2011 International Conference on Bioscience, Biochemistry and Bioinformatics
IPCBEE vol.5 (2011) © (2011) IACSIT Press, Singapore

The Effects of Supplementary L-Arginine Dietary on Serum Nitric Oxide Concentration in the Male Bodybuilders

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***Abstract*—Aim: Arginine has been classified as a 'semi-essential' or 'conditionally essential' amino acid. This characterization alludes to the fact that arginine has to be extracted from the diet as a supplement to the endogenous synthesis in growing mammals and in adult animals or humans during disease or trauma; however, arginine can be synthezised in sufficient quantity in the healthy adult. The aim of this study was to investigate the effect of long term arginine supplementation on Nitric oxide in intermittent anaerobic exercise and the underlying mechanism in well-trained male bodybuilders.**

Method: 30 elite male bodybuilder athletes recruited from The clubs of bodybuilding of Tehran participated in this Study. The subjects were 21.5±0.3 years old. The height were 1.71±0.06 m. The body weight in arginine (ARG) and control (CON) trials were 69.4±3.8 and 71.3±1.0 kg, respectively. This study used a randomized cross-over, placebo controlled design. Each subject was randomly assigned to ARG or CON trial separated.ARG trial consumed 12 g/day arginine tablets for 6 weeks.

Result: No significant difference was found between the 2 trials at any time point. In both trials, NOx concentration was significantly higher during and 6 min after exercise comparing to the basal concentration. This study suggested that the long-term supplementation of arginine had no effect on plasma NO in intermittent anaerobic tests in well-trained male athletes. The use of arginine supplementation to increase NO production, reduce metabolites accumulation and improve exercise performance in athletes should be critically reevaluated even though it may have beneficial effect in certain patients and general population.

Keywords-L-Arginine dietary, serum nitric oxide, male bodybuilders

I. INTRODUCTION

Arginine has been classified as a 'semi-essential' or 'conditionally essential' amino acid. This characterization alludes to the fact that arginine has to be extracted from the diet (i.e. is an 'essential' amino acid) as a supplement to the endogenous synthesis in growing mammals and in adult animals or humans during disease or trauma; however, arginine can be synthezised in sufficient quantity in the healthy adult (Rose, 1937;Barbul, 1986).Nevertheless, even in the adult mammalnot every tissue expresses all enzymes necessary for the de novo synthesis of arginine and also catabolism of the amino acid is highly compartmented. Therefore, a complex inter-organ trafficking of the amino acids arginine and citrulline has to secure the balance between arginine production and arginine consumption, and intracellular metabolism as well as membrane transport are important determinants of the roles that arginineplays in the normal and pathophysiology of the body. The prominent feature of arginine metabolism is a complex differential expression of relevant enzymes when one looks at the major mammalian organs.The central pivot in arginine metabolism is the urea cycle. This first biochemical cycle to be described (Krebs and Henseleit, 1932) is the body's route of disposal of surplus nitrogen and thus, outside the nervous system, provides the means of detoxification of neurotoxic ammonia (Withers, 1998). It is the input of ornithine, the ability to convert ornithine into citrulline, and the catabolism of arginine mainly by arginase and NO synthase that determine the role of an organ or cell as arginine producer or consumer.

NO also plays a role in exercise-induced vasodilation in patients and healthy subjects(1). The impairment of NO production and the resulting endothelial dysfunction are the major factors that limit exercise capacity in patients with various cardiopulmonary conditions. As the result, argininesupplementation has been shown to improve exercise capacity in patients with hypercholesterolemia (2). chronic heart failure (3), pulmonary hypertension (4) and stable angina pectoris (5). De novo synthesis of arginine depends on the presence of ornithine carbamoyltransferase (OCT; EC 2.1.3.3) which, together with carbamoylphosphate synthetase I, is located in the mitochondrial matrix. Expression of both enzymes in animals is restricted to the periportal hepatocytes in liver, the epithelial cells in the mucosa of the small intestine and, to a minor extent, the colonocytes of the large intestine (Raijman, 1974; Knecht et al., 1979; Morris, 1992), and is notably lacking in cells of the nervous system. Consequently, only the former tissues are able to utilize ornithine for the generation of L-citrulline. Due to a further cellular restriction of biosynthetic enzymes in the adult, substantial production of ornithine from diet- and blood derived glutamine or enteral praline occurs exclusively in the gut which releases citrulline into the circulation (Windmueller, 1982). Most of this citrulline is taken up into the kidney and utilized by the cells of the proximal tubulus for the synthesis of arginine which, in turn, is released into the blood for the benefit of other organs

