



## The effect of Dimethylglycine (DMG) administration on Biochemical Blood Parameters in Youth elite Basketball Players

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### Abstract

The purpose of the study was to compare the effect of sodium bicarbonate and dimethylglycine on biochemical blood parameters and anaerobic function in youth elite basketball players. Fifteen youth elite basketball players with 15 – 17 years old and 72.02 ± 2.56 Kg divided into three groups: placebo group, sodium bicarbonate group and dimethylglycine group. During the study amount of 500 (m.l) solutions that content 300(m.g) sodium bicarbonate per/kg of body weight was given to the sodium bicarbonate group. In addition, the dimethylglycine group received a 400(m.g) dimethylglycine per/kg of body weight and third group received a 500(m.l) placebo solution. Blood samples were collected before and after of supplement administration and especially after of performance the Wingate test (30 second). Process of data gathering was analyzed by SPSS software. Results of the study shown that after administration of sodium bicarbonate and dimethylglycine supplements, blood lactate was increased but it finding statistically not significant ( $P > 0/05$ ). Nevertheless, after administration of sodium bicarbonate amount of extracellular bicarbonate buffers statistically was significant increase ( $P < 0/05$ ) and amount of pH was increased but it finding statistically not significant ( $P > 0/05$ ). In addition, none of biochemical and functional indexes for Dimethylglycine group statistically was significant. Collectively, ingestion of sodium bicarbonate in youth athletes significantly can increase blood-buffering capacity during high intensity interval exercises. Simultaneously, ingestion of sodium bicarbonate maybe due to increase in buffering capacity improves anaerobic capacity. In addition, administration of dimethylglycine supplement not improves anaerobic function in high intensity interval exercises.

**Keywords:** Sodium bicarbonate, dimethylglycine, anaerobic capacity.

### Introduction

During the past several years, a great furor has arisen over the promotion and sale of pangamic acid also known as Vitamin B15<sup>1</sup>. This was stimulated to some extent by an article in New York magazine<sup>2</sup>. Extolling this "vitamin", resulting in near exhaustion of supplies in local retail outlets, a phenomenon which eventually spread to other areas of the country. This article presents some of the evidence supporting the claims of efficacy by the proponents of B15 as well as some pertinent history and facts surrounding the recent court decision regarding sale of this controversial substance. Vitamin B15 has been particularly touted as an "energiser, a natural metabolite which increases the efficiency of certain metabolic oxidative processes, thereby being recommended for use by athletes to increase their performance and endurance", for example. Vitamin B15 and pangamic acid are used synonymously in this article. However, this substance is not specifically defined because studies noted in this article have used various chemical entities, all designated as B15. Most of these substances contained dimethylglycine alone or mixed with gluconic acid. Until recently, Vitamin B15 was quietly being sold in health food stores and, despite the controversy it later aroused, it

attracted relatively little attention from the medical profession, the regulatory authorities represented by the FDA, or the consumer. Nevertheless, pangamic acid was stigmatized at the outset. It was introduced and promoted by the same Dr. Krebs who was responsible for Laetrile, the anti-cancer agent the use of which is considered quackery and pseudo-science by the established medical community. Despite the provocative claims made by Krebs, virtually no substantial research was conducted in this country following the initial 1951 publication of the announcement of its isolation from apricot pits and its chemical structure<sup>3</sup>. According to Dr. Krebs, B15 could be found in rice bran, brewer's yeast, horse liver, barley, wheat seeds, oats and corn, being a companion to the recognized members of the B Complex vitamins, also found in these natural sources. Krebs, in an effort to improve upon his initial "discovery", announced an alternative to the original B15 which was a diisopropyl ammonium dichloroacetate (DIPA) derivative. DIPA was not new despite its promotion as the "real" B15<sup>4</sup>. It had been included in several OTC products for its vasodilator effect. It is now known that the ester structure with gluconic acid is very unstable<sup>5</sup> and to isolate the intact structure from natural sources would be very difficult. The procedure, as described by Krebs (1951) has yet to be reproduced by other scientists. Also, if it

