

## The Effect of Sodium Bicarbonate Supplement on Lactic Acid, Ammonia and the Performance of 400 Meters Male Runners

### Authors' Contribution:

A – Study Design  
 B – Data Collection  
 C – Statistical Analysis  
 D – Data Interpretation  
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**Key words:** alkalosis, anaerobic performance, lactic acid, ammonia

### Abstract

**Background:** The aim of the present study was to investigate the effects of sodium bicarbonate supplementation on anaerobic performance and some plasma metabolites.

**Material/Methods:** 16 young male athletes (age 20.58±3.25, Height 175.33±2.48 cm and BMI 21.57±2.68 kg/m<sup>2</sup>) participated in two 400 m running sessions one hour following sodium bicarbonate or placebo (calcium bicarbonate) supplementation with the counterbalanced order. There were three blood sampling phases (resting condition, 30 min after supplementation and 2 min after 400 m running) in both of the sessions and the blood pH; ammonia, lactate, and HCO<sub>3</sub><sup>-</sup> levels were measured.

**Results:** The results showed that running time in the sodium bicarbonate session was significantly lower (57.41±0.11 sec) than the placebo (59.01±0.78 sec) session (P<0.05). The blood HCO<sub>3</sub><sup>-</sup> levels (29.53±2.64 mmol/L) increased with respect to the resting values (23.13±1.84 mmol/L) with sodium bicarbonate administration; however, it decreased to the pre-exercise level (20.45±1.92 mmol/L) after running (P<0.05). The HCO<sub>3</sub><sup>-</sup> levels (17.89±2.48 mmol/L) also decreased under the resting values (23.31±2.15 mmol/L) after running in the placebo session (P<0.05). Significant increases were observed in blood lactate levels in both of the sessions; however, blood pH significantly decreased only in the placebo session (P<0.05).

**Conclusions:** It can be concluded that alkalosis can be considered as an ergogenic method and can prevent a decrease in blood HCO<sub>3</sub><sup>-</sup> during high intensity exercise.

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## Introduction

High-intensity muscle contraction can lead to intracellular changes in pH. As such, at the start of doing exercise, due to creatine phosphate catabolism, alkaline is created, and later with increased glycolysis, the acidic state becomes dominant [1]. There is evidence for the fact that using any buffer agent (such as sodium bicarbonate and sodium citrate) before exercise, along with repeated intensity construction exercise, can prevent the accumulation of  $H^+$  in skeleton muscles and decrease interstitial fluid and blood [2]. In addition, after short intensive exercise, the pressure of venous carbon dioxide increases [3], which causes hyperventilation at recovery. Therefore, it is expected that after using sodium bicarbonate, the decrease in pH level while exercising and during recovery will be compensated for. Therefore, the effect of using bicarbonate salts on exercise performance has been studied in several studies, and there have been reports of an increase in both buffer capacity and extracellular pH [4, 5], which may, in turn, cause an increase in the flow of H through sarcolemma. Similarly, there is clear evidence supporting the role of loading bicarbonate in preventing fatigue resulting from pH decrease [4]. Most probably, the effect results from facilitation of positive hydrogen and carboxylate transfer and the exchange of sodium and hydrogen through sarcolemma [6]. Moreover, there are clues confirming the fact that the creation of metabolic alkaloses can lead to an increase in the concentration of ionized lactate ( $[Lac^-]$ ) during the exercise and buffering the resulting lactate, while limiting the effects of pH reduction may lead to improved performance [7]. On the other hand, it has been reported that using sodium citrate at rest brings about a decrease in hydrogen ionic plasma and has no parallel effect on intercellular fluid, although it causes simultaneous improved performance [8, 9].

