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Bicarbonate supplementation via lactate efflux improves anaerobic and cognitive performance in elite combat sport athletes

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ABSTRACT: The aim of this study was the assessment of sodium bicarbonate supplementation ($NaHCO_3^-$) on anaerobic and cognitive performance, assuming ergogenic effect of HCO_3 by improving buffering capacity and greater lactate efflux, which may have indirect effect on circulating neurotrophin level (e.g BDNF, IGF-1) and memory. Sixteen well-trained judo athletes completed a randomized trial of either a $NaHCO_3^-$ (EG) (5000 mg x 2/day/90 min before training) or placebo for 21 days (CG). Before and after treatment, athletes completed double Wingate test (Wt) protocol following which they performed perceived Working Memory test (pWM). Results suggested significant increase in Upper Limb Total Work (with $p = 0.011$), Mean Power (with $p = 0.001$), post exercise LA concentration (from 15.51 mmol/L to 18.10 mmol/L with $p = 0.01$) and $HCO3_{rest}$ concentrations (from 27.37 mmol/l to 28.91 mmol/l with $p = 0.001$), when compared to baseline values in EG. The analysis showed statistically significant increase in values for IGF-1 (with $p = 0.001$) and decrease for cortisol and BDNF (with $p = 0.001$) in EG and CG, when pre and post exercise values were compared. We also revealed statistically significant decrease in values for display time after ingestion of HCO_3 between pre and post exercise (with $p = 0.002$) In conclusion, the lack of a substantial relationship between exerkines (IGF-1, BDNF) and memory in the present study might suggest that exercise induced lactate levels is dominant mechanism improving working memory in well-train athletes.

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INTRODUCTION

In open-loop sports such as combat sports, the surrounding environment changes very rapidly during training. Therefore, athletes are required to process a large amount of external information and take appropriate actions in limited time. In the field of neuropsychology, these cognitive abilities are referred to as executive functions (EF) [1, 2]. EFs are a set of cognitive processes that are necessary for the control of behavior [3, 4]. Working memory is strongly related to cognitive flexibility, and linked to creativity, inhibitory control, and focused attention [4]. Higher-order EFs consist of reasoning, problem solving, and planning, which are built based on the core EFs [2]. In sports, EFs relate to cognitive processes that regulate thought and action, especially in non-routine situations. These abilities are called tactical intelligence [3]. Sports tests for assessing of perceptual functions including EFs have often been designed for a specific discipline or study [5, 6].

It has been documented that physical exercise has beneficial effects on cognition [7]. Chronic effects as well as single exercise sessions have been shown to improve a variety of cognitive tasks, including working memory [8, 9]. The characteristics of exercise, especially its intensity and volume may be an important factor determining the potential impact on cognitive functions. The results also seem to depend on the type of memory being tested [10].

The brain-derived neurotrophic factor (BDNF), insulin growth factor one (IGF-1), and cortisol have been considered as possible neuroendocrine mediators of exercise induced memory changes [11, 17]. BDNF is a member of the neurotrophin family of factors that supports neural survival, growth, and synaptic plasticity which is highly concentrated in the hippocampus and cortex [11, 12]. The molecular properties of BDNF allow the assessment in the blood, serum, or plasma as peripheral BDNF (pBDNF), making measurements of acute exercise-induced changes in BDNF concentrations relatively accessible. The efficacy of using peripheral measures is supported by research showing the ability of BDNF to pass through the blood–brain barrier (BBB) [13], and positive correlations between concentrations of central (neural) and pBDNF levels [14].

IGF-I, which is mainly known for its role in energy metabolism and homeostasis, is emerging as a key growth factor involved in modulation of synaptic plasticity, synapse density, neurotransmission,

and even adult neurogenesis. BDNF and IGF-I have been shown to protect cultured hippocampal neurons from serum deprivation-induced cell death using similar downstream pathways [15]. IGF-1 promotes the release of BDNF in the brain, which has been identified as one of the principal factors mediating the effect of exercise on cognitive functions [16].

The relationship between catecholamines levels and cognitive performance during acute exercise has also been explored [17]. For example, evidence indicates that exercise, especially at high intensity can activate the hypothalamic-pituitary-adrenal axis [18]. In addition, memory consolidation can be enhanced by glucocorticoids. However, depending on the timing of the stressor, a stress-related increase in glucocorticoids may also impair memory retrieval and reconsolidation [19]. For these reasons, stress hormones released during exercise may mediate exercise-related memory effects.

