NRC Research Press

ARTICLE

Sodium bicarbonate supplementation improved MAOD but is not correlated with 200- and 400-m running performances: a double-blind, crossover, and placebo-controlled study

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Abstract: The aim of the study was to investigate the effects of acute supplementation of sodium bicarbonate ($NaHCO_3$) on maximal accumulated oxygen deficit (MAOD) determined by a single supramaximal effort ($MAOD_{ALT}$) in running and the correlation with 200- and 400-m running performances. Fifteen healthy men (age, 23 ± 4 years; maximal oxygen uptake, 50.6 ± 6.1 mL·kg^{-1}·min^{-1}) underwent a maximal incremental exercise test and 2 supramaximal efforts at 110% of the intensity associated with maximal oxygen uptake, which was carried out after ingesting either 0.3 g·kg^{-1} body weight $NaHCO_3$ or a placebo (dextrose) and completing 200- and 400-m performance tests. The study design was double-blind, crossover, and placebo-controlled. Significant differences were found between the $NaHCO_3$ and placebo conditions for $MAOD_{ALT}$ (p = 0.01) and the qualitative inference for substantial changes showed a very likely positive effect (98%). The lactic anaerobic contribution in the $NaHCO_3$ ingestion condition was significantly higher (p < 0.01) and showed a very likely positive effect (99% chance), similar to that verified for peak blood lactate concentration (p < 0.01). No difference was found for time until exhaustion (p = 0.19) or alactic anaerobic contribution (p = 0.81). No significant correlations were observed between $MAOD_{ALT}$ and 200- and 400-m running performance tests. Therefore, we can conclude that both $MAOD_{ALT}$ and the anaerobic lactic metabolism are modified after acute $NaHCO_3$ ingestion, but it is not correlated with running performance.

Key words: anaerobic capacity, blood lactate, ergogenic aid, sodium bicarbonate, supramaximal efforts.

Résumé : Cette étude se propose d'examiner les effets d'une supplémentation ponctuelle en bicarbonate de sodium ($NaHCO_3$) sur le déficit maximal d'oxygène accumulé (« MAOD ») déterminé au cours d'un seul effort supramaximal (« $MAOD_{ALT}$ ») à la course et d'évaluer sa corrélation avec la performance à la course sur 200 m et 400 m. Quinze hommes en bonne santé (âge : 23 ± 4 ans; consommation maximale d'oxygène : 50,6 ± 6,1 mL·kg^{-1}·min^{-1}) participent à un test d'effort progressif jusqu'au maximum, à deux efforts supramaximaux équivalant à 110 % de la consommation maximale d'oxygène après avoir consommé soit 0,3 g de $NaHCO_3$ par kg de masse corporelle, soit un placebo (dextrose) et à des tests de performance sur 200 m et 400 m. Le devis expérimental est à double insu, croisé et avec groupe placebo de contrôle. On note une différence significative de $MAOD_{ALT}$ (p = 0,01) entre les deux conditions ($NaHCO_3$ et placebo) et l'inférence qualitative de modifications substantielles révèle fort probablement un effet positif (98 %). La contribution anaérobie lactique dans la condition $NaHCO_3$ est significativement plus grande (p < 0,01) et révèle fort probablement un effet positif (99% de chance), semblable aux observations relatives à la concentration sanguine de pointe du lactate (p < 0,01). On n'observe aucune différence du temps jusqu'à épuisement (p = 0,19) et de la contribution anaérobie alactique (p = 0,81). On n'observe aucune corrélation significative entre $MAOD_{ALT}$ et la performance à la course sur 200 m et 400 m. En conclusion, l'apport ponctuel de $NaHCO_3$ modifie le $MAOD_{ALT}$ et le métabolisme anaérobie lactique, mais n'est pas corrélé à la performance à la course. [Traduit par la Rédaction]

Mots-clés : capacité anaérobie, lactate sanguin, facteur ergogène, bicarbonate de sodium, efforts supramaximaux.

Introduction

The maximal accumulated oxygen deficit (MAOD) is currently the most accepted procedure for estimating anaerobic capacity (Noordhof et al. 2013). The classical model of MAOD represents the difference between the area of predicted oxygen demand and oxygen uptake accumulation measured during an exhaustive supramaximal test (Medbo et al. 1988; Bertuzzi et al. 2010). However, the accurate estimation of oxygen demand corresponding to a supramaximal effort requires the application of at least 10 submaximal trials (i.e., intensities of 30%–90% of maximal oxygen uptake ($\dot{V}O_{2max}$)) (Noordhof et al. 2010), requiring a large time expenditure that is unfeasible for scientists and sports technicians (Bertuzzi et al. 2010; Noordhof et al. 2010).

Based on these limitations, Bertuzzi and co-authors (2010) proposed a method to estimate the MAOD using only a single supramaximal exhaustive test ($MAOD_{ALT}$) that is based on the sum of the alactic and lactic anaerobic energy system contribution. According to the findings of Di Prampero and colleagues (di Prampero 1981; di Prampero and Ferretti 1999), the contributions of the alactic anaerobic metabolism are estimated by the fast component of excess

Received 27 January 2015. Accepted 8 May 2015.

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Table 1. Performance and energy metabolism contribution in the supramaximal efforts in the placebo and $NaHCO_3$ conditions (n = 15).

