Effects of oral L-arginine supplementation on vasodilation and $\dot{V}O_2\text{max}$ in male soccer players

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Summary

Study aim: To determine the effects of oral L-arginine supplementation on vasodilation, blood flow and maximum oxygen uptake ($\dot{V}O_2\text{max}$) in soccer players.

Material and methods: A group of 24 healthy male soccer players aged 20 – 26 years volunteered to participate in the study. The subjects were randomly assigned into 3 groups: experimental, placebo and control. Experimental group was given L-arginine (6 g orally) and the placebo group – starch (6 g orally) daily for a week; subjects from the control group remained untreated. Before the test and at the end of the week blood samples were collected, and systemic blood pressures, blood flows and maximal oxygen uptake ($\dot{V}O_2\text{max}$, by Queens College step test) were recorded. Blood samples were assayed for HDL, LDL, triglyceride and urea concentrations. The study was conducted in a randomised, single-blinded, placebo-controlled fashion consisting of 7-day treatment periods.

Results: Oral supplementation of L-arginine significantly ($p<0.01$) decreased blood pressure indices and increased $\dot{V}O_2\text{max}$ ($p<0.01$), blood flow ($p<0.05$), femoral artery diameter ($p<0.05$) and urea levels ($p<0.05$). There was no change in blood lipid levels ($p<0.05$). No significant changes were noted in the placebo and control groups.

Conclusions: Oral supplementation of L-arginine may have beneficial effect on vasodilation and $\dot{V}O_2\text{max}$, therefore may increase the exercise capacity of soccer players.

Key words: L-Arginine – Maximal oxygen uptake – Blood flow – Vessel diameter

Introduction

Arginine is classified as a conditionally essential amino acid. Although it has numerous important physiological functions, L-arginine supplements have been used for two main reasons: to increase growth hormone secretion and to augment nitric oxide (NO) synthesis. L-arginine is the biologic precursor of NO, an endogenous messenger molecule involved in a variety of endothelium-dependent physiological effects in the cardiovascular system [14,24]. Many of the clinical effects of L-arginine are thought to be mediated by its endothelium-derived relaxing factor, NO.

A sizable body of research has explored the biological roles and properties of NO which appears to be of critical importance in the maintenance of normal blood pressure, myocardial function, inflammatory response and protection against oxidative damage [2]. Because NO is synthesised from the amino acid L-arginine by NO synthase [7], supplementation of L-arginine may have beneficial effects on the exercise capacity of athletes. In fact, oral supplementation of L-arginine improves endothelium-dependent vasodilation, haemodynamics and the exercise capacity in pulmonary hypertensive patients [8]. Most studies showed that oral administration of L-arginine improved vasodilation, blood pressure, blood flow, and exercise capacity of hypertensive and pre-eclampsia patients [15,19,26,27,29]. However, no reports were found in the available literature on the effects of oral administration of L-arginine in athletes and on sport performance. Thus, the purpose of this study was to investigate the effects of short-term oral supplementation of L-arginine on vasodilation, $\dot{V}O_2\text{max}$, haemodynamics and biochemical factors in trained male soccer players.

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Material and Methods

Subjects: Twenty-four university male students, members of a soccer team, volunteered to participate in the study. They trained regularly, three times a week. Their age was 23.0 ± 2.1 (20 – 26) years, body mass 73.2 ± 7.9 (60 – 88) kg, and body height 175.1 ± 5.2 (166 – 185) cm. All subjects submitted their written consents to participate and the study was approved by the local Committee of Ethics.

Experimental protocol: The study, lasting 7 days, was conducted in a randomised, single-blinded, placebo-controlled fashion. The examinations were conducted in an air-conditioned hall with an ambient temperature of 20 – 21°C. The subjects were requested to abstain from alcohol, caffeine, cigarettes and maximal exercise on the day preceding the onset of the study and to observe a nitrate-reducing diet.