The indicated mechanisms of neuroendocrine response to high intensity anaerobic exercise (HIAE) are regulated by lactate shuttle mechanisms. Therefore, lactate concentration kinetics (La) can be a mirror of exercise intensity. Anaerobic glycolysis leads to an equal production of La and hydrogen ions [20]. Most of the released hydrogen ions are buffered; however, a portion (~0.001%) that stays in the cytosol results in a decrease in muscle pH and impairment of exercise. The rationale for the ergogenic effects of bicarbonate is that the increase in extracellular pH and bicarbonate can enhance the efflux of lactate and H^+ from the muscle cell [21]. Buffering of protons can attenuate changes in pH and enhance the muscle's buffering capacity, allowing for a greater amount of lactate to accumulate in the muscle [22]. La as an endogenous metabolite is rapidly produced in response to HIAE. La, released by the muscles into the blood stream, has the ability to cross the BBB via endothelial monocarboxylate transporters (MCTs) [23] and act as an energy source and a neuroprotective factor for the brain [24, 25]. In addition, La is indicated as a stimulus for exerkine release of both BDNF and IGF-1 [26].

Based on this information, we hypothesized that La concentration can serve as a direct exercise mediator to enhance EFs, and indirectly to induce the mechanisms of BDNF and IGF-1 release. Given the limited research on well-trained athletes and the possible contribution of bicarbonate ingestion to the relationship between HIAE and cognitive function, this study was aimed at determining the effects of buffering capacity on lactate efflux, BDNF, IGF-1 serum concentration and memory performance. We further hypothesized that bicarbonate supplementation can induce an ergogenic effect by improving blood and muscle buffering capacity, allowing for a more intense exercise and greater lactate efflux. These events may have an indirect enhancing effect on circulating neurotrophin levels and memory.

MATERIALS AND METHODS

1.1 Participants characteristics

Sixteen well-trained male combat athletes participated in the study. All participants had at least twelve years of training experience, international sports level, and they were members of the Polish National Team The athletes constituted a homogenous group in regard to age (average age of 24.3 ± 0.5 years), somatic characteristics, as well as aerobic and anaerobic performance. A summary of the participants characteristics is given in Table 1. The participants (n = 16) were randomly divided into two groups, an experimental group (EG; n = 8), which received a sodium bicarbonate supplement, and a control group (CG; n = 8), which received a placebo. All participants had valid medical examinations and showed no contraindications to participate in the experiment. The participants were informed verbally and in writing of the experimental protocol, and the possibility to withdraw at any stage of the experiment. The participants were excluded if they reported a history of cognitive deficiencies, or were taking antioxidants, neuroactive or psychoactive drugs, stimulants, or other illegal substances. The study was approved by the Research Ethics Committee at the Academy of Physical Education in Katowice, Poland.

TABLE 1. Characteristics of the study participants.

Variables	Experimental Group (n = 8) Mean ± SD	Control Group (n = 8) Mean ± SD
Age (yrs.)	24.3 ± 0.5	23.2 ± 1.1
Height (cm)	181.0 ± 2.3	178 ± 2.0
Body mass (kg)	81.0 ± 2.4	84.2 ± 3.0
$TW_{-upper\ lumbs}$ (J/kg)	192.10 ± 5.5	195.1 ± 8.4
$TW_{-lower\ limbs}$ (J/kg)	246.0 ± 6.5	248.7 ± 12.4
$MP_{-upper\ limbs}$ (W/kg)	7.22 ± 0.47	7.12 ± 0.63
$MP_{-\ lower\ limbs}$ (W/kg)	9.14 ± 0.87	8.99 ± 0.43
VO_{2max} (ml/kg/min)	59.7 ± 3.2	58.8 ± 2.1

Note: TW– Total Work; MP– Mean Power.

1.2 Experimental design

This study was a single-blind randomized controlled trial. The experiment lasted 42 days, during which three sessions and three series of laboratory evaluations were performed. The tests were carried out during familiarization, at baseline and after three weeks of interventions, which consisted of bicarbonate supplementation or placebo treatment (Fig.1). The study was conducted during the preparatory period of the annual training cycle, when a high volume of work dominated the daily training loads. The participants refrained from exercise for two days before testing to minimize the effects of fatigue.

The assessment of selected neurocognitive functions in response to HIAE is presented in Figure 2.