	Placebo	$NaHCO_3$	Δ%	Coefficient of correlation (r)
t_{lim} (s)	163.0±39.8 (140.9–185.1)	171.7±40.5 (149.2–194.1)	5.3	0.81†
$MAOD_{ALT}$ (L)	3.7±0.5 (3.4–4.0)	4.0±0.6 (3.6–4.3)*	8.1	0.73†
$MAOD_{ALT}$ (mL·kg^{-1})	51.5±5.7 (48.3–54.7)	55.9±8.2 (51.3–60.4)*	8.4	0.69†
$MAOD_{ALT}$ (mL·kg^{-1} lean mass)	70.5±7.1 (66.6–74.5)	76.3±8.9 (71.4–81.3)*	8.2	0.53†
AER (L)	7.2±2.2 (6.0–8.4)	7.6±2.3 (6.4–8.9)	6.1	0.78†
AER (mL·kg^{-1})	101.5±32.2 (83.7–199.3)	107.8±33.3 (89.3–126.3)	6.2	0.75†
AER (mL·kg^{-1} lean mass)	138.3±41.8 (115.1–161.4)	146.4±40.4 (124.0–168.8)	5.9	0.72†
$ANAER_{[La]}$ (L)	2.1±0.4 (1.9–2.3)	2.4±0.6 (2.1–2.7)*	14.9	0.71†
$ANAER_{[La]}$ (mL·kg^{-1})	29.8±5.3 (26.9–32.7)	34.4±7.9 (30.0–38.7)*	15.3	0.70†
$ANAER_{[La]}$ (mL·kg^{-1} lean mass)	40.9±7.1 (36.9–44.8)	46.9±10.0 (41.4–52.4)*	14.8	0.67†
$ANAER_{[PCR]}$ (L)	1.5±0.4 (1.3–1.8)	1.5±0.3 (1.4–1.7)	–1.3	0.61†
$ANAER_{[PCR]}$ (mL·kg^{-1})	21.7±4.5 (19.2–24.2)	21.5±4.3 (19.1–23.9)	–1.0	0.53†
$ANAER_{[PCR]}$ (mL·kg^{-1} lean mass)	29.7±5.7 (26.5–32.8)	29.4±5.4 (26.4–32.4)	–0.9	0.44
A_1 (L·min^{-1})	1.4±0.2 (1.3–1.6)	1.4±0.2 (1.3–1.6)	–0.7	0.92†
τ_1 (s)	63.8±10.4 (58.1–69.6)	63.9±11.8 (57.3–70.4)	0.1	0.31

Note: Values are means ± SD (95% confidence interval). t_{lim}, time to exhaustion; $MAOD_{ALT}$, estimated maximal accumulated oxygen deficit; AER, net aerobic contribution; $ANAER_{[La]}$, lactic anaerobic contribution; $ANAER_{[PCR]}$, alactic anaerobic contribution; A_1, fast component amplitude; τ_1, fast component time constant.

*Significant difference to placebo condition ($p < 0.05$).

†Significant correlation ($p < 0.05$).

postexercise oxygen consumption ($EPOC_{FAST}$), while the lactic anaerobic metabolism is estimated by delta lactate concentration, assuming an energetic equivalent of oxygen of 3 mL·kg^{-1} per each 1 mmol·L^{-1} of delta lactate (Margaria et al. 1933; di Prampero and Ferretti 1999). Therefore, considering that this $MAOD_{ALT}$ determination is basically dependent on the blood lactate concentration and the behavior of EPOC (i.e., oxygen uptake amplitude and time constant), any intervention that alters these variables could compromise the reproducibility of the test, such as caffeine, which modifies the EPOC (Astorino et al. 2011), or sodium bicarbonate, which can improve lactate production (Cameron et al. 2010).

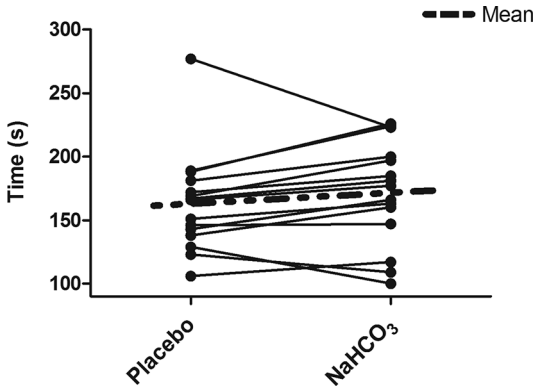
Sodium bicarbonate ($NaHCO_3$) supplementation has been used as a nutritional strategy capable of improving performance (Iwaoka et al. 1989; Van Montfoort et al. 2004; Higgins et al. 2013; Mueller et al. 2013), delaying fatigue during high-intensity exercise ($NaHCO_3$) (de Salles Painelli et al. 2013), increasing the buffering capacity and the extracellular efflux of hydrogen ions (H^+) from the muscle, keeping the muscle pH levels closer to normal during high-intensity exercise (Bishop 2010).