Upon reporting at the laboratory, the subjects rested for at least 10 min and then the initial measurements of the systemic arterial blood pressures and heart rate were performed and followed by blood sampling from the ante-cubital vein for HDL, LDL, urea and triglyceride assays. Next, the femoral artery blood flow and vessel diameter were measured ultrasonographically. Finally, aerobic submaximal exercise tests were performed according to the Queens College Step test protocol [22]. After these procedures have been completed, the subjects were randomly assigned into 3 groups, 8 subjects each: experimental, receiving L-arginine, placebo and control. The experimental group was given orally 6 g of L-arginine daily (2 capsules, 1 g each, 3 times a day for 7 days), and the same protocol applied to the placebo group which received starch instead of L-arginine. The control group was not given any supplementation. At the end of the study the tests were repeated. All the measurements were taken by the same investigators to reduce operator-induced variability and to increase the reliability of the results.

Measurements: The systolic (SBP) and diastolic (DBP) blood pressures were measured on the brachial artery by the standard sphygmomanometric method using an automatic device (DBW128, Oregon Scientific, USA). Mean arterial blood pressure (MAP) was calculated from the formula: MAP = DBP + (SBP-DBP) / 3. Femoral artery diameter (D) was measured with B mode ultrasound images at rest using a standard 7.5 MHz linear array transducer and Logic-400 system (Logic-400, GE, USA). Blood flow (peak systolic velocity; PSV and end diastolic velocity; EDV) in the femoral artery was determined by ultrasonography-combining pulsed Doppler beams using a pulsed-wave Doppler signal at a 59º angle to the vessel, 4 cm below the bifurcation.

Biochemical assays were performed using an automated analyser (C-8000, Abbot, USA). The submaximal Queens College Step Test was used to indirectly estimate maximal oxygen uptake (VO₂max), the error amounting to about 10% [22].

Statistical analysis: ‘Post’-‘Pre’ differences in blood flows, blood pressures, pulse rate, biochemical data, vessel diameter and exercise performances within the three groups (L-arginine, placebo and control) were assessed using the Wilcoxon’s signed rank test and by the Kruskal-Wallis one-way ANOVA followed by post-hoc Mann-Whitney’s rank sum test. The level of p≤0.05 was considered significant. SPSS® software was used in data processing.

Results

The results of the study are presented in Tables 1 – 3. No significant between-group differences were found for any variable at the beginning of the study (‘Pre’). However, L-arginine administration for a week resulted in significant (p<0.01) decreases in SBP, DBP and MAP. No significant changes were observed in the resting heart rate (Table 1).

Table 1. Mean (±SD) pre- and post-study values of haemodynamic variables in male soccer players

<table>
<thead>
<tr>
<th>Variable</th>
<th>L-Arginine</th>
<th>Placebo</th>
<th>Control</th>
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<tbody>
<tr>
<td>SBP (mm Hg)</td>
<td>Pre 123 ± 14.1</td>
<td>118 ± 9.5</td>
<td>120 ± 15.6</td>
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<tr>
<td></td>
<td>Post 109 ± 10.1**</td>
<td>118 ± 9.0</td>
<td>118 ± 15.5</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>Pre 68 ± 9.9</td>
<td>63 ± 11.5</td>
<td>71 ± 11.8</td>
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<tr>
<td></td>
<td>Post 61 ± 8.2**</td>
<td>61 ± 12.7</td>
<td>69 ± 13.5</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>Pre 86 ± 9.1</td>
<td>81 ± 9.3</td>
<td>87 ± 10.9</td>
</tr>
<tr>
<td></td>
<td>Post 77 ± 6.7**</td>
<td>80 ± 9.5</td>
<td>85 ± 12.3</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>Pre 69 ± 12.6</td>
<td>65 ± 9.3</td>
<td>67 ± 11.5</td>
</tr>
<tr>
<td></td>
<td>Post 69 ± 12.4</td>
<td>66 ± 7.9</td>
<td>66 ± 11.8</td>
</tr>
<tr>
<td>D (mm)</td>
<td>Pre 9.2 ± 0.2</td>
<td>9.6 ± 0.6</td>
<td>9.6 ± 1.1</td>
</tr>
<tr>
<td></td>
<td>Post 9.5 ± 0.3*</td>
<td>9.6 ± 0.8</td>
<td>9.1 ± 0.6</td>
</tr>
<tr>
<td>PSV (mL/min)</td>
<td>Pre 51.0 ± 4.9</td>
<td>65.1 ± 8.7</td>
<td>65.4 ± 13.4</td>
</tr>
<tr>
<td></td>
<td>Post 56.4 ± 5.9*</td>
<td>58.7 ± 9.1</td>
<td>56.6 ± 13.7</td>
</tr>
<tr>
<td>EDV (mL/min)</td>
<td>Pre 7.4 ± 2.0</td>
<td>9.7 ± 5.3</td>
<td>8.6 ± 2.2</td>
</tr>
<tr>
<td></td>
<td>Post 12.6 ± 2.2**</td>
<td>10.5 ± 4.3</td>
<td>10.1 ± 3.2</td>
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</tbody>
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Significantly different from the ‘Pre’ value: * p<0.05; ** p<0.01

