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Effect of Glutamate Administration on the Performance of Long-distance Runners and Cyclists

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=Abstract=**The accumulation of lactate in the muscle tissues after strenuous exercise induces a phenomenon of fatigue with a lowering of performance capability. The endeavor to overcome the fatigue via supplying high energy sources faces the contradictory reality that such a strategy might cause a higher accumulation of lactate in the tissues. As for the fate of lactate in the anerobic state of exercising muscle tissues, the transamination of pyruvate to alanine would eventually lower the content of lactate in addition to the well-known Cori cycle (Park *et al.*, 1989). Moreover, since the enzymes of GPT and LDH are of near-equilibrium properties, the reactions are mostly dependent on the concentration of substrates. Therefore, in the present experiment, we tried to determine the effect of amino acid administration, especially of glutamate, on the exercise performance in long-distance runners and high school cyclists to see whether the lactate level could be lowered. The results can be summarized as follows: (1) the lactate clearance in the blood was accelerated in the glutamate-administered group; (2) the blood level of lactate during strenuous exercise was lowered in the glutamate-treated group; (3) the performance of exercise was improved partially in the glutamate-treated group. From these results, it can be suggested that glutamate administration before exercise might reduce lactate accumulation, leading to improvement of exercise performance.**

Key Words: *Glutamate, Performance, Lactate, Fatigue*

INTRODUCTION

In the hot field of sports competition, the development of a strategy to improve the performance capacity of the athletes would have a profound impact. The essential problem in performance improvement is to overcome fatigue, inevitably resulted from the strenuous exercise. It is generally accepted that muscle fatigue can be caused by the accumulation of lactate in the tissues, leading to metabolic acidosis and consequently to a cascade of biological deterioration. The accumulation of lactate can be accelerated in the strenuous exercise over anerobic threshold (Wasserman *et al.*, 1973). The accumulated lactate can lower muscle pH (Hermansen & Osnes 1972), which causes the decrease in isotonic muscle contraction (Fitts & Holloszy, 1976, Donaldson *et al.*, 1978), in myosin ATPase activity (Schädler 1967) and in the content of muscle creatine phos-

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phate (Dawson *et al.*, 1978; Sahlin *et al.*, 1981) as well as the increase in ADP content, and calcium binding to proteins in sarcoplasmic reticulum (Nakamura & Schwartz, 1972). These multiple reactions induced by metabolic acidosis through lactate accumulation can lead to the fatigue phenomenon, interrupting the transformation process from chemical energy to mechanical energy in the muscle cells (Sahlin, 1986). Therefore, there has been much effort to overcome lactate accumulation in order to improve exercise performance via increasing the lactate threshold and accelerating the lactate clearance. The increase of lactate threshold can be achieved through the induction of high buffering capacity of the tissues, while the acceleration of lactate clearance can be fulfilled by speeding up the metabolic turnover of lactate through activation of oxygen utilization and increase of vascular supply, which are supposed to be induced by endurance training (Rowell *et al.*, 1966; Hajek & Perry 1967). However, all of these strategies are based on the biased biochemical view of lactate metabolism that lactate can be turned over metabolically only in the liver tissues after transportation from muscle tissues via circulation, and that the metabolic elimination by the muscle tissue itself may not play an important role. But when we considered the metabolic role of the muscle tissues during exercise, we reached the conclusion that a considerable part of lactate might be modulated in the muscle tissues either by preventing its formation or by elimination through turning over into alanine via coupling reactions of LDH (lactate dehydrogenase) and GPT (alanine aminotransferase) (Choe *et al.*, 1989; Park *et al.*, 1989). Moreover, the nature of both enzymes, LDH and GPT, as the near-equilibrium enzymes led us to assume and confirm the possible application of glutamate for compensation of lactate-induced performance decrease of muscle contraction (Choe *et al.*, 1989) and for positive effect of glutamate in exercise perfomance (Park *et al.*, 1989). Therefore, in the present experiment, we tried to extend our idea to humans, whether the application of glutamate and other aminoacids could improve the exercise performance, especially in sports competition.