In addition to the proven ergogenic effect (Carr et al. 2011), $NaHCO_3$ seems to be responsible for raising the values of blood lactate concentration postexercise (Cameron et al. 2010; Carr et al. 2012, 2013; Kupcis et al. 2012; Price and Cripps 2012; Siegler et al. 2013; Hobson et al. 2014) and leads to a reduction in the amplitude of the slow component of oxygen uptake ($\dot{V}O_2$) and a slower constant time during the rapid component phase (Kolkhorst et al. 2004). In this way, using this alkalotic buffer may influence $MAOD_{ALT}$ values.

Furthermore, although the accumulated oxygen deficit has been correlated with short-distance running performance (Ramsbottom et al. 1994), there is no evidence regarding the association between $MAOD_{ALT}$ and running performance at 200 and 400 m, which have a predominance of the anaerobic energy systems (Spencer and Gastin 2001).

Therefore, the aim of the present study was to investigate the effects of acute supplementation of $NaHCO_3$ on $MAOD_{ALT}$ determination, comparing the absolute (L), relative to total body mass (mL·kg^{-1}), and relative to lean mass (mL·kg^{-1} lean mass) values, as well as possible associations of $MAOD_{ALT}$ with 200- and 400-m running performance tests. Based on findings that acute supplementation of $NaHCO_3$ can improve lactate concentration (Cameron et al. 2010) and the time to exhaustion (t_{lim}) (Iwaoka et al. 1989), it was expected that acute supplementation with $NaHCO_3$ would change the values of $MAOD_{ALT}$ and t_{lim}.

Fig. 1. Effect of sodium bicarbonate ($NaHCO_3$) and placebo ingestion on time to exhaustion in supramaximal efforts.

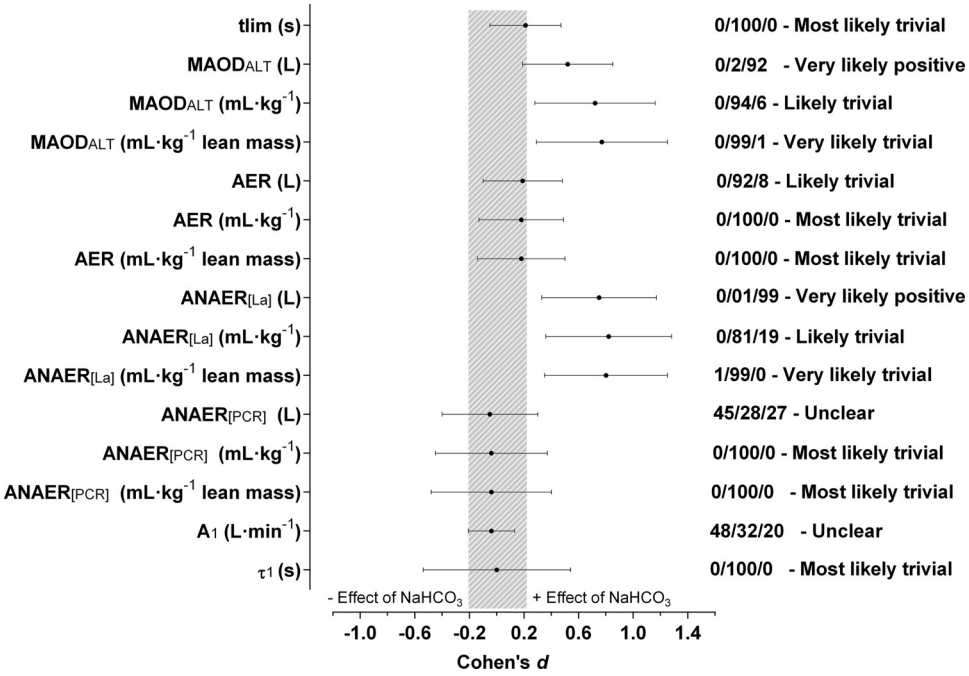


Materials and methods

Subjects

Fifteen healthy and moderately active men participated in the present study (mean ± SD; age, 23 ± 4 years; total body mass, 71.4 ± 9.6 kg; body fat, 21.5% ± 4.4%; height, 174.9% ± 6.5 cm; $\dot{V}O_{2max}$, 50.6 ± 6.1 mL·kg^{-1}·min^{-1}). The subjects were instructed to avoid alcohol and caffeine during the evaluation period and not to perform strenuous exercise for at least 24 h prior to each session. Furthermore, the participants were screened to ensure that they were not currently undertaking or had not undertaken a nutritional regime involving supplements such as $NaHCO_3$, β-alanine, creatine, or thermogenic supplements within the previous 3 months. The subjects were informed about the possible risks and benefits of the study prior to signing an informed consent, and all procedures were conducted respecting the declaration of Helsinki. The experimental procedures used in the study as well as the informed consent were approved by the Research Ethics Committee of the University (Protocol number 900.421/2014).

Fig. 2. Effect of sodium bicarbonate ($NaHCO_3$) ingestion on time to exhaustion (t_{lim}), estimated maximal accumulated oxygen deficit ($MAOD_{ALT}$) and energy metabolism contributions. A_1, amplitude fast-component; AER, net aerobic energy; $ANAER_{[La]}$, lactic anaerobic metabolism; $ANAER_{[PCR]}$, anaerobic alactic metabolism; δ_1, time delay fast-component.



Experimental design

The study design was double-blind, crossover, and placebo-controlled.