(Windmueller and Spaeth, 1981; Dhanakoti et al. 1992: Rabier and Kamoun, 1995).

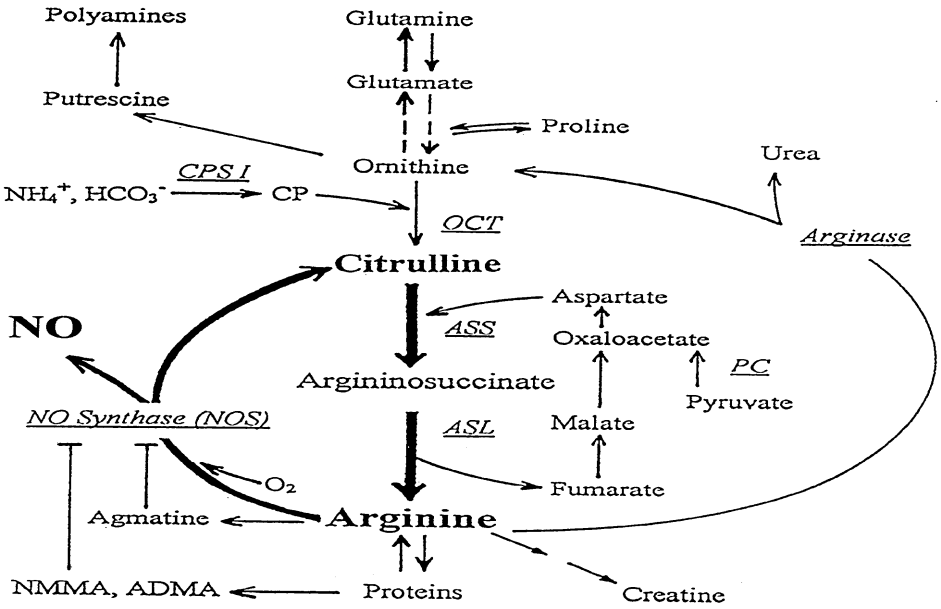

Figure 1. Arginine metabolism in mammalian cells with emphasis on its interrelationship with the generation of NO and a citrulline–NO cycle.

II. Methods And Materials

30 elite male bodybuilder athletes recruited from The clubs of bodybuilding of Tehran participated in this Study.The subjects with similar body weight were selected to avoid potential difference in exercise performance and supplementation dosage. The potential subjects with known cardiovascular disease risks, with musculoskeletal injuries, or who have taken any protein supplement in the previous 3 months were excluded. All athletes have undergone regular bodybuilding training for at least 3 years. The subjects were 21.5±0.3 years old. The height were 1.71±0.06 m. The body weight in arginine (ARG) and control (CON) trials were 69.4±3.8 and 71.3±1.0 kg, respectively. The body weight was not significantly different between the 2 trials. The subjects were asked to maintain their regular training schedule and diet habits during the study period. The subjects were asked to consume the same food on the day prior to each trial. All subjects gave their written informed consent after the experimental procedure, and possible risks were explained.

A. Experimental design

This study used a randomized cross-over, placebo controlled design. Each subject was randomly assigned to ARG or CON trial separated.ARG trial consumed 12 g/day arginine tablets for 6 weeks. Blood samples were obtained from a cannula in the antecubital vein by licensed personnel. Blood samples were collected before supplementation, after 3 weeks exercise and after 6 weeks.