Using alkaline may cause the concentration of venous lactate and may be associated with the transfer of lactate from intercellular fluid to venous space. However, in the study no effect was reported on buffer capability and  $H^+$  concentration [10]. As such, considering the fact that lactate and  $H^+$  transfer from muscle cells depends on  $H^+$  concentration, it is supposed that an increase in the activities of mechanisms involved in ionic transfer accounts for increased venous concentration during the exercise [11]. It is also assumed that with an increase in performance level with using alkaline, the role of glycolysis in generating energy for activities automatically increases. Therefore, in such cases observing higher lactate plasma may be a usual phenomenon [12]. Due to the lack of some research evidence on the ineffectiveness of ergogenic loading of bicarbonate at present [11], no definite conclusions have been made about these findings. In addition, the role of low pH on muscle fatigue has been questioned [13].

Moreover, it seems necessary to mention that ammonium is formed in the kidneys as a result of the activity of the glutaminase enzyme dependent on phosphate (far curved tube) [14] and independent of phosphate (far curved tube) and all the produced  $NH_3$  in the urine tubes appears in the form of ammonium ion ( $NH_4^+$ ). Due to the fact that in urine collecting tubes there is little penetrability for ammonium release mechanism, a little proportion of ammonia from intercellular fluid enters through non-ionic distribution.

In addition, in case of an increase in urine pH by the injection of bicarbonate or stasolamid, ammonium decomposes and ammonium enters blood [15]. Therefore, there are reports indicating that prolonged intensive exercise can increase the plasmid ammonium rate; the athletes have been observed to have lower rates [16]. On the other hand, some evidence shows that lactate and ammonia metabolism during exercise are independent of each other. It has also been reported that in intensive progressive exercises, the metabolic alkalosis has no effect on the accumulation of ammonia in plasma or the endurance capacity [17].

Therefore, it becomes clear that despite obvious evidence indicating the ergogenic effects of using buffer material, there is no definite evidence for these findings or the quality of the involved processes. In the present research, for the first time, many of the probably involved factors are simultaneously studied. The researchers also emphasize the role of ammonia in interaction with pH and lactate, as well as its involvement in phosphate purine cycle and ultimately its role in fatigue. Moreover, in most of the previous research the original tamponic reservoirs of the body has not been identified, which is believed to affect interpreting the results.

## Material and Methods

### Participants

Sixteen 400 meters male runners voluntarily participated in the study (Table 1). The necessary instructions were given to avoid any unusual changes in daily physical activities or food consumption patterns in the days before the test.

Tab. 1. The participants' general characteristics

	Sodium bicarbonate	Placebo
Age (years)	20.58±3.25	20.35±3.65
Height (cm)	175.33±2.48	175.42±2.35
Weight (kg)	64.43±1.34	64.53±1.23
BMI(kg/m <sup>2</sup> )	21.57±2.67	21.61±2.74

### Sodium Bicarbonate Supplement

Sodium bicarbonate supplement (alkalosis) and calcium carbonate placebo in the form of dark hygienic capsules, with the dosage of .3 grams per kilogram [18, 19] of body weight were used one hour before the test. Like Nilson and others (2002), no food interference was introduced, yet the volunteers had not eaten or drunk for 12 hours before the test [10]. The participants were randomly put into two groups, in a way that half of the participants (8 people) took bicarbonate supplement, and the other half took placebo.

### 400 Meters Running Test

After a full warm-up, the participants entered a 400-meter running race in groups of 4 (on a standard running track). The way the participants were put in track lines was randomly decided. In the second session, just the type of the supplement used by the participants was altered, so that each participant took both supplements in two sessions.

### Blood Sampling

There were three blood sampling phases which include, resting condition, 30 min after supplementation and 2 min after 400 m run [10, 20]. Needless to mention, in both sessions the blood samples were poured into lab tubes containing heparin. They were immediately sent to a laboratory for analysis.

### Biochemical Measurements

To measure blood parameters, like Ball and Mogan's method (1997), a blood gas analyzer (A.V.L model) was used through the enzymatic method [18]. To measure blood lactate, a commercial auto analyzer was used.

