to a decrease in the number of CD4+ T cells after an 8-week period of intense exercise training. However, Jensen et al. (1996) demonstrated that glutamine feeding in critically ill patients augmented the CD4/CD8 ratio.

Not all studies show an effect of glutamine supplementation after intense training. In runners who had completed a marathon, there was no reported effect of glutamine ingestion on lymphocyte distribution (Castell et al., 1997). HIV-positive individuals who cycled for 1 hour at 75% of maximal oxygen uptake had a lower phytohemagglutinin-stimulated proliferative response versus an HIV-negative
group (Rohde et al., 1995). Although the addition of glutamine increased the proliferative response and killer-cell activity of blood mononuclear cells in normal healthy subjects, no such effect was observed in the HIV-positive group (Rohde et al., 1995). Further, glutamine supplementation provided no additional benefit in immune function to exercise-trained rats, but it did to sedentary rats (Shewchuk et al., 1997a).

Differences in nutrient intake, psychological state, and stress levels (including mode, volume, and intensity of exercise) would necessarily have an impact on the effect of glutamine in humans. Nevertheless, there is substantial evidence that the maintenance of normal plasma glutamine levels is essential for normal functioning of lymphocytes and macrophages. A decrement in plasma glutamine levels likely contributes in part to the impairment of the immune system under various stressful conditions.

POSTSURGERY AND ILLNESS

In dogs that had undergone a laparotomy, the effects of a saline or an amino acid solution (with or without glutamine) on skeletal muscle nitrogen were ascertained before and 24 hours after surgery (Kapadia et al., 1985). Skeletal muscle nitrogen declined in the placebo treated animals as well as in those receiving only 2 grams per kilogram of an amino acid solution (with or without glutamine). However, both intracellular nitrogen and glutamine were maintained in animals receiving 4 g per kilogram of solution, regardless of whether glutamine was present. In this case, providing sufficient amino acid nutrition may preserve intramuscular glutamine levels and may be needed to preserve muscle protein. Roth et al. (1988) found that infusion of the dipeptide alanylglutamine reduced nitrogen release from the hindlimb of anesthetized postsurgical dogs.

In septic rats, the rate of glutamine production in skeletal muscle was found to be markedly elevated (Ardawi and Majzoub, 1991). Fang et al. (1995) found that protein synthesis was not affected by glutamine supplementation in septic rats (i.e., sepsis was induced by cecal ligation and puncture). On the other hand, the infusion of an alanyl-glutamine dipeptide via osmotic pumps increased protein synthesis in liver and skeletal muscle of rats that were infected with Escherichia coli; furthermore, it protected the morphology of the intestinal mucosa and increased the survival rate (Naka et al., 1996).

In rats that were implanted with the Morris Hepatoma 777, investigators examined the effects of L-glutamine provision (20 g/kg) and swim training (3 hrs/day) on tumor growth (Shewchuk et al., 1997b). After 14 days of glutamine treatment, the mean tumor weight of the glutamine treated rats was lower than for the untreated group. Exercise had no effect on tumor growth regardless of whether the animal received glutamine or not (Shewchuk et al., 1997b). Austgen et al. (1992b) found, however, that the provision of 20% of total TPN protein as glutamine had no effect on tumor growth in rats implanted with methylcholanthrene-induced fibrosarcoma.

Several studies involving human subjects suggest that glutamine administration has a significant protein-sparing effect. Hammarqvist et al. (1989) examined the role of glutamine as part of total parenteral nutrition in patients who had undergone elective abdominal surgery. Patients received a conventional amino acid
solution with or without glutamine. The glutamine group received 0.285 g per kilogram body weight per day. It was found that the addition of glutamine to total parenteral nutrition improved nitrogen balance and ameliorated the decline in protein synthesis. In patients who had undergone cholecystectomy, the addition of an alanyl-glutamine dipeptide improved nitrogen balance and prevented a decline in muscle protein synthesis (Hammarqvist et al., 1990).