B. Statistical analysis

All values were expressed as means±S.E.M. Changes in plasma metabolites concentrations were analyzed by a two-way analysis of variance with repeated measures. The difference between baseline and other time points in plasma metabolites were analyzed by post hoc Tukey's test. The biochemical and intermittent exercise performance parameters at each time point were analyzed by Student's paired t test. The difference between area under the curve of the two trials was analyzed by Student's paired t test. The analysis was performed with SPSS. A P value less than .05 was considered statistically significant.

III. Result

Plasma NOx concentrations at each time point in both trials are depicted in Fig. 2. No significant difference was found between the 2 trials at any time point. In both trials, NOx concentration was significantly higher during and 6 min after exercise comparing to the basal concentration. The increase in NOx concentration during exercise in both trials was parallel to the increase in plasma citrulline concentrations (Fig. 3). There was no significant difference in citrulline concentration between the 2 trials at any sampling time poin.

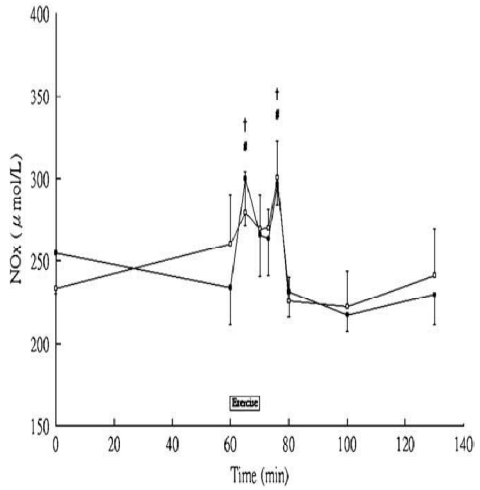

Figure 2. Plasma NOx concentrations

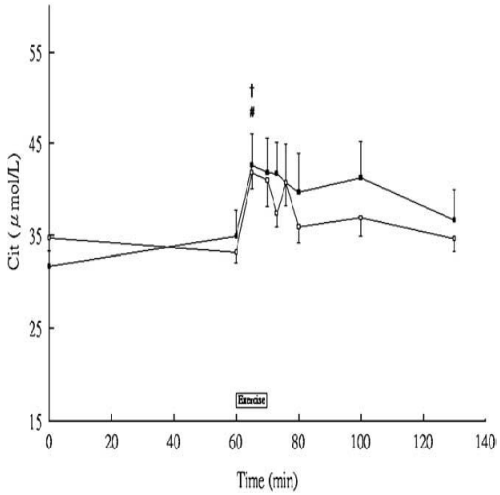

Figure 3. Plasma citrulline concentrations

IV. DISCUSSION:

The results of this study suggested that long-term arginine supplementation had no effect on NO production, in intermittent anaerobic exercise in well-trained male bodybiulding athletes. Both ARG and CON trials showed exercise-induced NO production, as NOx and citrulline concentrations were significantly elevated during exercise. The mechanism for the increase in NOx at 6 min after exercise was not clear, as plasma citrulline concentration did not change. However arginine supplementation had no effect on exercise-induced NO production in our well-trained subjects. This result was in contrast to Kanaya(1999) studies that suggested arginine supplementation could improve exercise-induced NO production and vasodilation in subjects with various cardiovascular diseases (6). In addition, it has been reported that regular exercise training and arginine supplementation may have additive effects on improving endothelium-dependent vasodilation in chronic heart failure patients (7). It is possible that our athletes already had higher basal concentration of NO than general population and these patients (8). Regular exercise training has been shown to increase basal NO production (9) by stimulating endothelial NO synthase expression and phosphorylation (10). Therefore, arginine supplementation did not provide any additional effect on NO production in our subjects. This study suggested that the long-term supplementation of arginine had no effect on plasma NO in intermittent anaerobic tests in well-trained male athletes. The use of arginine supplementation to increase NO production, reduce metabolites accumulation and improve exercise performance in athletes should be critically reevaluated even though it may have beneficial effect in certain patients and general population. Further investigations with higher dosages, extended supplementation periods or in combination with other compounds are warranted.

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