Prior to the tests, the body composition of the participants was assessed by means of a whole-body dual-energy X-ray absorptiometry scan (Hologic QDR, Discovery, Bedford, USA). The subjects underwent a maximal incremental exercise test (GXT) (Zagatto et al. 2015) to determine $\dot{V}O_{2max}$ and the intensity associated with $\dot{V}O_{2max}$ ($i\dot{V}O_{2max}$); 2 efforts with a supramaximal intensity corresponding to 110% $i\dot{V}O_{2max}$ for the determination of $MAOD_{ALT}$ with ingestion of $NaHCO_3$ or a placebo, performed in a random order; and 200- and 400-m maximal running tests to evaluate performance.

The GXT and supramaximal efforts were performed on a treadmill (ATL, Inbramed, Inbrasport, Porto Alegre, RS, Brazil) 5 min after a 5-min warm-up at an intensity of 7 km·h^{-1}, with a fixed treadmill incline of 1%, in a laboratory with controlled conditions (43.8% ± 6.3% relative humidity and 22.9 ± 1.3 °C temperature). The speed and slope of the treadmill had been previously calibrated according to the standard measurement reliability suggested by Padulo et al. (2014). In all supramaximal efforts, each participant wore a safety belt attached to his chest to ensure maximal effort. Performance in the 200- and 400-m running tests was estimated on a 400-m running track. All sessions were separated by a minimum interval of 48 h and in all tests the subjects were verbally encouraged.

During the GXT and the 2 supramaximal efforts, $\dot{V}O_2$, carbon dioxide production ($\dot{V}CO_2$), and pulmonary ventilation ($\dot{V}E$) responses were measured breath-by-breath using a stationary gas analyzer (Quark PFT, COSMED, Rome, Italy). The gas analyzer was calibrated using a sample of known gases (3.98% CO_2 and 16.02% O_2) and the spirometer with a 3-L syringe (Hans Rudolf, Kansas City, Miss., USA), according to the manufacturer's recommendations. For analysis of respiratory variables, the data were smoothed every 5 points and interpolated every 1 s to eliminate outlying data (Ozyener et al. 2001). Heart rate (HR) was measured beat-by-beat using a transmitter belt (Wireless HR Monitor, COSMED) coupled to the gas analyzer.

Blood samples were collected 3, 5, and 7 min after the test to determine peak blood lactate concentration in all the tests (Zagatto et al. 2011). In the supramaximal efforts, blood samples at rest (after 10 min sitting) were also collected to estimate delta lactate concentration (peak minus rest). Blood samples were collected from the earlobe (25 μL) using heparinized capillaries and transferred to Eppendorf tubes containing 50 μL of sodium fluoride 1%. The samples were analyzed in an electrochemical lactimeter YSI 2300 STAT (Yellow Spring Instruments, Yellow Spring, Ohio, USA).

GXT

The GXT began at 8 km·h^{-1} with stage increments of 1.5 km·h^{-1} every 2 min until exhaustion, given voluntarily by the participant or by the inability to perform the effort at the predetermined speed. The GXT was based on the guidelines of Howley et al. (1995) for estimating $\dot{V}O_{2max}$ and was designed to last 8–12 min. The Borg scale (6–20) (Borg 1982) was used to assess the rate of perceived exertion (RPE) at the end of each stage of the GXT.

The highest average of the $\dot{V}O_2$ during the final 30 s of each stage was considered as $\dot{V}O_{2max}$ (Howley et al. 1995), considering the verification of at least 2 of the following criteria: the plateau in $\dot{V}O_2$ (variation in $\dot{V}O_2$ < 2.1 mL·kg^{-1}·min^{-1} between the last and penultimate stage of exercise); maximal HR (HR_{max}) ≥ 90% of predicted HR_{max} (220 – age); respiratory exchange ratio (RER) ≥ 1.10; and peak lactate ≥ 8.0 mmol·L^{-1} (Howley et al. 1995). If at least 2 criteria were not observed, a new test was applied. The exercise intensity at which the subject reached $\dot{V}O_{2max}$ was considered as $i\dot{V}O_{2max}$. If the final stage had not been completed, the $i\dot{V}O_{2max}$ was calculated using the equation proposed by Kuipers et al. (1985).

Supramaximal efforts and supplement ingestion

Subjects ingested $NaHCO_3$ (0.3 g·kg^{-1} of body weight) or a placebo (0.3 g·kg^{-1} of dextrose) 90 min before the supramaximal efforts. The $NaHCO_3$ and placebo substances were ingested through an identical 1-g gel capsule along with 500 mL of water. The subjects were instructed to drink the water and supplement

(i.e., placebo or $NaHCO_3$) within 10 min. Each subject performed the supramaximal efforts in both the $NaHCO_3$ and placebo supplementation conditions, which were performed in random order and separated by a minimum interval of 48 h for recovery.

Prior to supramaximal testing, subjects remained seated for 10 min to determine resting values of $\dot{V}O_2$ and blood lactate concentration. The subjects underwent 2 continuous exercises at an intensity corresponding to 110% of $i\dot{V}O_{2max}$, which was determined during GXT. The time to exhaustion was measured in each effort (t_{lim}) and was considered as an indicator of performance. After the test, the $\dot{V}O_2$ was measured for 7 min for determination of $EPOC_{FAST}$.