MATERIALS AND METHODS

1. Materials

The diagnostic kit for lactate was purchased from Sigma Co. (St. Louis, Mo., USA). The aminoacids, monosodium glutamate, and monosodium aspartate were donated by MI-Won Co. (Seoul, KOREA), and other reagents of analytical grade were obtained from local commercial sources. The equipment for the present experiment was as follows: auto-respiratory gas analyzer (Ergo-oxyscreen model from Erich Jager Co. of Germany), auto-blood lactate analyzer (YSI 123 model from Yellow Springs Co., USA), and bicycle ergometer (Monark 65 model of Monark, Sweden).

2. Subjects

The subjects of the present study were 10 long-distance runners in the grade of national champion competition and 9 competitive cyclists with experience of more than 3 years from Seoul Athletes High School.

3. Exercise loading procedure

1) Maximal test

In order to test the physiological condition of the subjects and to determine the level of exercise load in submaximal test, we monitored VO_2 max, work load, anaerobic threshold, and blood lactate content with bicycle ergometer via the gradual increase of workload. After the subjects warmed up initially for 3 minutes with a workload of 15 watts, they took a rest for 5 minutes. Then they started a graded exercise on the ergometer until exhaustion by gradual increase of the workload, 30 watts per 2 minutes from the initial workload of 30 watts.

2) Submaximal test

The workload for an individual was determined respectively from the result of maximal test as 100 % AT level (average 77.9%, VO_2 max, range 72.7% - 85% VO_2 max) of exercise load (210 W - 245 W),. and the subjects were forced to have a 15-minute exercise with a fixed rpm of the bicycle ergometer (60 rpm) (Fig. 1).