does exist naturally in foods as the ester, it would undoubtedly be broken down by the gastric juices into free gluconic acid and DMG or DIPA. In this country, the designation, "Vitamin" is now unanimously considered by both FDA and the manufacturers to be a misnomer. There is no deficiency disease associated with it and minimum daily requirements cannot be defined. Therapeutic doses range from 50 to 150 mg daily. Russia and two volumes of research reports resulting from symposia on this substance have been published, available in English translation<sup>6</sup>. It should be noted that it is not always clear as to the exact composition of the B15 substance referred to in those papers, although it probably contained DMG mixed with calcium gluconate or gluconic acid for the most part. B15 seems to be a safe substance despite the lack of published long-term toxicity studies; no untoward effects have been reported to date. Doses as high as 200 mg/kg given s.c. to rats caused no toxic effects<sup>7</sup>. The lack of toxicity is not surprising since DMG is a natural metabolite, a simple glycine derivative. The Russians feel that pangamic acid is a valuable therapeutic agent, and they have studied it clinically in many disease states. The conclusions of efficacy as a result of this research should be tempered by the fact that most of these studies were not controlled, often lacking adequate comparable control groups, or including other drugs with the B15 therapy. DMG, a component of the Russian B15, is purported to contribute methyl groups in the transmethylation process which results in improved energy utilization. Methionine, choline and betaine are some other common substances capable of donating methyl groups in certain metabolic synthetic processes occurring in the body. It is suggested, based on experimental evidence in rat experiments, that the DMG component of pangamic acid acts similarly, a participant in transmethylation processes<sup>4</sup>. This possible mode of action is often proposed as the basis of its activity, such as the observation that various animal species — rats, mice, cats and dogs — have increased tolerance to altitude, asphyxia, hypoxia (particularly that induced in brain and heart tissue), and fever induced by certain pathogens<sup>8,9</sup>. So, purpose of the study was to compare the effect of sodium bicarbonate and Dimethylglycine on biochemical blood parameters and anaerobic function in youth elite basketball players.

## Material and Methods

**Subjects characteristics:** The subjects were 15 young elite basketball players, with at least  $5.3 \pm 0.3$  years of national competition. The research was conducted during the pre-competitive season thus the values of anaerobic and aerobic capacity were at maximum or near maximum levels. Three of the subjects resigned during the experiment due to participation in league competitions. All subjects were randomly chosen from between of basketball players that preparation to take a part at national competition. The Subjects characteristics were consisting of: number of subjects = 45; training experience of the subjects =  $5.3 \pm 0.3$  years; age of they were  $16.46 \pm 0.37$  years old;  $VO_{2max} = 51.53 \pm 4.46$  ml.kg<sup>-1</sup>.min<sup>-1</sup>; body height (BH) =  $1.82 \pm 0.01$  m; body mass (BM) equal with  $72.02 \pm 2.5$  Kg; fat free mass (FFM) =  $65.22 \pm 5.02$  Kg; fat content (FAT

) =  $7.01 \pm 1.33$  %), and BMI equal with  $21.62 \pm 0.54$ . The research project was approved by the Ethics Committee for Scientific Research at the Academy of Physical Education and sport sciences in Mashhad, Iran.

**Experimental design:** The experiment had two phases. Before the start of the experiment, initial values of body mass and body composition (BM, FFM, FAT% and total body water (TBW)) were evaluated with the use of electrical impedance (In body model of 720, made in South Korea). To increase the reliability and validity of body composition measurement by electrical impedance all tested subjects were evaluated under the same conditions during all 2 phases of the experiment (measurement during the same time of the day 8-9 am o'clock, active rest the day before testing, full hydration of the body, last meal at 8pm on the day before evaluation. In fact, all of subjects were 12 hours in breakfast position). Resting blood samples were drawn from the med-cubital vein to determine several biochemical variables. Blood samples were drawn upon arrival to the laboratory, immediately after doing of Wingate test, during passive recovery. It should be noting that type of recovery in all of young elite basketball players was from type of passive recovery. In fact, the subjects immediately after execute of both test lying on the bedstead. Also, a progressive ergocycle test (Wingate) was administered to determine several anaerobic functions (experimental test). For increase of accuracy in the study, both of blood sampling and analyze of body composition were executed to homogenate all of subjects in one day pre-execution of first phase.

Plasma lactate (LA) concentration was determined enzymatically using commercial kits (Boehringer Diagnostika, Mannheim, Germany). Blood PH, standard bicarbonate (SB) and base excess (BE) were measured using a 168 pH Blood-Gas Analyzer (Ciba-Corning, Basel, Switzerland). The intra and interassay coefficients of variation for lactate were 3.2 and 8.9% respectively.