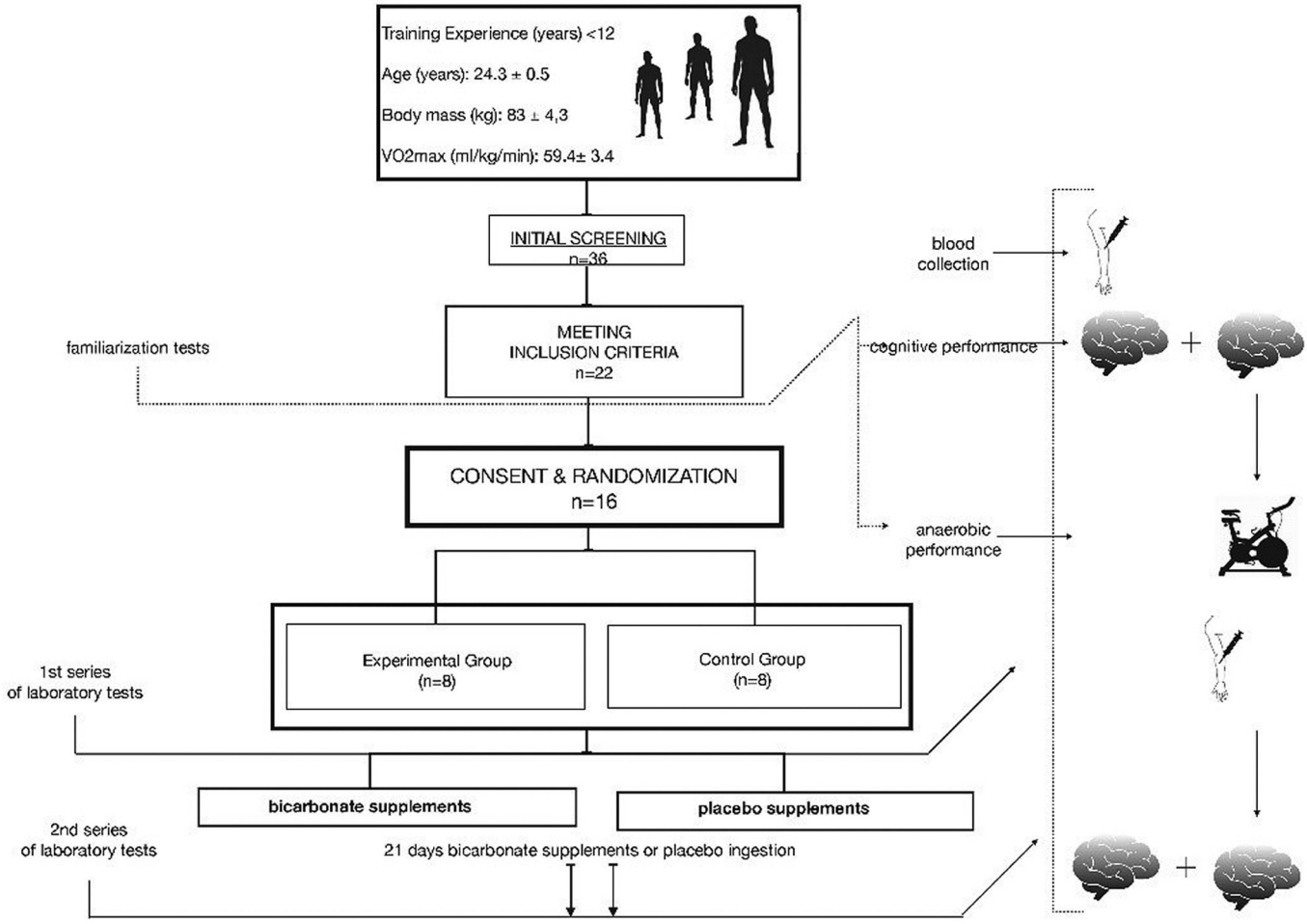

FIG. 1. Overview of the experimental protocol.