3) Field-simulated competition experiment

In order to test the applicability of our assump-

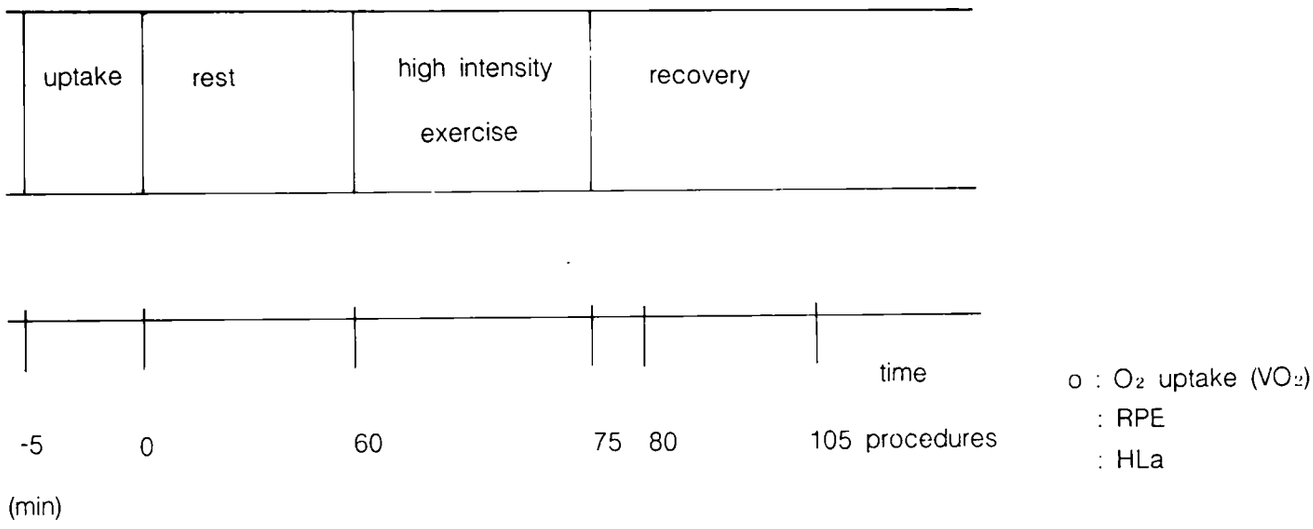

Fig. 1. Submaximal test procedures

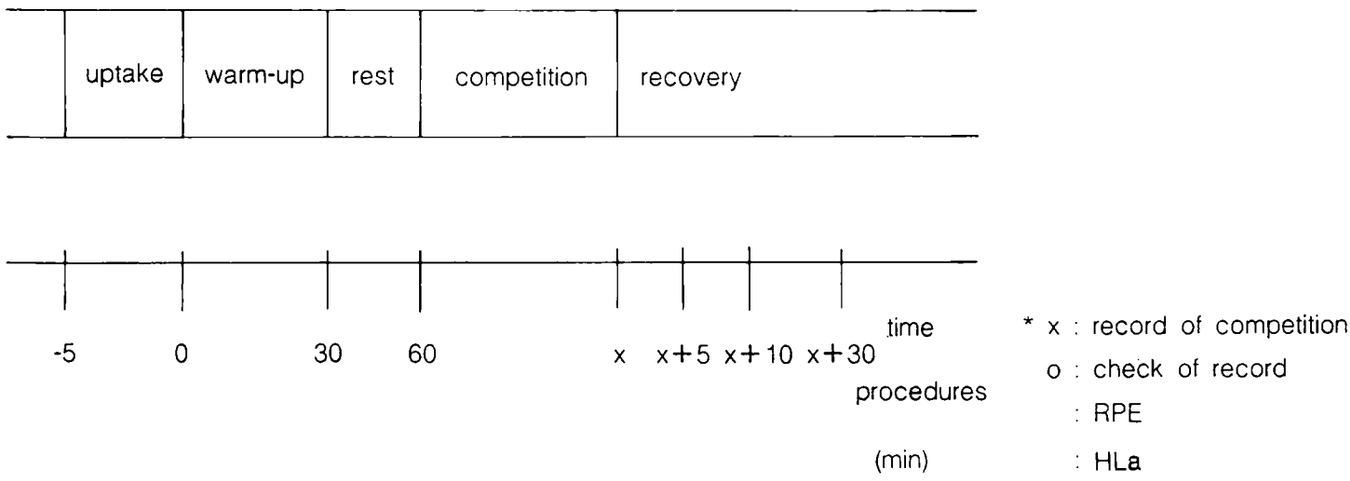

Fig. 2. Simulated-competition test procedures

tion to real competition racing, we compared the effect of glutamate administration on field-simulated competition racing of cyclists. The cycle racing was performed at Bellodrome (Chunchon) for 25 rounding (8333.3 m). The cyclists took the test drinks one hour prior to competition and warmed up themselves for 30 minutes by rounding Bellodrome and then after 30 minutes of rest, the cycle racing was carried out by starting in opposite directions pair-wise.

4) Treadmill test

The effect of glutamate uptake on the exercise performance was tested by treadmill test on long-distance runners of national champion class.

4. Composition of amino acid solution

The basic formula of the amino acid drinks in the present experiment was boiled bean sprout juice, to which glutamate was added (0.3% w/v). The control group was advised to drink physiological saline, the experimental solution A group was recommended to drink the bean sprout juice only, while the experimental solution B group, to drink the glutamate-enriched bean sprout juice. A half liter of the respective drink was administered 1 hour prior to the exercise test.

5. Procedure

The atmospheric condition of the laboratory was maintained at 21 $\pm$ 1℃ of temperature and 60 $\pm$ 5% humidity. All the subjects were restricted from having any strenuous exercise and irregular food intake for at least 48 hours before the exercise test. The blood gas and lactate were monitored

every 30 seconds by the auto-respiratory gas analyzer and an auto-blood lactate analyzer by fingertip method. After the completion of exercise load, the rate of perceived exertion (RPE) by Borg scale was subjectively checked and the blood was sampled from the antecubital vein at 5 min, 30 min, and 2 hour after the exercise to monitor the level of blood lactate. To determine the blood level of lactate, we followed the protocol supplied by the manufacturers (Sigma Co.). For the quality control of the assay we used standard metabolite control serum each time of determination. For quality control of the exercise test, we carried out the experiment of double-blind fashion and the random but triple repetitive loading test of each solution was performed on every athlete. The interval period between the tests was adjusted to be at least 3 days.