**Experimental Protocols: Wingate test:** The Wingate protocol has five distinct time periods: i. prior exercise; ii. recovery interval; iii. acceleration interval; iv. the Wingate Test (Bar-Or, 1983); and v. cool down recovery. As with the other anaerobic tests, prior exercise recommended. Two proponents of the test encourage the use of a warm-up that includes five minutes of low intensity pedaling interspersed with four: to five all-out sprints of 4-6-s duration; each of the sprints should be against the prescribed resistance for the Wingate Test (Inbar and Bar-Or, 1986). The recovery interval between the ends of the prior exercise and the beginning of the Wingate Test should not be less than two minutes after the prior exercise, not more than 5 minutes after the warm-up portion of the prior exercise. The 2-min minimum would provide some time for recovery from any possible fatigue that may have occurred during the warm-up; the 5-min maximum would still retain muscle temperature and blood flow to a significant extent. The activity during the recovery interval may consist of simply resting while seated on

the bike or pedaling at a minimal resistance (e.g., 1 Kg or 10 N at an rpm between 10 and 20). The acceleration period is very brief. It begins immediately after the recovery interval and consists of two time components. In the first, the subjects pedals at about 20 rpm for about 10 s at a resistance that is about one third of the prescribed the Wingate resistance. In the second, the subjects gradually increases the rpm while the technician increases the resistance to the prescribed force (F) setting in less than 5 seconds; thus the acceleration period lasts no more than 15 s. The actual duration of all-out cycling for the Wingate Test is 30 seconds. The 30 seconds is divided into six continuous time intervals of 5 seconds each. The cool down period lasts for one to two minutes and consists of pedaling at a low to moderate aerobic power level on the bike ergometer immediately after the Wingate Test.

**Experimental evaluations: Wingate test:** The calculations for the Wingate test result in anaerobic capacity measurements that may be expressed in units of Joules, Kilogram-meters in 30 seconds (Kgm-30 s) or watts (W), and in anaerobic power measures expressed as Kgm-5 s or W. Expressions of these units may be expressed also in relative terms by dividing them by body weight (e.g., W. Kg). To calculate peak anaerobic power in units of Kgm-5 s, equation 1 is used whereby the highest number of revolutions among the 5 second intervals in multiplied by force (Kg) setting. This is then multiplied by six (the distance in meters that the wheel travels for each pedal revolution).  $\text{Peak-AnP (Kgm-5 s)} = R_{\text{max}} \text{ in } 5 \text{ s} \times D/r \text{ (m)} \times F \text{ (Kg)}$  Eq.1. To find the anaerobic capacity (AnC), the total number of revolutions in 30 s is multiplied by six meters in order to obtain the total distance. Then, as before, the distance is multiplied by the force to obtain the work accomplished in 30 s. Equation 2 is used to calculate anaerobic capacity.  $\text{AnC (Kgm-30 s)} = \text{total R in } 30 \text{ s} \times 6 \text{ m} \times F \text{ (Kg)}$  Eq.2. To convert Peak-AnP and AnC to watts, a true power unit, equations 3 and 4 were used respectively. Both of the watts conversions are based upon a hypothetical one minute time period.  $\text{Peak-AnP (W)} = 2 \times \text{Kgm-5s}$  Eq.3. Thus, from our previous equations peak- AnP would be converted to watts (W) by multiplying the number by 2. The conversion of AnC from Kgm-30s to W is made in equation 4.  $\text{AnC (W)} = (\text{Kgm-30s})/3$  Eq.4. Because relative anaerobic power is often more important than absolute power, the Wingate scores that were expressed in watts are often divided by body weight (equations 5 and 6). Thus, the scores to indicate relative (Rel) Peak-AnP and rei AnC were expressed in units of W. Kg<sup>-1</sup>.  $\text{Rel Peak-AnP (W.Kg}^{-1}\text{)} = \text{W/kg}$  Eq.5 and  $\text{Rel AnC (W.Kg}^{-1}\text{)} = \text{W/Kg}$  Eq.6. In parallel to it, fifteen youth elite basketball players with 15 – 17 years old and 72.02 ±2.56 Kg divided into three groups: placebo group, Sodium bicarbonate group and Dimethylglycine group. During the study amount of 500 (m.l) solutions that content 300(m.g) sodium bicarbonate per/kg of body weight was given to the sodium bicarbonate group. In addition, the Dimethylglycine group received a 400 (m.g) Dimethylglycine per/kg of body weight and third group received a 500(m.l) placebo solution. Blood samples was collection before and after of supplement administration and

especially after of performance the Wingate test (30 second)<sup>10</sup>.