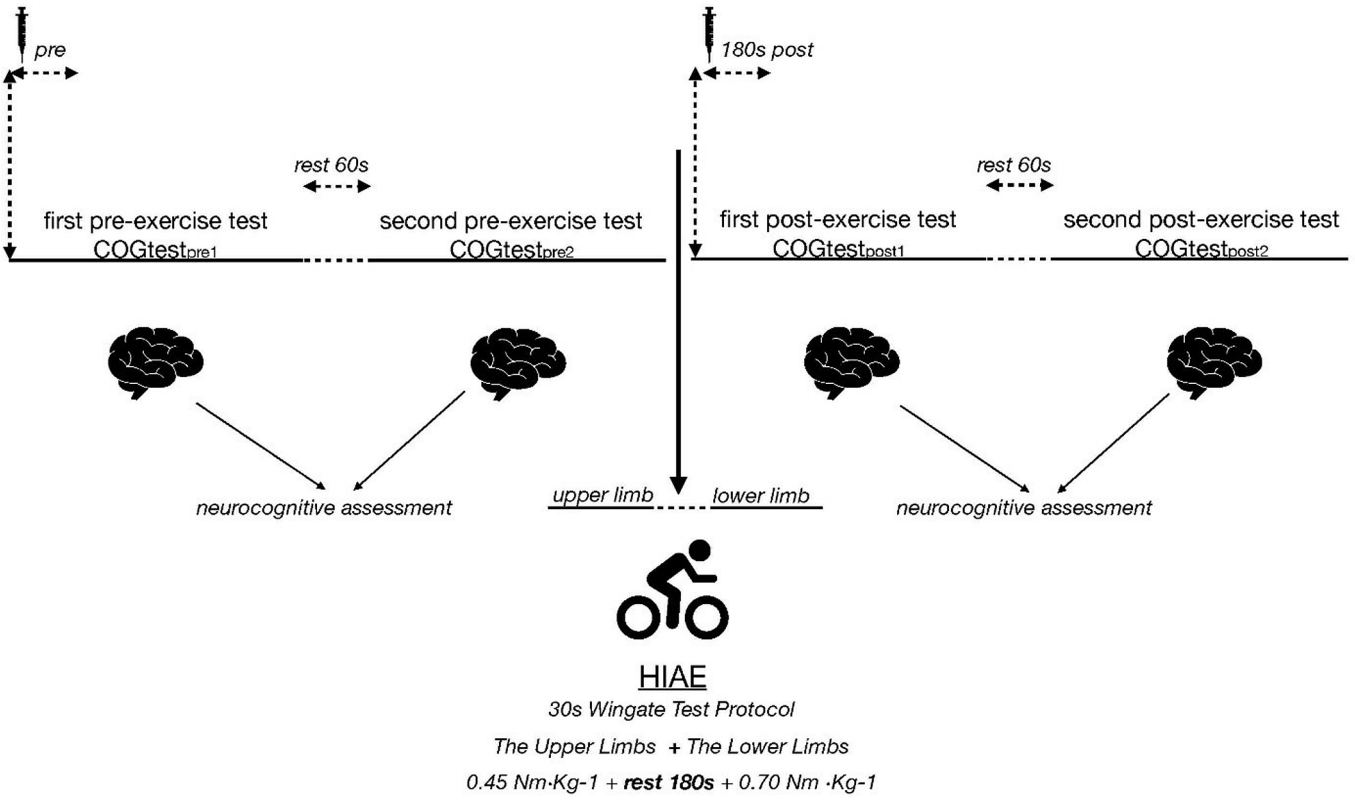

FIG. 2. Flowchart.

1.2.1 Diet and supplementation protocol

The supplementation protocol used in our previous study [22] was used as a reference. The participants of the experimental group ingested a single dose of 5 g of sodium bicarbonate, twice a day, 90 min before each practice session [11]. The control group ingested identical capsules containing corn starch. Supplements were ingested with water (600 mL). The supplementation protocol included an additional 5 g doses of bicarbonates 90 min before the exercise test protocol and the day before the test. The dose of bicarbonate was chosen according to the literature recommendations, where amounts ranging from 5 to 9 g /day are suggested. Such doses have shown significant improvements in buffering capacity with no gastrointestinal distress [49].

Energy intake, as well as macro and micronutrient intake of all subjects was determined by the 24 h nutrition recall 3 weeks before the study commencement. The participants were placed on an isocaloric (3455 ± 436 kcal/d) mixed diet (55% carbohydrates, 20% protein, 25% fat) prior to, and during the investigation. The pre-trial meals were standardized for energy intake (600 kcal) and consisted of carbohydrate (70%), fat (20%) and protein (10%).

1.3 Biochemical assays

1.3.1 Lactate, acid-base balance, ions

To determine lactate concentration (LA), acid-base equilibrium and electrolyte status, the following variables were evaluated: LA (mmol/L), blood pH, pCO_2 (mmHg), pO_2 (mmHg), $HCO_3^-{}_{act}$ (mmol/L), $HCO_3^-{}_{std}$, (mmol/L), BE(mmol/L), O_{2SAT} (mmol/L), $ctCO_2$ (mmol/L), Na^+ (mmol/L), K^+ (mmol/L) and Ca^{2+} (mmol/L). The measurements were performed on fingertip capillary blood samples at rest, and after 3 minutes of post exercise recovery. Determination of LA was based on an enzymatic method (Biosen C-line Clinic, EKF-diagnostic GmbH, Barleben, Germany). The remaining variables were measured using a Blood Gas Analyzer GEM 3500 (GEM Premier 3500, Germany).