The variables $\dot{V}O_2$, $\dot{V}E$, and $\dot{V}CO_2$ were analyzed taking an average every 20 s from rest to the hundredth second during the effort and at exhaustion (the last 20 s were considered) of the supramaximal efforts (Yunoki et al. 2009).

$MAOD_{ALT}$ determination

The $MAOD_{ALT}$ was assumed as the sum of the anaerobic lactic metabolism and anaerobic alactic metabolism (Bertuzzi et al. 2010; Zagatto and Gobatto 2012). The $EPOC_{FAST}$ was used to estimate the contribution of the anaerobic alactic metabolism ($ANAER_{[PCR]}$), which was calculated using a bi-exponential fit (eq. 1) in OriginPro 8.0 software (OriginLab Corp., Microcal, Mass., USA) (Bertuzzi et al. 2010).

$$(1) \quad \dot{V}O_{2(t)} = \dot{V}O_{2baseline} + A_1[e^{-(t-\delta)/\tau_1}] + A_2[e^{-(t-\delta)/\tau_2}]$$

where $\dot{V}O_{2(t)}$ is the oxygen uptake at time (t); $\dot{V}O_{2baseline}$ is the oxygen uptake at baseline; A is the amplitude, δ is the time delay, and τ is the time constant — 1 and 2 represent the fast and slow components, respectively — and the $EPOC_{FAST}$ was calculated by the product of A_1 and τ_1.

The contribution of the lactic anaerobic metabolism ($ANAER_{[La]}$) was estimated by the difference between the amounts of blood lactate concentration at peak and rest, considering for each 1 mmol·L^{-1} lactate is equivalent to 3 mL O_2·kg^{-1} (di Prampero and Ferretti 1999). Net aerobic energy (AER) was estimated by subtracting the resting $\dot{V}O_2$ from the $\dot{V}O_2$ area during the supramaximal effort using the trapezoidal method (Bertuzzi et al. 2007).

The $MAOD_{ALT}$, $ANAER_{[PCR]}$, $ANAER_{[La]}$, and AER are presented in absolute values (liters) and normalized by total body mass (mL·kg^{-1}) and lean mass (mL·kg^{-1} lean mass).

Performance tests

The running tests (200 and 400 m) were performed on 2 separate days after a warm-up lasting about 10 min (running at low intensity for 3 laps and 1 at moderate intensity) and 4 min of rest. The test consisted of performing the established distances (200 and 400 m) in the shortest time possible. After completion of the test, blood samples were collected at 3, 5, and 7 min for determination of peak blood lactate concentration values. The same evaluator measured the displacement time using a manual timer (JS-6610, Junsd, Guangzhou, China).

Statistical analysis

The sample size was calculated (software G*Power 3) based on power analysis (statistical power of 90% and α error probability of 0.05) necessary for finding differences between 2 dependent-means blood lactate concentration after t_{lim} on a treadmill in healthy men (Price and Simons 2010) with adequate precision, which indicated 8 subjects. Data are presented as means ± SD and confidence interval of 95% (95% confidence interval (CI)). The variables were examined using the Shapiro–Wilk's test to verify the normality of data. For analysis of the values of MAOD, t_{lim}, AER, $ANAER_{[La]}$, and $ANAER_{[PCR]}$ of the placebo and $NaHCO_3$ groups, the paired t test was used for comparisons. In addition, the between-group differences of these variables from both the placebo and $NaHCO_3$ groups were analyzed qualitatively by magnitude-based, and expressed as standardized mean differences (Cohen's d) (Cohen 1988). The threshold values for Cohen's d statistical power were considered as >0.2 (small), >0.5 (moderate), and >0.8 (large). The chances of a possible substantial benefit or harm were calculated (0.2 multiplied by the between-subject deviation). Thus, the changes were qualitatively evaluated as follows: <1% = most unlikely; 1%–5% = very unlikely; 5%–25% = unlikely; 25%–75% = possibly; 75%–95% = likely; 95%–99% = very likely; and >99% = most likely (Hopkins et al. 2009). When the positive and negative values were both >5%, the inference was classified as unclear. The Pearson's correlation test was used to verify the association between these variables and between the $MAOD_{ALT}$ values and performance in the 200 m, 400 m, and t_{lim}. The coefficient of correlation was classified as very weak to negligible (0 to 0.2), weak (0.2 to 0.4), moderate (0.4 to 0.7), strong (0.7 to 0.9), and very strong (0.9 to 1.0) (Rowntree 1991). In all cases, a significance level of 5% was assumed.