RESULTS

1. Changes in blood lactate after treadmill test

The effect of glutamate uptake on the treadmill exercise was tested in 10 long-distance runners of national champion class by administering a half-liter of solutions orally one hour prior to the test and for the effect of solution uptake on exercise efficiency, the treadmill test after experimental solution drinking was carried out twice per each individual. After warming up for 3 minutes running (200 m/min), a submaximal exercise at 80% VO_2 max (280 m/min) on the treadmill with fixation of 3° slant was loaded. Thereafter, the antecubital venous blood was sampled and determined for its con-

Table 1. Effect of amino acid uptake on the level of blood lactate in recovery period from treadmill exercise in mid-distance runners

Time after exercise	Control gorup (n=10)	Solution A group (n=20)	Solution B group (n=20)
Resting	2.490 ± 0.396	2.266 ± 0.627	2.103 ± 0.561*
3 min.	12.012 ± 2.046	11.968 ± 1.968	9.823 ± 1.947*
30 min.	6.292 ± 2.684	4.829 ± 1.958	3.003 ± 0.968*
2 hr.	2.321 ± 0.495	2.277 ± 0.759	1.584 ± 0.396*

All the data are means ± S.D. (mmol/l)

* $P < 0.005$: Values of solution B group were compared to those of control group

Table 2. Effect of amino acid uptake on the level of blood lactate during the submaximal test in high-school cycling athletes

Time of work load	Control group (n=9)	solution A group (n=9)	Solution B group (n=9)
5 min	4.34 ± 1.0	5.03 ± 1.36	4.56 ± 0.86
10 min	5.21 ± 1.31	5.79 ± 1.63	5.18 ± 1.26
15 min	6.56 ± 1.37	7.46 ± 1.23	7.24 ± 1.69

All the data are means ± S.D. (mmol/l)

Table 3. Effect of amino acid uptake on VO_2 during the submaximal test in high-school cycling athletes

Time of work load	Control group (n=9)	Solution A group (n=9)	Solution B group (n=9)
5 min	227.59 ± 21.86	223.87 ± 20.34	227.08 ± 15.14
10 min	275.16 ± 21.31	278.46 ± 13.61	276.48 ± 21.27
15 min	302.48 ± 24.43	306.08 ± 21.8	298.75 ± 25.28

All the data are means ± S.D.

Table 4. Effect of amino acid uptake on heart rate during the submaximal test in high-school cycling athletes

Time of worklod	Control group (n=9)	Solution A group (n=9)	Solution B group (n=9)
5 min	155.6 ± 7.17	156.02 ± 9.98	155.74 ± 7.02
10 min	173.76 ± 5.81	173.54 ± 9.13	171.36 ± 8.05
15 min	182.49 ± 5.48	183.84 ± 8.2	179.06 ± 7.66

All the data are means ± S.D. (beats/min)

Table 5. Effect of amino acid uptake on the level of blood lactate in the recovery period from the field-simulated competition test in high-school cycling athletes

Time after exercise	Control group (n=9)	Solution A group (n=9)	Solution B group (n=9)
resting	1.24 ± 0.21	1.41 ± 0.32	0.84 ± 0.35
5 min	8.24 ± 2.34	8.66 ± 2.13	7.56 ± 2.76
10 min	3.46 ± 1.29	3.47 ± 1.81	3.23 ± 1.96

All the data are means ± S.D. (mmol/l)

Table 6. Improvement of exercise efficiency by amino acid uptake in submaximal test and the field simulated competition test

Items	Number of improved cases / total cases
Decreased VO_2	3/9
Decreased blood lactate	5/9
Decreased heart rate	6/9
Lower RPE scale	4/9
Improved records	5/9

tent of lactate by function of time after exercise load. As shown in Table 1, the blood lactate level at 3 minutes after exercise was significantly lowered in the glutamate-enriched solution group ($P < 0.005$), where 12.012 ± 2.046 m mol/l in control A group, 11.968 ± 1.958 m mol/l in A group and 9.823 ± 1.947 m mol/l in solution B group. Moreover, the blood lactate level was also significantly decreased ($P < 0.005$) in solution B group of glutamate-enriched drink at 30 minutes and 2 hours after exercise.