1.3.2 IGF-1, noradrenaline, cortisol, and BDNF

During the experiment, at each of the two time points, venous blood samples were collected to determine IGF-1, cortisol and noradrenaline. All hormone concentrations were evaluated in blood serum. Noradrenaline was measured once by the high-performance liquid chromatography method, cortisol and IGF-1 were measured in duplicate using EDTA plasma and immunoassay kits.

For BDNF analysis, 4.5 mL of venous blood from the antecubital vein was collected with clotted blood tubes at six time points. The first blood sample was taken at the beginning of the experimental session, while the second before the cognition task. The second blood sample was collected after the cessation of the exercise protocol. BDNF concentrations were measured in serum samples with an ELISA kit (R&D systems, cat no. DBD00) according to manufacturer instructions. Blood samples were incubated at room temperature to clot for 30 minutes and then centrifuged for 20 min at 1300 x g. Serum was aliquoted and stored at -80 °C until in use. The assay was performed in duplicate for each sample. The optical density was determined using a microplate reader (Biotek, Synergy LCX) set to 450 nm with a wavelength correction set to 540 nm. A standard curve was created by generating a four-parameter logistic (4-PL) curve-fit with GraphPad Prism 8.4.1.

1.4 Anaerobic and cognitive performance

1.3.3 Anaerobic performance

Anaerobic performance was evaluated by two 30-s Wingate tests for lower and upper limbs respectively, with a passive rest interval of 3 minutes between the bouts of exercise. The test was preceded by a 5 min warm-up with a resistance of 100 W and cadence within 70–80 rpm for lower limbs and 40 W and 50–60 rpm for the upper limbs. Following the warm-up, the test began, in which the objective was to reach the highest cadence in the shortest possible time, and to maintain it throughout the trial. The lower limb Wingate protocol was performed on an Excalibur Sport ergocycle with a resistance of 0.8 Nm·Kg-1 (Lode BV, Groningen, Netherland). The upper body Wingate test was carried out on a rotator with a load of 0.45 Nm·Kg-1 (Brachumera Sport, Lode, Netherland). Each subject completed the test trials with incomplete rest intervals. The variables of peak power – P_{max} (W/Kg), mean power – P_{mean} (W/Kg), total work performed – W_t (J/Kg), rate of fatigue (%) and fatigue slope were registered and calculated by the Lode Ergometry Manager (LEM, software package, Netherland) (Fig. 2).

1.3.4 Working Memory

Cognitive performance was assessed using the dedicated neurocognitive assessment system (Microgate, Witty, cognitive tests Brain HQ, Italy). The objective of the test was to identify the location of the shapes of the same color. In the "Eye for Detail" protocol, a series of 3 to 5 shapes briefly appeared, one at a time in different positions (supplementary data). Of the shapes, some matched precisely, while others were similar but not the same. The challenge was to identify where identical shapes appeared. As the exercise's difficulty increased, the signals flashed by more quickly, according to BrainHQ^R mathematical algorithms [50].

In the present study we called this task a test of perceived Working Memory (pWM). We selected this test because no cognitive manipulation is needed in order to solve it. It can be repeated several times and it can be applied to a chosen exercise protocol. Moreover, the test is more demanding than the back memory-test and better mirrors the cognitive challenge during sport-competition, where a constant flood of information and events from different time points must be processed.

1.4 Statistical analysis

The Shapiro–Wilk, Levene and Mauchly's tests were used to verify the normality, homogeneity and sphericity of the sample's data variances, respectively. Verifications of the differences between the