Lipid profiles did not change significantly after L-arginine and placebo supplementation (Table 2). There was, however, a small but significant (p<0.05) rise in blood urea in the experimental group.
proved endothelium-dependent vasodilation of the bra-

short-term oral administration of L-arginine also im-

proved vasodilation, blood pressure, blood flow and ex-

ercise capacity of healthy subjects [5,6,9,10,12,13,16,18,21].

Authors suggested that supplementation of arginine im-

proved endothelial function in experimental animals and

oral administration of L-arginine also improved endo-

thelial function in the femoral artery of athletes, reduced arte-

rial blood pressure and increased the femoral artery dia-

meter and after 6 months. Coronary blood flow in the L-

arginine group increased compared with the placebo group. These studies support our findings that short-term oral L-arginine supplementation improved endothelial function in the femoral artery of athletes, reduced arterial blood pressure and increased the femoral artery diameter, peak systolic velocity, end-diastolic velocity and VO2max. In contrast, other authors [1,9] reported no significant effect of L-arginine on vasodilation in healthy subjects.

L-arginine is a precursor of endothelium-derived relaxing factor – nitric oxide (EDRF/NO), known to decrease blood pressure in healthy humans and to play an important role in the regulation of blood pressure and systemic haemodynamics [16] and this might explain our findings. EDRF/NO also stimulates the guanylate cyclase and increases cGMP content in vascular smooth muscle cells, resulting in relaxation of the vascular tone [7]. EDRF/NO levels were not, however, measured in the present study and investigating into the role of the endothelial function of L-arginine in healthy athletes would be desirable.

In addition to being a precursor for NO production and protein synthesis, L-arginine is known to increase insulin secretion and this may decrease blood pressure by reducing hyperglycemia [17]. Short-term elevation of plasma glucose levels in normal subjects produced significant increases in systolic and diastolic blood pressures, heart rate, and plasma catecholamine levels and.
decreased blood flow in lower extremities, thus suggesting vasocstriction [11,17]. Therefore, the L-arginine-induced improvement in blood flow reported by us may not have been related to nitric oxide synthesis. For example, L-arginine may increase insulin release from pancreatic islet cells. A rise in serum insulin levels might potentially increase blood flow by decreasing blood glucose level which, in turn, produces vasodilation and this calls, again, for adequate studies.

Another effect attributed to L-arginine supplementation was an increase in VO2max; this might have been mediated by an exercise-induced increase in cardiac output via decreasing the ventricular afterload. It was demonstrated that short-term administration of L-arginine improved endothelial dysfunction related to a reduced exercise capacity in patients with congestive heart failure [26]. It is thus possible that the L-arginine-induced increase in VO2max may be partly attributable to an improvement in endothelium-dependent peripheral vasodilation. Another study [8] demonstrated that supplemental oral arginine improved exercise capacity in patients with stable angina pectoris. The increased VO2max following arginine intake suggests that the mismatch between the myocardial oxygen supply and demand had been altered in favour of vasodilating effects that facilitate oxygen supply and reduce its demand. However, further studies should be conducted involving body mass monitoring to make the VO2max changes more reliable.

In conclusion, oral supplementation of L-arginine produced small but significant increase in VO2max, and enhanced haemodynamics and endothelium functions in soccer players. This may have important implications for both athletes as well as patients, even though the mechanisms are not clear. Certainly, more studies are needed to understand the mechanisms of L-arginine function in healthy subjects.

References