2. Changes in cyclists after field-simulated competition

The blood lactate changes after the submaximal test in experienced high-school cyclists are summarized in Table 2. Contrary to data from long-distance runners, the blood lactate level was not significantly changed by the amino acid-enforced drinks in the submaximal treadmill exercise among the high-school cyclists, and no significant differences were observed in VO_2 max among the tested groups, although the number of cardiac beats seemed to be decreased in the experimental solution B group (Table 4). Among the test groups, the individual variations were so wide that it was difficult to form a unified conclusion; for example, the administration of glutamate enriched solution lowered VO_2 max in 3 cases (30%), blood lactate content in 5 cases (56%), subjective fatigue feeling in 4 cases (44%) among 9 cases (Table 6). Nevertheless it seemed to be clear that in some individuals, the uptake of glutamate-enriched solution could improve the exercise performance. And in case of field-simulated competition test of cycling, the glutamate uptake group showed the improvement of racing records in 5 cases (56%) out of 9 cases, though there were no significant changes in RPE scale. In addition, the changes of blood lactate content after the competition indicated the possible acceleration of lactate clearance though statistical significance was low; 7.56 ± 2.76 m mol/l in group solution B while 8.24 ± 2.34 m mol/l in control A group at 5 minutes after exercise, and

3.23 ± 1.96 mmol/l in solutuion B group, while 3.46 ± 1.29 m mol/l in control group at 30 minutes after the test.

DISCUSSION

It is well known that under conditions of high-energy demand, rapid fluctuation of the energy requirement, and an insufficient supply of oxygen, lactic acid is accumulated in the peripheral tissues (Hill & Lupton, 1923, Sahlin, 1986). Although inefficient in the aspect of ATP production, lactate formation has several advantages over aerobic process, such as rapid acceleration of ATP generation, ATP production in the absence of oxygen, and compensation of NAD consumption via glycerol phosphate shuttle (Sahlin, 1986). Despite some metabolic advantages of lactate generation, the product lactate contributes more than 85% of the released H^+, the major cause of lowering tissue pH and the consequent fatigue phenomenon (Sahlin, 1978). Therefore, there have been many efforts to overcome fatigue and lactate accumulation in the tissues in order to improve the perfomance efficiency either by increasing the lactate threshold or by supplying more energy sources, mostly of high calorie carbohydrate. Nonetheless, these strategies were limited in the biochemical aspect that the high concentration of lactate in the exercising muscle tissues would essentially remain un affected (Stenvold & Hermansen, 1969). Consequently, we devised the novel strategy of reducing the lactate level by activating the coupling reactions of lactate dehydrogenation to pyruvate and transamination into alanine (Park *et al.*, 1989). As both enzymes of LDH and GPT are of near-equilibrium properties, one of the important rate-regulating factors is the concentration of the substrates, themselves.

In this case, the high concentration of glutamate would activate the transamination reaction of pyruvate into alanine, which consequently reduce the level of lactate via the equilibrium reaction of LDH. Actually, the enzymatic activities of LDH and GPT are considerably high and inducible by exercise training in the muscle tissues (Park *et al.*, 1989). Moreover, the arteriovenous differences of the muscle tissues in amino acid compositions were marked by the extremely high level of alanine and glutamine in the venous blood, suggesting the ready synthesis of 2 amino acids in the exercising muscle tissue consuming the glutamate (Ahlborg *et al.*, 1974).

With the above assumption, we carried out a series of experiments; for example, the enzymes for the coupling reactions in the muscle tissues were shown to be induced by the exercise training, and the glutamate administration prolonged the survival time in the swimming test with rats (Park *et al.*, 1989), and the improvement in lactate-induced performance decrease of muscular contraction by administration of glutamate in cats (Choe *et al.*, 1989). From these results, we tried to extend our assumption to human subjects, especially of long-distance runners and cyclists. In the case of long-distance runners of national champion grade, the administration of glutamate-enriched solution lowered the level of blood lactate significantly after exercise (Table 1). Significantly, the blood lactate level at 3 minutes after the exercise was shown to be decreased 18% in the glutamate-administered group. Moreover, recovery to the control level of blood lactate was accelerated in glutamate group. These data indicated that glutamate administration prior to the strenuous exercise could prevent peripheral lactate accumulation, with a possible reduction in fatigue and thereby, might improve the performance efficiency. By the way in the case of the high-school cyclists, the data on blood lactate, VO_2 max and the number of cardiac beats were not significantly different between the control and the glutamate-intake groups (Table 2,3,4). However, the data analysis showed wide differences in individval responses to glutamate-intake on performance among the cyclists, such as decreases in VO_2 max (3/9), in blood lactate (5/9), cardiac beat (6/9) and in RPE scale (4/9). Therefore, these data might be compatible, with the assumption that glutamate intake might improve the performance efficiency, though not highly significant and variable up to individuals. In addition, the data from the field-simulated competition test of cyclists showed the similar tendency of decrease in blood lactate level and improvements in racing records (5/9) by glutamate-intake after work load, despite the low statistical