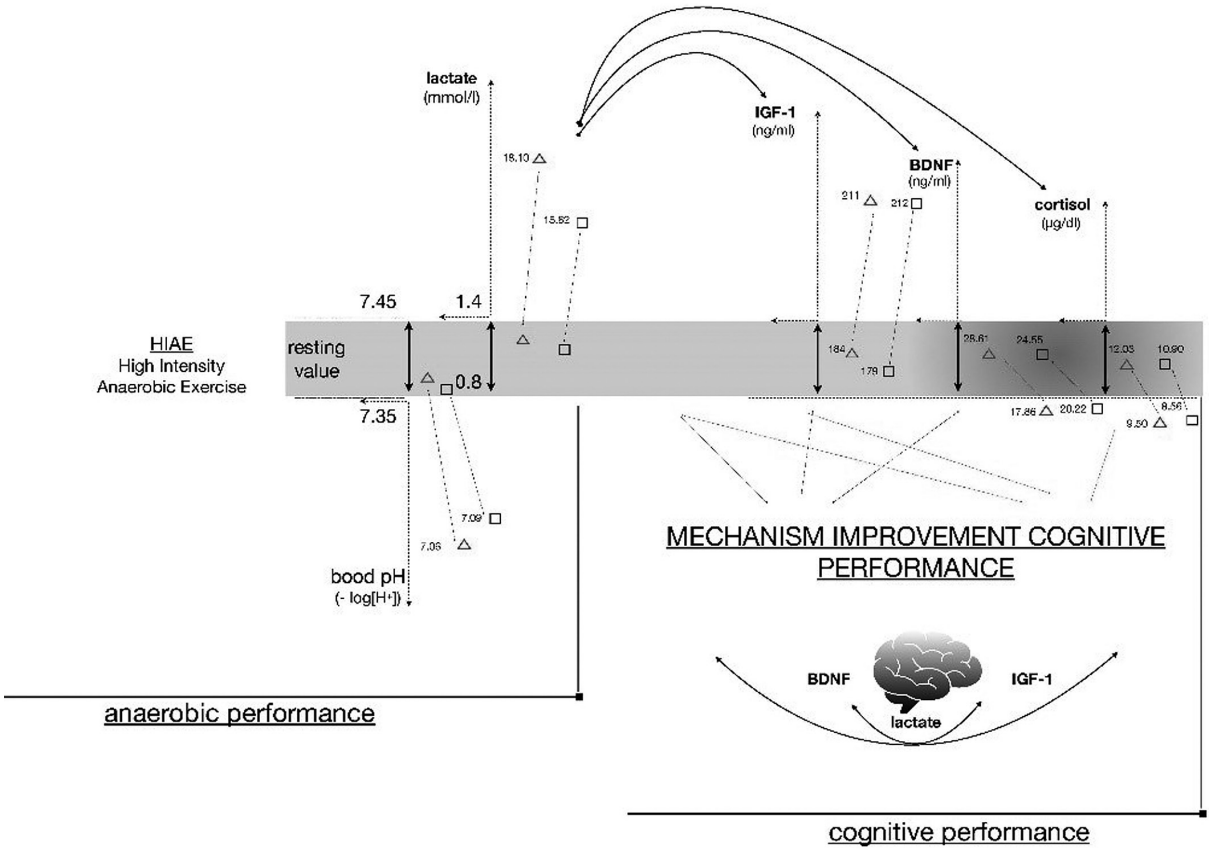

FIG. 3. The proposed exercise mechanism, regulating the improvement of brain executive functions in athletes. Control group – ☐, Experimental Group – △

considered values before and after bicarbonate supplementation, between baseline conditions and after high intensity anaerobic exercise in the EG and CG groups were verified using multivariate ANOVA with repeated measures. Effect sizes (Cohen's d) were reported where appropriate [51]. According to Hopkins guidelines [52], the effect size (eta-squared; η^2) was established as follows: 0.01 – small, 0.06 – medium, and 0.14 – large. Statistical significance was set at $p < 0.05$. All statistical analyses were performed using Statistica 9.1 (TIBCO Software Inc., Palo Alto, California, CA, USA) and Microsoft Office (Redmont, Washington, DC, USA), and are presented as means with standard deviations.

RESULTS

All participants completed the described testing protocol. The ANOVA analysis revealed significant differences between the baseline and post-intervention period – 21 days ingestion of bicarbonate supplements, in the following variables:

Anaerobic performance lactate and acid-base balance parameters. Post-hocks tests revealed a statistically significant increase in Upper Limb Total Work (with $p = 0.011$), Mean Power (with $p = 0.001$) and PP (with $p = 0.013$) in EG with bicarbonate intervention, when baseline and post intervention values were compared. In contrast, the control groups, which received placebo supplements, did not reveal any statistically significant results (Table 2). There were no statistically significant differences in lower limbs when baseline and post intervention values were compared in EG and CG.

A significant increase in post exercise LA concentration (from 15.51 mmol/L to 18.10 mmol/L with $p = 0,001$) and $HCO3_{rest}$ concentrations (from 27.37 mmol/l to 28.91 mmol/l with $p = 0,001$) were observed (Table 3).

Hormones and BDNF

The analysis showed a statistically significant increase in values for IGF-1 (with $p = 0.001$) and decrease for cortisol (with $p = 0.001$) when pre and post exercise values were compared for EG and CG. Additionally, we determined significant decrease in values for BDNF, when pre and post exercise values were compared after 21 days bicarbonate ingestion (Table 4).