Pettersson et al. (1994a) found that the administration of glutamine (20 g per day for 3 days) after cholecystectomy helped counteract the decline in muscle protein synthesis that occurred in the unsupplemented group. Similar work (Pettersson et al., 1994b) examined the effect of long-term parenteral administration of glutamine, in the form of glycyl-glutamine on free amino acid levels in skeletal muscle of patients who had undergone abdominal surgery. They found that glycyl-glutamine preserved free glutamine levels in skeletal muscle after surgery, but when treatment was discontinued, skeletal muscle levels of free glutamine dropped despite the maintenance of normal enteral nutrition. However, in patients who had undergone heart surgery, the administration of large doses of glutamine did not prevent endotoxemia during or after surgery (Suojaranta-Ylinen et al., 1997).

SAFETY OF GLUTAMINE USE IN HUMANS

Glutamine is efficiently absorbed in the human jejunum in vivo (Dechelotte et al., 1991). Ziegler et al. (1990) conducted an extensive examination concerning the effects of both short-term and long-term administration of glutamine on human subjects. Acute oral ingestion of glutamine at doses of 0.1 and 0.3 g per kilogram body weight showed no evidence of clinical toxicity. As a component of total parenteral nutrition (glutamine dose of 0.285 and 0.570 g per kg body weight per day), glutamine had no harmful effects after 5 days of administration in normal subjects. The safety of glutamine use was confirmed in patients receiving glutamine for several weeks (Ziegler et al., 1990).

Glutamine infusion as a dipeptide (glycyl-glutamine) was examined in polytrauma patients. Using doses equal to 14, 21, and 28 g of glutamine (calculated for a 70-kg individual) per day, Weingartmann et al. (1996) found no ill effects of glycyl-glutamine. Although glutamine is absorbed efficiently in the human jejunum in vivo and is demonstrably safe, it should be noted that the doses used to elicit a positive effect on nitrogen balance are quite large (~0.2–0.6 g per kg body weight per day) (Weingartmann et al., 1996). This would be comparable to a 70-kg individual consuming 14 to 42 g of glutamine per day.

THEORETICAL BASIS FOR GLUTAMINE USE BY ATHLETES

Although there is a paucity of data on the use of glutamine by athletes, based on the available data it would seem plausible that glutamine supplementation may exert a beneficial effect for individuals engaged in chronic and intense exercise training. It is known that muscle glutamine levels fall in a dose-dependent manner to the degree of stress (Lacey and Wilmore, 1990). Further, the amount of glutamine released by skeletal muscle under such stressful situations is greater than the amount found in the intracellular pool and incorporated into proteins.

The provision of glutamine after stressful events (e.g., surgery, sepsis) maintains intramuscular glutamine concentration. Additionally, glutamine may improve
Theoretical Basis for Glutamine Supplementation in Exercising Individuals

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<tr>
<th>Reported effects</th>
<th>Hypothetical reason for glutamine supplementation by athletes</th>
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<tr>
<td>Immune system: important fuel source for immune cells; decrease incidence of infection.</td>
<td>⇒ May prevent or lessen the severity of illness or infection after an intense bout of exercise, thus enabling the athlete to resume intense training more quickly.</td>
</tr>
<tr>
<td>Skeletal muscle: maintenance of muscle protein levels during times of critical illness; counteract proteolytic effect of glucocorticoids; promote cell volumization.</td>
<td>⇒ May have an anti-proteolytic effect in individuals undergoing intense exercise training; in athletes who may have elevated glucocorticoids due to overtraining or medicinal steroid use (e.g., prednisone), glutamine ingestion may offset part of the catabolic effects of these hormones; the provision of glutamine might promote increases in cell volume which itself is an anabolic signal.</td>
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<tr>
<td>Glucose regulation: precursor for glucose/glycogen formation; improve insulin sensitivity.</td>
<td>⇒ May provide additional substrate for gluconeogenesis and glycogenesis; may offset negative effects of excess fat consumption.</td>
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<tr>
<td>Fuel for cells: gastrointestinal tract is the primary site of glutamine utilization; other organs that use glutamine include liver, kidney, immune cells.</td>
<td>⇒ The provision of glutamine to fuel other organs could spare the potential loss of glutamine due to inadequate dietary intake, thus sparing muscle protein.</td>
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the hydration status of skeletal muscle, resulting in an increase in cellular volume. The increase in cell volume could itself be an anabolic signal for the muscle cell (Haussinger et al., 1994). Because intracellular glutamine concentrations decline in a dose-dependent manner (i.e., the greater the stress, the greater the decline), one could posit that chronic exercise training would increase the requirements for glutamine such that exogenous self-administration may be necessary for top performance.