significance (Table 5). These human data might not be conspicuous as data from our previous animal experiments (Park *et al.*, 1989; Choe *et al.*, 1989). With the human subjects, it was very difficult to standardize the subjects in all the biological conditions, and the individual differences in many biochemical parameters were so wide that the statistical analysis data might be required to design a large-scale of experiment to test the effectiveness of our prescription. Moreover, it was very hard to control the physiological and psychological factors of the human subjects in order to induce maximum performance capacity.

However, from our experiments, we observed that the biochemical responses to glutamate intake were essentially different between the long-distance runners and the cyclists. Actually, in long-distance running, the continuous muscle contraction caused accumulation of high levels of lactate, while in cycling, swimming or in intermittent exercise, the lactate was not accumulated (Vollestaad & Sejersted, 1988). Therefore, this kind of difference in metabolic response to workload might partially explain the differences in the present data between the treadmill test of the long-distance runners and the bicycle ergometer test of the cyclists.

From these results and our previous data of animal experiments (Park *et al.*, 1989; Choe *et al.*, 1989), it could be concluded that the administration of glutamate and the related amino acid components as forms of drinks or enriched foods might improve exercise performance and could reduce fatigue of the tissues. In order to extend this concept to human beings, it is necessary to confirm the safe dose of the prescription before determination of the effective dose, for which at least 5 g per day of glutamate is reported to be tolerable (Airoldi *et al.*, 1979). Actually, our prescription is far below the tolerable dose. This kind of idea can be applied not only for sports but also for other general phenomenon of fatigue either from physical or from psychological causes, as far as the basic mechanism of fatigue is related with the lactate accumulation.

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=국 문 초 록=

글루탐산투여가 중장거리선수 및 싸이클링선수의 경기력에 미치는 영향

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격심한 운동시 근육내 lactate가 축적되고, 결과적으로 근육의 피로가 증가되어 근육의 작업수행능력이 저하됨은 잘 알려져 있다. 그러나 운동부하가 증가됨에 따라 ATP 에너지 수요가 증가되기 때문에 고농도 당질을 섭취케 하여 에너지원을 보충시킴으로써 운동효율을 증가시키려고 노력해 왔으나, 동 시도가 결과적으로 lactate의 축적을 오히려 가속시키는 현상이 초래될 가능성을 간과할 수 없다. 근육조직내의 lactate는 Cori cycle에 의하여 당신생반응(gluconeogenesis)에 재이용되거나, pyruvate를 통해 citric acid cycle에서 산화되는 경로에 의하여 처리된다고 일반적으로 이해되고 있으며, lactate dehydrogenase(LDH)에 의하여 lactate가 가역적으로 변환되는 pyruvate가 transamination reaction에 의하여 쉽게 alanine 등으로 전환되어 처리될 수 있음은 흔히 간과되어 왔다. 따라서 본 연구에서는 이러한 사실을 바탕으로, 조직내 transamination 반응의 기질인 glutamate을 보충공급하여 줌으로써 pyruvate를 alanine으로 전환하는 반응을 촉진하여 부차적으로 조직내 lactate의 축적을 방지할 수 있으리라는 가정하에 실험을 시행하여 다음과 같은 결과를 얻었다. 첫째, treadmill test에서 glutamate 보충 아미노산액의 투여시, 운동 후 혈액중 lactate clearance가 유의하게 촉진되었다. 둘째, glutamate 보충아미노산액 투여군에서 운동중 혈액중 lactate 함량이 저하되어 있었다. 셋째, glutamate 보충아미노산액 투여군에서 경기력은 일부선수에서 향상되었다. 이상의 결과로 운동전 glutamate 보충 아미노산의 투여는 근육운동으로 생성되는 lactate의 조직내 축적을 방지하여 근피로도를 감소시키고 아울러 운동효율을 증진시킬 수 있는 것으로 사료되었다.