Working memory

We observed a statistically significant decrease in values for display time before supplementation of bicarbonate between pre and post exercise (with $p = 0.002$ and $\eta^2 = 0,074$) and medium effect size

TABLE 2. The differences in anaerobic performance variables – upper limbs, before and after bicarbonate ingestion in the experimental and control groups (placebo).

Variables		EG	$CG_{control}$
		Mean ± SD	Mean ± SD
TW/kg (J/kg)	Before	195.55 ± 6.50	190.40 ± 13.42
	After	210.01 ± 4.11*	187.40 ± 7.98
MP/kg (W/kg)	Before	7.32 ± 0.67.	7.26 ± 0.63
	After	8.40 ± 1.11*	7.44 ± 1.22
PP/kg (W/kg)	Before	12.73 ± 2.45	12.2 ± 2.96
	After	14.11 ± 1.93*	12.6 ± 3.21

Note: MP – Mean Power; PP – Peak Power; TW – Total Work**;** *- statistically significant difference. ; # statistically

TABLE 3. The differences in POST exercise blood plasma lactate concentration, as well as the resting concentration of di-carbonate and blood pH values before and after bicarbonate ingestion in the experimental and control groups (placebo).

Variables		EG	$CG_{control}$
		Mean ± SD	Mean ± SD
LA_{max} (mmol/l)	Before	15.57 ± 1.06	15.21 ± .44
	After	18.10 ± 1.04*	15.62 ± .94
HCO_3^- rest (mmol/l)	Before	27.37 ± 0.07	27.20 ± .07
	After	28.91 ± 0.09*	27.22 ± 0.05
pH (- log[H^+])	Before	7.43 ± 0.003	7.43 ± 0.01
	After	7.44 ± 0.005	7.43 ± 0.008

Note: LA_{max} – post exercise blood plasma lactate concentration; HCO_3 – di-carbonate**;** *- statistically significant difference. ; # statistically

TABLE 4. The differences in PRE- and POST- exercise BDNF concentrations before and after bicarbonate supplementation in the experimental and control groups.

Variables			EG	CG
			Mean ± SD	Mean ± SD
BDNF (ng/ml)	Before	$PRE_{exercise}$	26,55 ± 7.84	26.31 ± 7.22
		$POST_{exercise}$	24.81 ± 10.08	23.73 ± 12.45
	After	$PRE_{exercise}$	28.62 ± 11.14	28.20 ± 13.30
		$POST_{exercise}$	14.86 ± 7.83*	20.20 ± 10.09

Note: *- statistically significant difference. ; # statistically

TABLE 5. The differences in neurocognitive functions PRE and POST- exercise, as well as before and after bicarbonate supplementation in the experimental and control groups.

Variables			EG	CG
			Mean ± SD	Mean ± SD
Display Time (ms)	Before	$PRE_{exercise1}$	0.376 ± 0.101	0.392 ± 0.102
		$PRE_{exercise2}$	0.357 ± 0.091#	0.362 ± 0.353#
		$POST_{exercise1}$	0.310 ± 0.11	0.312 ± 0.21
		$POST_{exercise2}$	0.233 ± 0.05#	0.250 ± 0.09#
	After	$PRE_{exercise1}$	0.396 ± 0.10	0.392 ± 0.103
		$PRE_{exercise1}$	0.367 ± 0.10$	0.371 ± 0.366$
		$POST_{exercise1}$	0.276 ± 0.11	0.318 ± 0.10
		$POST_{exercise2}$	0.217 ± 0.09$	0.250 ± 0.10$

Note: *- statistically significant difference. ; # Pre – post before;. $; # Pre – post after

in EG. The same analysis revealed significant differences for display time between pre and post exercise (with $p = 0.001$ and $\eta^2 = 0{,}1524$) and large effect size when the values after bicarbonate supplementation are compared in EG (Table 5).

In CG, ANOVA analysis detected a statistically significant decrease in values for display time before ingestion of bicarbonate between pre and post exercise with $p = 0.002$ and small effect size ($\eta^2 = 0{,}010$) as well as significant differences for display time between pre and post exercise when the values after bicarbonate supplementation were compared with $p = 0.001$ and medium effect size $\eta^2 = 0{,}072$).

DISCUSSION

The aim of the present study was to assess the effects of HIAE on perceived working memory and the release of La, BDNF, cortisol and IGF-1, before and after three weeks of sodium bicarbonates ingestion. In addition, the main findings of this study was made to indicate a potential mechanism affecting the change of working memory assessment (Fig. 3).