Although the primary route of glutamine administration in clinical situations is parenteral, glutamine is absorbed efficiently by the human gastrointestinal tract. Glutamine-enriched enteral and parenteral feeding results in similar amino acid profiles when given in identical doses (Fish et al., 1997). Thus, oral supplementation would be an effective method of delivering exogenous glutamine. Further, the doses required by athletes would be probably be less than those given to postsurgical patients.
With regard to overtraining, glutamine supplementation could possibly benefit the athletic individual. A decrease in the testosterone: cortisol ratio is one indicator of an overtrained state (Hoogeveen and Zonderland, 1996). It reflects a shift toward a catabolic state. Further, it is known that the administration of glucocorticoids accelerates the release of intramuscular glutamine and that the subsequent provision of glutamine alleviates the loss of muscle glutamine and protein. Perhaps if the overtrained athlete supplemented with glutamine, it might prevent the loss of protein due to elevated cortisol levels. The additional glutamine could help maintain normal immune system function, which in the overtrained athlete may be depressed.

The role of glutamine in the regulation of glucose is intriguing. Glutamine can provide substrate for glycogenesis and gluconeogenesis. It is debatable whether this is better than providing carbohydrate as substrate in exercising individuals. However, its role in lessening insulin resistance secondary to high fat consumption might be important in the prevention of excess fat gain.

**Summary**

The use of glutamine as a dietary supplement may have possible ergogenic benefits in athletes (see chart). The protein-sparing effect of glutamine would certainly be important for athletes engaged in strength-power sports, which call for a large amount of skeletal muscle mass. In all athletes, glutamine could serve to ameliorate the effects of overtraining on the immune system.

The provision of glutamine could also serve to augment muscle glycogen accumulation. Because glutamine is such an important fuel for the gastrointestinal tract and cells of the immune system, one could theorize that self-administration of dietary glutamine could in essence spare muscle protein while providing fuel for other cells and tissues. Otherwise, one could speculate that in athletes engaged in intense exercise training, the need for glutamine utilization might exceed the amount released by various organs/tissues, especially skeletal muscle and adipose tissue, to maintain normal plasma levels. As a consequence, the catabolism of muscle protein might occur. Certainly, for many athletes the preservation of muscle protein mass is a critical factor that could have an impact on performance.

Using doses lower than those provided to postsurgical patients, we would speculate that glutamine supplementation could prevent the loss of lean body mass which might result from overtraining and perhaps promote gains in lean body mass in strength-power athletes. Furthermore, glutamine supplementation could have a beneficial effect on the immune system and thus decrease the incidence of infection or illness secondary to overtraining.

Whether glutamine could be as effective as carbohydrate in the accumulation of muscle glycogen after prolonged exercise remains to be examined. Nonetheless, there is prolific evidence supporting the classification of glutamine as a conditionally essential amino acid in patients who are critically ill. Furthermore, we hypothesize that glutamine might also be a conditionally essential amino acid for athletes engaged in intense exercise training. Future work may determine whether the benefits of exogenous glutamine supplementation shown in clinical situations can also be duplicated in the athletic population.
References


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