**Statistical analysis:** The obtained data were analyzed statistically with the use of SPSS (V18). The results were presented as means ( $\bar{X}$ ) and standard deviation (SD). As for that, type of validity in the study was kind of interval variable we used from repeated measure with Tukey HSD and Bonferroni test as post hoc tests. Statistical significance was accepted at  $p < 0/05$  <sup>11,12,3,14,15</sup>.

## Results and Discussion

Results of the study shown that after administration of sodium bicarbonate and dimethylglycine supplements, blood lactate was increase but it finding statistically not significant ( $P>0/05$ ). Nevertheless, after administration of sodium bicarbonate amount of extracellular bicarbonate buffers statistically was significant increase ( $P<0/05$ ) and amount of pH was increase but it finding statistically not significant ( $P>0/05$ ).

In parallel of our results one author shown that in a crossover study, either a placebo paste or N,N-dimethylglycine was administered orally at a dose rate of 1.2 mg/kg twice daily for five days to six thoroughbred horses, with bodyweights ranging from 424 to 492 kg. Using previously determined regression equations for oxygen uptake ( $\text{VO}_2$ ) against speed for each horse, a standardised exercise test was given with speeds equivalent to fixed percentages of the maximum oxygen uptake ( $\text{VO}_2\text{max}$ ). The test consisted of two minutes at speeds equivalent to approximately 40 per cent and 50 per cent  $\text{VO}_2$  max, and one minute at speeds that produced approximately 60, 70, 80, 90 and 100 per cent  $\text{VO}_2$  max. During the last five seconds of each exercise stage, the values of  $\text{VO}_2$ , carbon dioxide production ( $\text{VCO}_2$ ), heart rate, arterial blood and plasma lactate concentrations, arterial blood gases and pH were measured. Before and immediately after the exercise test, muscle biopsies were collected from the middle gluteal muscle to determine the muscle lactate concentrations. The administration of N,N-dimethylglycine produced no significant differences in any of the measured values, and it is concluded that the compound has no beneficial effects on cardiorespiratory function or lactate production in the exercising horse<sup>16</sup>.

But in other study shown effects of Pangamic acid (B-15) ingestion was examined during acute submaximal exercise on eight volunteer subjects. During the experimental condition subjects orally ingested 0.40-g tablets, six per day, of an equal molar mixture of calcium gluconate and N,N-dimethylglycine for 12 weeks. When compared to a control condition there were no differences in oxygen kinetics during exercise or recovery, exercise heart rates, and resting or exercise blood lactates. It was concluded that a 2-week ingestion of Pangamic acid does not result in any metabolic or circulatory advantages for human subjects during short-term submaximal exercise<sup>17</sup>. Exercise endurance and metabolic patterns were determined in rats that had been administered commercial preparations of “pangamic acid,” dimethylglycine, or diisopropylamine subcutaneously.

None of these substances affected (a) the time required to reach exhaustion by treadmill running, (b) the specific activities of succinate dehydrogenase of liver and muscle, (c) the capacity of liver mitochondria for oxidative N-demethylation, (d) the content of mitochondria in liver, (e) the ratios of soluble to membranous protein of the liver mitochondria, or (f) the level of urinary excretion of creatinine. The concentrations of glycogen in muscle and liver were not increased. These results do not substantiate previous reports by Russian investigators on whose work many athletes base their practice of consuming pangamate to improve performance<sup>18</sup>. In contrast, one author survey the effects of DMG on the maximal exercise performance of well-trained college women. they hypothesized that pre-exercise DMG consumption would lead to an increase in [latin capital V with dot above]Vo<sub>2</sub>max. Nine well-trained college women (age = 20 +/- 1 y, height = 165.2 +/- 4.5 cm, body mass = 63.6 +/- 5.6 kg, [latin capital V with dot above]o<sub>2</sub>max = 49.4 +/- 2.8 ml/kg/min were recruited as subjects. Subjects ingested three, 800mg doses of DMG or a placebo (PLC) on two occasions in a