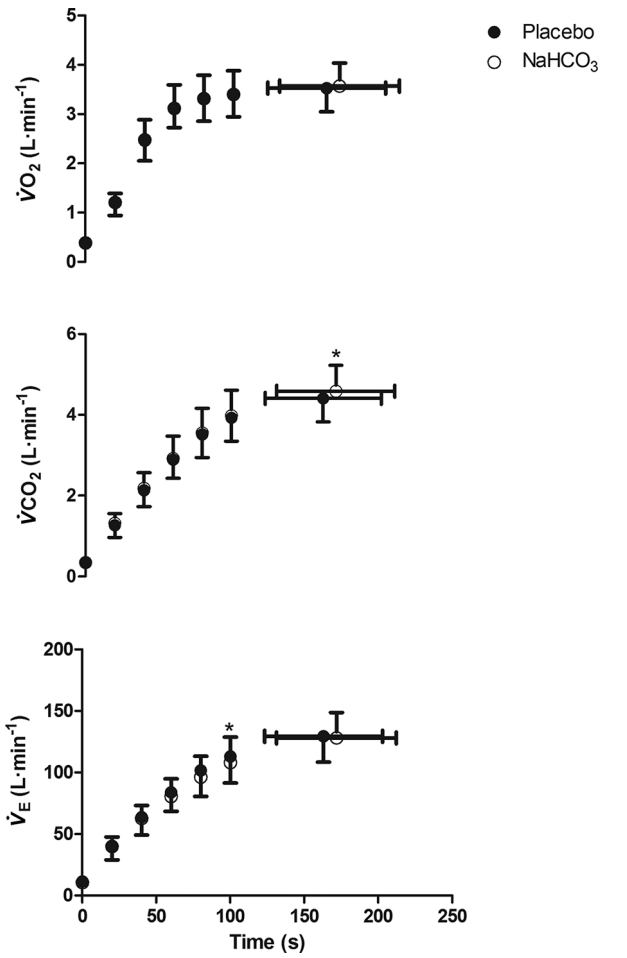


Fig. 3. Behavior of the variables oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), and pulmonary ventilation ($\dot{V}E$) during supramaximal efforts after sodium bicarbonate ($NaHCO_3$) and placebo ingestion. *, Significant difference to the placebo condition at the same time ($p < 0.05$).

Results

All subjects reached the exhaustion criteria in the GXT and did not need to repeat the test. The physiological response values at the exhaustion moment in the GXT were 3.7 ± 0.6 L·min^{-1} (95%CI 3.3 to 4.0 L·min^{-1}) and 50.6 ± 6.1 mL·kg^{-1}·min^{-1} (95%CI 47.3 to

Table 2. Coefficient of correlation (*r*) between performance and $MAOD_{ALT}$.

	200 m		400 m		t_{lim}	t_{lim}
	Time	$[La^-]$	Time	$[La^-]$	placebo (time)	$pNaHCO_3$ (time)
Placebo						
$MAOD_{ALT}$ (L)	0.25	0.07	−0.14	0.48	0.25	0.27
$MAOD_{ALT}$ (mL·kg^{-1})	−0.11	0.21	−0.34	0.58*	0.43	0.32
$MAOD_{ALT}$ (mL·kg^{-1} lean mass)	0.17	0.15	−0.05	0.44	0.34	0.14
AER (L)	0.11	0.03	−0.19	0.44	0.89*	0.74*
AER (mL·kg^{-1})	−0.10	0.22	−0.28	0.42	0.95*	0.74*
AER (mL·kg^{-1} lean mass)	0.04	0.14	−0.15	0.36	0.95*	0.69*
$ANAER_{[La]}$ (L)	0.25	0.05	0.14	0.22	0.07	0.14
$ANAER_{[La]}$ (mL·kg^{-1})	0.00	0.20	0.04	0.18	0.10	0.10
$ANAER_{[La]}$ (mL·kg^{-1} lean mass)	0.18	0.17	0.23	0.09	0.04	−0.01
$ANAER_{[PCR]}$ (L)	0.09	−0.15	−0.34	0.45	0.29	0.24
$ANAER_{[PCR]}$ (mL·kg^{-1})	−0.16	0.03	−0.49	0.52*	0.42	0.29
$ANAER_{[PCR]}$ (mL·kg^{-1} lean mass)	−0.01	−0.02	−0.35	0.44	0.38	0.18
$NaHCO_3$						
$MAOD_{ALT}$ (L)	0.13	0.11	−0.23	0.48	0.43	0.57*
$MAOD_{ALT}$ (mL·kg^{-1})	−0.23	0.35	−0.38	0.43	0.50	0.56*
$MAOD_{ALT}$ (mL·kg^{-1} lean mass)	−0.01	0.38	−0.16	0.35	0.52*	0.52*
AER (L)	−0.06	0.11	−0.25	0.32	0.64*	0.86*
AER (mL·kg^{-1})	−0.31	0.32	−0.36	0.30	0.69*	0.87*
AER (mL·kg^{-1} lean mass)	−0.22	0.27	−0.26	0.26	0.71*	0.86*
$ANAER_{[La]}$ (L)	0.10	0.21	−0.07	0.39	0.55	0.69*
$ANAER_{[La]}$ (mL·kg^{-1})	−0.14	0.35	−0.18	0.36	0.59*	0.66*
$ANAER_{[La]}$ (mL·kg^{-1} lean mass)	0.00	0.35	−0.02	0.28	0.59*	0.62*
$ANAER_{[PCR]}$ (L)	0.05	−0.18	−0.29	0.17	−0.21	−0.18
$ANAER_{[PCR]}$ (mL·kg^{-1})	−0.17	0.01	−0.38	0.17	−0.13	0.15
$ANAER_{[PCR]}$ (mL·kg^{-1} lean mass)	−0.03	−0.03	−0.29	0.06	−0.23	−0.30

Note: $[La^-]$, blood lactate concentration; $NaHCO_3$, sodium bicarbonate; $MAOD_{ALT}$, estimated maximal accumulated oxygen deficit; AER, net aerobic contribution; $ANAER_{[La]}$, lactic anaerobic contribution; $ANAER_{[PCR]}$, alactic anaerobic contribution.