### Statistical Analysis

Having ensured the natural distribution of all data by the K-S test, the data for the factors of ammonia, sodium bicarbonate, lactate, and blood PH, were compared using a repeated measures test in three phases: at rest (R), 30 minutes after taking the supplement (PS), and 2 minutes after 400 meters run (PE). In case there were any differences, the LSD test was used for further analysis. Moreover, after calculating the difference in the data for each factor at R and PS, as well as PS and PE levels, the amount of these changes between bicarbonate supplement-taking and placebo-taking groups and the time spent to run 400 meters in the two groups were compared using independent t-tests. Statistical significance was accepted at  $P \leq 0.05$ .

## Results

The preliminary results indicated that by using sodium bicarbonate, the time of the running performance was better than when placebo was used ( $p < 0.05$ ) (Fig 1).

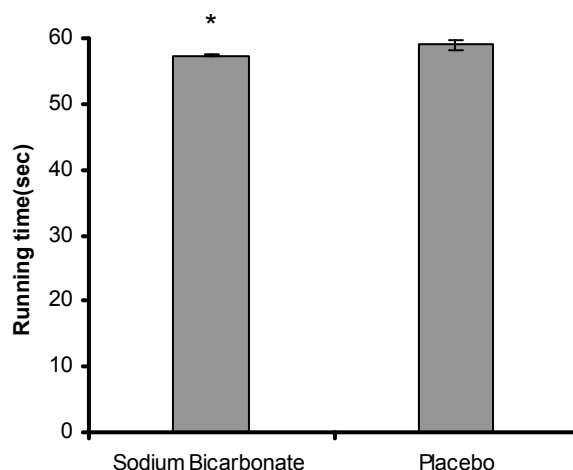


Fig 1. Comparison of running time (sec) of the sodium bicarbonate and placebo groups (mean  $\pm$  SEM)

\* denotes statistical differences in comparison to the placebo group

Also with the use of sodium bicarbonate the plasmid levels of this material significantly increased in comparison to the rest state ( $p < 0.05$ ), yet after running, the values changed to their original amount. Moreover, when placebo was used, the bicarbonate level of blood after running came to lower levels than its rest level ( $p < 0.05$ ). In both cases of taking sodium bicarbonate and placebo, there was a significant increase in the lactate level of blood after running. In addition, the decrease in the blood pH level was significant only at the use of placebo ( $p < 0.05$ ). The results are separately given in Table 2.

Tab. 2. The differences of ammonia, sodium bicarbonate, lactate, and pH in the sodium bicarbonate and placebo groups. Values are mean  $\pm$  SEM

	Rest		After taking supplement (PS)		After 400 meters race (PE)	
	Sodium bicarbonate	Placebo	Sodium bicarbonate	Placebo	Sodium bicarbonate	Placebo
Ammonia (mmol/l)	18.57 $\pm$ 4.42	18.43 $\pm$ 4.46	19.58 $\pm$ 6.94	18.98 $\pm$ 7.26	18.02 $\pm$ 5.50	18.79 $\pm$ 4.36
Sodium bicarbonate (mmol/l)	23.13 $\pm$ 1.84	23.31 $\pm$ 2.15	29.53 $\pm$ 2.64*	23.38 $\pm$ 2.18	20.45 $\pm$ 1.92	17.89 $\pm$ 2.48*
Lactate (mmol/l)	1.25 $\pm$ 0.37	1.31 $\pm$ 0.32	1.3 $\pm$ 0.45	1.27 $\pm$ 0.34	18.28 $\pm$ 2.41*†‡	15.61 $\pm$ 2.35*†
pH	7.40 $\pm$ 0.10	7.41 $\pm$ 0.02	7.49 $\pm$ 0.21	7.42 $\pm$ 0.08	7.20 $\pm$ 0.38	7.10 $\pm$ 0.11

\* Denotes statistical differences in comparison to the rest state ( $p < 0.05$ ).