Anaerobic performance and lactate metabolism

The current investigation demonstrated a significant improvement in anaerobic performance of athletes in the experimental group

supplemented with sodium bicarbonate. The ergogenic effect of sodium bicarbonate on exercise performance stems from the reinforced extracellular bicarbonate buffer capacity to regulate acid-base balance during high intensity exercise [22,27]. The elevated bicarbonate enlarges the gradient between extracellular and intracellular H^+, which stimulates the lactate/H^+ cotransporter [28]. The rationale for the ergogenic effects of bicarbonate is that the increase in extracellular pH and bicarbonate can enhance the efflux of lactate and H^+ from the muscle cell. Buffering of protons can attenuate changes in pH and enhance the muscle's buffering capacity, allowing for a greater amount of lactate to accumulate in the muscle cell [22]. It was a key mechanism for the methodology we selected. As expected, the results of the current study demonstrated a significant increase in resting HCO_3^- concentration (from 27.37 to 28.91 mmol/L) and post exercise lactate concentration (from 15.57 to 18.10 mmol/L) in the experimental group supplemented with bicarbonate. This made it possible to compare post-exercise changes in cBDNF, IGF-1 and cortisol levels to the scores of working memory tests and in relation to improved buffer capacity and increased lactate efflux.

Lactate is the preferred metabolite over glucose in the brain, and most importantly, in humans in vivo [36,37]. It is defined as "lactormone", which functions as a signalling molecule with autocrine, paracrine and endocrine-like effects [38]. According to the lactate shuttle hypothesis the linkage between driver cells of lactate formation and recipient cells of lactate use or signalling can transcend compartment barriers [39,40], including the BBB. This process can also be bidirectional from astrocytes and neurons [42,43]. The Astrocyte-Neuron Lactate Shuttle (ANLS) establishes that lactate is extracted by astrocytes and then actively consumed and oxidized by neurons involved in glutamatergic signalling [42,44]. Relevant to lactate shuttling in the brain [45], neurons possess the cellular components necessary for glucose uptake and lactate production as well as direct arterial lactate uptake and use by the Intracellular Lactate Shuttle. Shuttle mechanisms are driven by concentration or pH gradients, or by redox state. However, numerous body compartments and systems such as the interstitial space, vasculature and circulation contribute to lactate shuttling in vivo [41]. The brain relies mainly on glucose metabolism at rest, however the cerebral consumption of glucose decreases during or after high-intensity exercise, along with an increase in blood lactate of consequence for the cerebral uptake [46]. There are numerous scientific data which indicate that HIAE may facilitate neuronal activation and excitation levels to an extent that cognitive performance is improved [47, 48].

In the present study, there was a positive correlation between post-exercise lactate increase and working memory functions when analyzing data of all participants. However there appears to exist an ergogenic effect in response to sodium bicarbonate and increased lactate concentration which may explain the large effect size in the experimental group. The higher the post-exercise lactate concentration, the better the working memory test scores.

BDNF and cognitive performance

In our study, serum cBDNF was significantly decreased after HIAE. This is in contrast to most previous studies reporting an increase in cBDNF after acute response for high-intensity exercise protocols [29,30]. As acute exercise in humans leads to a transient increase in cBDNF, it might be speculated that this exerkine mediates the beneficial effects of acute exercise on memory processes. Changes in cognitive tasks like executive functions or attention [31,32] were found to be unrelated to changes in BDNF. Winter et al. [33] found a positive correlation between the increase in BDNF after exercise and short-term memory task, but no relationship to long-term memory. In each case it is disputed whether an increase in central BDNF is accompanied by an increase in serum BDNF. While some results provide evidence for a strong correlation between cortical and serum BDNF, other data speak against such a relationship [34,35]. The results of our experiment are in line with many other well controlled research projects, which have used high intensity exercise protocols to enhance neurocognitive performance. However, there are several novelties to our study, which should be addressed. First, we conducted research on well-trained, elite combat sports athletes. The results of our research seem to be beyond this discussion, marginalizing the role of BDNF and pointing to alternative dominant mechanisms of exercise-induced improvement of brain executive functions.

CONCLUSIONS

In conclusion, the present study supports the possibility that high intensity anaerobic exercise decreased the release of BDNF and cortisol in elite combat sports athletes. In contrast to most previous scientific data assessing the effects of acute exercise on memory, the athletes in the present study used sodium bicarbonate supplementation to improve their buffering capacity, and we demonstrated the potential role of lactate in this mechanism. The lack of a substantial relationship between exerkines (IGF-1, BDNF) and memory in the present study may suggest that exercise induced alterations in cerebral blood flow and lactate concentration constitute the dominant mechanism enhancing working memory in highly trained athletes.

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