randomized, double-blind fashion. Thirty minutes after the last dose subjects performed graded, maximal treadmill exercise to exhaustion. [latin capital V with dot above]o<sub>2</sub>, heart rate, perceived exertion, and time to exhaustion were compared between conditions. They results not shown significant differences were found using independent t-tests in the DMG versus the placebo trials on maximum heart rate (DMG 191.4 +/- 7.5 bpm PLC 191.2 +/- 6.1 bpm, p = 0.79), [latin capital V with dot above]o<sub>2</sub>max (DMG 48.8 +/- 2.6 ml/kg/min PLC 49.2 +/- 2.6 ml/kg/min, p = 0.56), and exercise time (DMG 12.81 +/- 42 s PLC 12.84 +/- 0.45 min, p = 0.81). Further, during submaximal exercise stages, heart rate (p = 0.52, perceived exertion (p = 0.48), and respiratory exchange ratio (p = 0.65) were not different between treatments. Based on these results, it can be concluded that DMG has no significant effect on improving the endurance capacity of well-trained women. Practical Applications: There is no evidence to recommend DMG as an ergogenic aid to endurance athletes<sup>19</sup>.

**Table-1**  
**Amounts of blood biochemical variables in youth elite basketball players during pre, immediately after administration and after of wingate test**

Blood biochemical variables						
Stages	Groups	PH	PCO2 (mmol/L)	PO2 (mmol/L)	Bicarbonate (mmol/L)	Lactate (mg/dL)
Before administration	Sodium Bicarbonate	7.34 ± 0.03	44.7 ± 4.2	38.3 ± 4.43	24.2 ± 2.14	10.46 ± 1.91
	Dimethylglycine	7.34 ± 0.02	40.82 ± 1.96	40.7 ± 7.8	22.38 ± 2.42	9.4 ± 2.85
	Placebo	7.34 ± 0.02	43.28 ± 1.8	41.7 ± 5.28	23.1 ± 1.21	9.42 ± 2.18
After administration	Sodium Bicarbonate	7.37 ± 0.02	46.9 ± 4.11	44.4 ± 3.27	26.7 ± 2.32	13.3 ± 2.83
	Dimethylglycine	7.3 ± 0.05	48.2 ± 4.16	45 ± 4.83	23.9 ± 1.14	10.9 ± 2.31
	Placebo	7.3 ± 0.03	44.4 ± 2.27	48.1 ± 10.4	22.7 ± 1.73	12.3 ± 2.52
After of wingate test	Sodium Bicarbonate	7.2 ± 0.07	65.2 ± 5.78	28 ± 3.1	24.7 ± 2.07	54.3 ± 9.61
	Dimethylglycine	7.1 ± 0.08	70.2 ± 13.5	31.9 ± 6.74	22.1 ± 1.01	71.8 ± 18.7
	Placebo	7.1 ± 0.04	65.7 ± 7.7	29 ± 4.42	22.2 ± 0.41	66.5 ± 15.7
<b>Chenges</b>						
Within groups	F	147.7	83.49	29.57	4.24	160.27
	significant	0.000 <sup>+</sup>	0.000 <sup>+</sup>	0.000 <sup>+</sup>	0.026 <sup>+</sup>	0.000 <sup>+</sup>
Between groups	F	1.75	0.27	0.79	6	1.34
	significant	0.24	0.767	0.42	0.016 <sup>+</sup>	0.297
groups × factor	F	1.28	1	0.35	1.43	1.83
	significant	0.305	0.426	0.834	0.253	0.156

Data shown as mean ± standard deviation. <sup>+</sup> Significant level was p < 0.05.

## Conclusion

In addition, none of biochemical and functional indexes for dimethylglycine group statistically was significant. Collectively, ingestion of sodium bicarbonate in youth athletes significantly can increase blood-buffering capacity during high intensity interval exercises. Simultaneously, ingestion of sodium bicarbonate maybe due to increase in buffering capacity improves anaerobic capacity. In addition, administration of dimethylglycine supplement not improves anaerobic function in high intensity interval exercises.

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