*$p < 0.05$. t_{lim}, time until exhaustion in the supramaximal effort.

54.0 mL·kg^{-1}·min^{-1}) for the $\dot{V}O_{2max}$, 190 ± 6 beats·min^{-1} (95%CI 186 to 193 beats·min^{-1}) for HR_{max}, 1.17 ± 0.06 (95%CI 1.13 to 1.20) for RER, 18 ± 2 (95%CI 17 to 19) for RPE and 10.4 ± 2.4 mmol·L^{-1} (95%CI 9.1 to 11.8 mmol·L^{-1}) for peak blood lactate concentration. The total time of the GXT was 11.4 ± 1.4 min (95%CI 10.6 to 12.2), whereas the $i\dot{V}O_{2max}$ was 15.0 ± 1.1 km·h^{-1} (95%CI 14.4 to 15.6 km·h^{-1}). Based on $i\dot{V}O_{2max}$ values, the intensity at 110% of the $i\dot{V}O_{2max}$ considered for supramaximal efforts was 16.5 ± 1.2 km·h^{-1} (95%CI 15.9 to 17.2 km·h^{-1}).

The t_{lim}, $MAOD_{ALT}$, and energy system contributions determined during supramaximal efforts in the acute $NaHCO_3$ and placebo (dextrose) conditions are presented in Table 1, as well as the percentage changes and coefficients of correlation. All variables except $ANAER_{[PCR]}$ (in mL·kg^{-1} lean mass) and τ_1 were significantly correlated. Figure 1 presents the t_{lim} individual values.

The $MAOD_{ALT}$, when expressed in absolute (L) values, relative to total body mass (mL·kg^{-1}) and relative to lean mass (mL·kg^{-1} lean mass) was statistically different ($p = 0.01$) under supplementation conditions (Table 1). The values of $MAOD_{ALT}$ increased with $NaHCO_3$ ingestion. Furthermore, the absolute $MAOD_{ALT}$ presented a likely positive effect size (98%) under the $NaHCO_3$ condition (Fig. 2).

The resting blood lactate concentration values were not different ($p > 0.05$) in the placebo (1.1 ± 0.4 mmol·L^{-1}; 95%CI 0.9 to 1.3 mmol·L^{-1}) and $NaHCO_3$ (1.0 ± 0.4 mmol·L^{-1}; 95%CI 0.8 to 1.3 mmol·L^{-1}) conditions. However, the peak blood lactate concentration in the placebo group (11.1 ± 1.7 mmol·L^{-1}; 95%CI 10.1 to 12.0 mmol·L^{-1}) was lower than in the $NaHCO_3$ (12.5 ± 2.6 mmol·L^{-1}; 95%CI 11.0 to 13.9 mmol·L^{-1}) group ($p < 0.01$), as well as $ANAER_{[La]}$ expressed in absolute (L), relative to total body mass (mL·kg^{-1}) values ($p < 0.01$), with percentage changes between 14.8% and 15.3% (Table 1). In addition, the $ANAER_{[La]}$ expressed as absolute value (L) showed a very likely positive effect (Fig. 2.).

The comparisons of the values of $\dot{V}O_2$, $\dot{V}E$, and $\dot{V}CO_2$ between the placebo and $NaHCO_3$ groups during the supramaximal efforts are shown in Fig. 3. Significant differences were found for $\dot{V}CO_2$ at exhaustion ($p < 0.05$) and for $\dot{V}E$ in the hundredth second ($p = 0.03$).

Performance in the 200 m and 400 m were 31.3 ± 2.7 s (95%CI 29.8 to 32.8 s) and 73.8 ± 6.1 s (95%CI 70.4 to 77.1 s), respectively. The peak blood lactate concentration was 11.5 ± 2.2 mmol·L^{-1} (95%CI 10.2 to 12.7 mmol·L^{-1}) for the 200 m running test, which was lower than the 400 m running test (12.5 ± 1.8 mmol·L^{-1}; 95%CI 11.5 to 13.6 mmol·L^{-1}).

The coefficient of correlation (*r*) values between $MAOD_{ALT}$, AER, $ANAER_{[La]}$, and $ANAER_{[PCR]}$ in both the placebo and $NAHCO_3$ conditions and performance in the 200 m, 400 m, and t_{lim} are presented in Table 2. A very strong and significant correlation was observed only between AER (in mL·kg^{-1} and mL·kg^{-1} lean mass) and t_{lim} in the placebo group. There were correlations between $ANAER_{[La]}$ and $MAOD_{ALT}$ with the t_{lim} only in the $NaHCO_3$ condition (moderate significance). There were no correlations between the $MAOD_{ALT}$ (in both the placebo and $NaHCO_3$ conditions) and performance in the 200 and 400 m running tests (Table 2).

Discussion

To our knowledge, this is the first study to analyze the effects of supplementation with $NaHCO_3$ on $MAOD_{ALT}$. The main finding of the present study was a significant difference and a likely positive effect on $MAOD_{ALT}$ after acute supplementation of $NaHCO_3$, suggesting that this protocol is sensitive to induced alkalosis. Moreover, a very likely positive effect for $ANAER_{[La]}$ (in liters) was found with acute supplementation of $NaHCO_3$. However, no significant correlations were found between $MAOD_{ALT}$ and 200- and 400-m running performances.