† Denotes statistical differences in comparison to after taking the supplement ( $p < 0.05$ ).

‡ Denotes statistical differences in comparison to the placebo group ( $p < 0.05$ ).

## Discussion

Sodium bicarbonate supplementation might have a positive effect on short term intensive exercise performance, and it probably decreases recovery time. In both the sodium bicarbonate and the placebo sessions, there were no inter-session differences with regard to the plasma ammonia level. During intensive exercise ammonia can be generated through deamination of adenosine mono-phosphate (AMP)<sup>1</sup> [11] and/or catabolism of some amino acids (long term sub-maximal exercise) e.g. the branched chain of amino acids (BCAAs). However, it is possible for cellular hydrogen peroxide to be promoted following degradation of inosine mono-phosphate into uric acid (action of xanthine oxidize) which may lead to decreased muscular contractility (local fatigue) [21]. Furthermore, exercise-induced blood ammonia elevation is thought to be associated

<sup>1</sup> AMP can be broken down into inosine mono-phosphate and ammonia.

with central fatigue [10] There is some evidence confirming that during progressive exercises, plasma ammonia level increases along with exercise intensity and then it reaches the threshold level similarly to those observed with the lactate [22,10]. This may indicate that plasma ammonia could be considered as a marker for the rate of anaerobic metabolism. Although previous studies have revealed no link between the plasma ammonia level and indices of anaerobic/aerobic capacity, a strong correlation between the plasma lactate and the ammonia levels has been repeatedly reported [23].

While there is no straight evidence in the existing literature with regard to the effect of alkalosis on plasma ammonia, we speculated that blood ammonia response is probably matched with the lactate response. Induced alkalosis in our study could efficiently limit the rate of blood pH decrease; however, it failed with regard to the blood ammonia level. This might be a consequence of short time span in between the end of 400 meters running and blood sampling as well as the difference in the rate of ammonia and hydrogen ions diffusion through muscle membranes to the bloodstream. Moreover, it is also possible that the amount of produced ammonia during 400 meters running is too little for any detectable values to be observed by a gas analyzer (limitation of the study methods). Therefore, well-controlled studies certainly remain to be done in this area.

The consumption of sodium bicarbonate caused a significant increase in the plasma bicarbonate level; however, its values returned to resting levels following to 400 meters running. It is noteworthy that in the placebo session the observed blood bicarbonate levels were even lower than the resting values following 400 meters running.

These findings can be indicative of the ergogenic effect of induced alkalosis on improvement in the anaerobic performance by possible increases in blood buffer capacity. Accordingly, it is expected that the amount of blood pH decrease during intensive exercise and also during the recovery period could be modified by sodium bicarbonate consumption, in accordance with the previous reports in this area [10]. It is well established that inhibition of carbonic anhydrase activity could neither decrease the rate of CO<sub>2</sub> production (VCO<sub>2</sub>) during the resting conditions as well as sub-maximal exercise with the continuous intensity; however, it can occur in the maximal exercise.

It should be noted that during too intensive exercises with continuous blood lactate elevations, the rate of generated CO<sub>2</sub> in the blood is influenced by the combined involvement of non-metabolic sources e.g. blood lactate buffering and hyperventilation. It was shown that during progressive exercise VCO<sub>2</sub> remains unchanged while VE decreases [24].

On the other hand, by the shift of hydrogen ions into red blood cells, the ionic gradient in the plasma facilitates the entrance of anions from muscles toward the bloodstream. Anyway, our unique finding was that bicarbonate consumption prior to exercise limits the rate of plasma bicarbonate decline during intensive exercise, which is thought to facilitate a quicker and easier recovery.