The difference in $MAOD_{ALT}$ after acute ingestion of $NaHCO_3$ can be explained by the significant increase in $ANAER_{[La]}$ (Table 1), which was due to the increase in blood lactate concentration postexercise. The increase in blood lactate concentration postexercise

was expected (Cameron et al. 2010; Carr et al. 2012, 2013; Kupcis et al. 2012; Price and Cripps 2012; Siegler et al. 2013; Hobson et al. 2014), and although the exact cause for the rise in blood lactate concentration should be further elucidated (Carr et al. 2013), some hypotheses are presented as possible explanations.

One possible explanation is the increase in cellular efflux of H^+ ions owing to maintenance of the pH gradient between the cells and blood (Kupcis et al. 2012). The lactate efflux across the sarcolemma occurs via a monocarboxylate transporter (i.e., MCT1) (Raymer et al. 2004). The MCT1 is a concentration and gradient-dependent transporter, so the oral ingestion of $NaHCO_3$ leads to a higher efflux of lactate as the result of higher gradients of H^+ (Zinner et al. 2011).

Other hypotheses — including the increase in intramuscular glycolytic flux generated by increased activity of the phosphofructokinase enzyme and glycogen phosphorylase (Trivedi and Danforth 1966), and reduced uptake of lactate by body tissues and release of lactate into the blood by the tissues (Carr et al. 2013) — also try to explain the increase in blood lactate concentration postexercise. In any case, the $MAOD_{ALT}$ seems to be sensitive enough to detect changes in the $ANAER_{[La]}$.

No changes were found in t_{lim} (Fig. 3), unlike in some studies (Iwaoka et al. 1989; Van Montfoort et al. 2004; Higgins et al. 2013; Mueller et al. 2013). However, there was a visible improvement tendency in the $NaHCO_3$ condition when analyzing individual data (Fig. 1). The lack of effect of $NaHCO_3$ on t_{lim} can be explained by the untrained nature of the participants involved, which caused great variation in t_{lim} (Higgins et al. 2013). Furthermore, correlations between $MAOD_{ALT}$, $ANAER_{[La]}$, and t_{lim} were observed only with the ingestion of $NaHCO_3$ (Table 2), suggesting that individuals who responded most to supplementation had the best t_{lim}, and therefore it is very difficult to exclude the possibility of a possible effect of $NaHCO_3$ supplementation on t_{lim}.

The increased $\dot{V}CO_2$ at exhaustion (Fig. 2) probably occurred as a consequence of an elevated rate of bicarbonate buffering in the blood, resulting in CO_2 release via carbonic acid (Vanhatalo et al. 2010). Furthermore, the decrease in $\dot{V}E$ with $NaHCO_3$ ingestion evidenced that $NaHCO_3$ supplementation generated acute physiological changes in the subjects. One possible explanation for the increase in $\dot{V}CO_2$ and decrease in $\dot{V}E$ is that CO_2 flow is not the cause of the close relationship between $\dot{V}CO_2$ and $\dot{V}E$ (Yunoki et al. 2009) and possibly $\dot{V}CO_2$ follows $\dot{V}E$ rather than vice versa (Péronnet and Aguilaniu 2006).

In relation to performance, negative correlations were expected in the 200-m and 400-m times with $MAOD_{ALT}$ (Table 2). Ramsbottom et al. (1994) showed that the accumulated oxygen deficit estimated on a treadmill was highly correlated with the times obtained in distances of 100 m ($r = -0.88$) and 400 m ($r = -0.82$) and showed a moderate correlation with time in the 800-m distance ($r = -0.61$). The average track times were 13.6 ± 1.3, 60.9 ± 6.8 and 138.8 ± 18.5 s for the 100-, 400-, and 800–m distances, respectively. In the present study, the lack of a significant correlation between $MAOD_{ALT}$ and performance may be related to the group fitness level, which was moderately active. It is probable that anaerobic capacity is not such a determinant for moderately active individuals as for athletes. Therefore, association analyses using athletes is recommended in future studies.

A possible limitation of the study was not measuring blood and muscle pH before and after the supramaximal efforts. In addition, higher blood lactate levels may be a result of facilitating lactate efflux and H^+ ions in response to a change in the electrochemical gradient (i.e., alkalosis induced because of acute ingestion of $NaHCO_3$) (Siegler et al. 2008). Thus, the values of $MAOD_{ALT}$, as well as the values of $ANER_{[La]}$, may have been overestimated.

A practical application of the study is the feasibility to measure anaerobic capacity using a single exhaustive supramaximal effort and verifying that $MAOD_{ALT}$ is sensitive enough to detect modifications in alactic and/or lactic energy systems such as adaptations, which occur with training and observed in the current study with supplementation of $NaHCO_3$. In addition to MAOD being considered the best procedure to evaluate anaerobic capacity, the $MAOD_{ALT}$ has the added advantage of estimating individually the maximal alactic and lactic energy system capacities, impossible in the conventional MAOD procedure.

In summary, acute $NaHCO_3$ ingestion improved $MAOD_{ALT}$ and seems not to be associated with 200- and 400-m running performance in moderately active individuals.

Acknowledgements

The authors would like to thank the FAPESP, process numbers 2013/12940-8 and 2014/02829-5.

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