In the other findings, no difference was observed between the rest level lactate after consuming bicarbonate. Two protein types of band-3 and MCT-1 are involved in transferring lactate and making acid-alkaline balance on the two sides of the surface of erythrocytes. They are responsible for transferring 5%, 80%, and 90% of lactates, respectively [25]. Alkalosis causes an increase in the transmission of lactate from active muscle. An increase in the transmission of lactate from muscles to blood, without any change in lactate concentration steepness, has also been reported, which seems to be a result of an increase in ion steepness due to alkalosis presence [11, 26]. As such, considering the fact that rest lactate level is fixed, except for pathologic conditions, no change in rest level lactate seems logical. In addition, at both times when sodium bicarbonate and placebo were used after 400 meters run a significant increase in blood lactate was observed. Meanwhile, no difference was observed for changes in the two sessions of using sodium bicarbonate and placebo (after supplement use until running ended). In other words, alkalosis could not affect the amount of lactate transfer to blood. It is assumed that in the early post-exercise minutes, the flow of H<sup>+</sup> from muscles to blood is quicker than lactate [18]. In the case of human beings, it has been shown that the speed of the flow of hydrogen ion is usually faster than that of lactate [19]. In addition, the absorption of lactate and hydrogen ion by inactive muscles affects the rate of their transfer from active muscles [27]. There are also reports indicating that lactate flow to

extracellular fluids is independent of acid and alkalosis state in intercellular fluid and it has a fixed rate [18]. This may help us justify the existence of no inter-group difference in the blood lactate level after running.

In some other findings, the use of sodium bicarbonate supplement increases rest pH, but it is not significant. Also following 400 meters race, in both sessions of using bicarbonate and the placebo, blood pH decreased, but this decrease was significant only in the placebo use. In other words, the use of sodium bicarbonate supplement could balance the pH decrease after hard non-aerobic exercise. It seems that the introduction of metabolic alkalosis could create only a fragmentary increase in absorbing hydrogen ion in active muscles [18]. It increases rest pH and controls non-aerobic glycolysis, which might initially cause an increased rate of oxide phosphorization [28]. The exit of hydrogen and lactate ions from muscles provides a supportive mechanism against intracellular pH decrease and lactate accumulation. There are reports indicating that carbonic anhydrase enzyme has a vital role in speeding up the mechanism. So far, two isoforms of this enzyme (CAIX, CAIV) have been identified in muscle surface, the active parts of which are outwards pointing to extracellular space. Meanwhile, the rate of proton and lactate production during maximal exercise is far more than intramuscular metabolic and buffer capacity or its emission outside the cell. So pH can decrease down to 6.4 inside the muscle and 6.9 in blood [26]. Therefore, due to the high involvement of glycolysis in generating energy for 400 meters running race, in the present study, pH and lactate changes had no exactly equal patterns. However, it seems that sodium bicarbonate supplement can be effective in adjusting pH and lactate changes in intensive activities.

In the last part of the results of this study, the performance in 400 meters running race in the bicarbonate use session was better than that of the session with the placebo use. There is a lot of evidence, indicating that alkalosis can improve short-term intensive performance [26], but much deterioration has been reported in improving performance resulting from alkalosis [11]. Metabolic alkalosis, with an increase in extracellular buffering capacity, causes the maintenance of higher ionic steepness between muscle and blood, as well as an increase in the flow of lactate and protons from muscle cells. Nevertheless, there is a lot of evidence indicating that in a person with MCT1 and higher carbonic anhydrase more benefits can be obtained by introducing alkalosis [26].

## Conclusion

The most important finding of the present study was the fact that body alkaline reservoir can increase by adding supplements, but during intensive non-aerobic exercise the amount of these reservoirs deteriorates. Moreover, plasma ammonia level is not affected by using alkaline material or doing hard non-aerobic exercise. This can challenge the hypothesis of the effect of ammonia from purine cycle on plasma PH. Furthermore, neither pH nor rest lactate in plasma are affected by an alkaline supplement, but a decrease resulting from activity can be counterbalanced by the use of alkaline material.

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