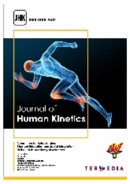

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Section II – Exercise Physiology and Sports Medicine

Acute Sodium Bicarbonate Supplementation in Finswimming: Performance, Stroke Mechanics, and Perceptual Responses

by
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This study examined the acute effects of sodium bicarbonate ($NaHCO_3$) supplementation on performance, stroke mechanics, as well as physiological and perceptual responses during a simulated 200-m Bi-Fins race. Nineteen national-level athletes (10 females, 9 males) completed a randomized, double-blind, placebo-controlled crossover trial under three different conditions: control, placebo, and $NaHCO_3$ supplementation ($300\ mg{\cdot}kg^{-1}$ body mass). Performance and biomechanical variables (split times, stroke rate, stroke length, stroke index) were obtained from video analysis, while blood samples were collected pre- and post-test to assess acid-base status and lactate. Ratings of perceived exertion (RPEs) and muscle soreness (VAS) were also recorded. $NaHCO_3$ supplementation induced systemic alkalosis, with higher pre-test HCO_3^- ($30.1 \pm 1.8\ mmol{\cdot}L^{-1}$), pH (7.484), as well as ABE ($+6.5\ mmol{\cdot}L^{-1}$), with these values remaining elevated post-test compared to control and placebo conditions (all $p < 0.001$). Early splits (25 and 50 m) were slower after $NaHCO_3$ supplementation ($p < 0.05$). Post-test blood lactate was slightly higher following the placebo supplementation (14.3 vs. $13.4\ mmol{\cdot}L^{-1}$, $p_{adj} = 0.030$), although the absolute difference ($<1\ mmol{\cdot}L^{-1}$) was physiologically small. RPEs were reduced after $NaHCO_3$ versus placebo supplementation ($p_{adj} = 0.002$; large effect), while no significant differences were observed in VAS scores. These findings demonstrate that although $NaHCO_3$ supplementation effectively induced alkalosis and lowered perceived exertion, it did not improve overall performance, with only very small changes in biomechanical variables during certain race sections. From a practical perspective, the attenuation of acidosis and reduced exertion may support faster recovery and performance stabilization in repeated-race formats (e.g., heats and finals).

***Keywords**: motor skills; blood lactate; acid-base equilibrium; exercise performance; metabolic alkalosis*

Introduction

Finswimming is a complex aquatic discipline that requires exceptional aerobic-anaerobic capacity and refined technical proficiency (Sládečková et al., 2024). In the Bi-Fins event, athletes swim the front crawl using two fins and a front-mounted snorkel. Among the biomechanical determinants of performance, the relationship between the stroke rate (SR) and stroke length (SL) directly determines swimming velocity, making their balance fundamental for efficiency (Almeida et al., 2023).

These requirements underscore the importance of maintaining stable inter-limb coordination in cyclic aquatic movements (Mezêncio et al., 2020). Any SR-SL imbalance—particularly with increasing velocity or accumulating fatigue—can disrupt stroke timing and elevate the energetic cost of propulsion (Dekerle and Paterson, 2016). These demands are even more pronounced in the 200-m Bi-Fins event, in which elite athletes typically achieve race times

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of ~1:38–1:44 (men) and ~1:47–1:54 (women). Beyond the clean-swimming phase of the race, overall performance is strongly influenced by acyclic phases—particularly the start (Kalaitzoglidis et al., 2025) and turn segments (Michalica et al., 2024) which require high levels of explosive force production, rapid underwater acceleration, and efficient execution of underwater segments (Veiga et al., 2022). Short-course (25 m) racing further increases technical complexity and modifies anaerobic loading due to more frequent turns and longer cumulative underwater distances, leading to distinct physiological responses compared with long-course (50 m) swimming (Keskinen et al., 2007).

These biomechanical demands interact closely with metabolic stress. Following a brief ATP-CP contribution, swimmers rapidly transition into anaerobic glycolysis, resulting in lactate accumulation and metabolic acidosis (Lindh et al., 2008). These conditions typically reduce SL, prompting athletes to elevate the SR to maintain velocity (Cuenca-Fernández et al., 2023). Because acidosis represents a major performance-limiting factor, nutritional strategies increasingly focus on buffering interventions. Sodium bicarbonate ($NaHCO_3$) is among the most widely studied buffers, inducing systemic alkalosis and enhancing extracellular buffering capacity (Grgic et al., 2021), thereby helping preserve contractility and delay both peripheral and central fatigue—mechanisms particularly relevant in anaerobically demanding aquatic sports (Domínguez et al., 2017).

Beyond its physiological effects, $NaHCO_3$ may also modulate perceptual load. Gurton et al. (2025) observed reduced ratings of perceived exertion (RPEs) during 200-m and 400-m swimming trials following $NaHCO_3$ ingestion despite an unchanged heart rate. While perceptual and metabolic benefits are well documented in pool swimming (Gough et al., 2023; Joyce et al., 2012; Kumstát et al., 2018), their practical relevance for finswimming remains unclear. This raises an important applied question: should finswimmers ingest $NaHCO_3$ before a 200-m race? Current literature reports small but meaningful performance improvements in 200–400-m swimming events (e.g., SMD −0.22; ~1–1.3%; Grgic et al., 2021) and approximately 0.6% gains in 400-m freestyle (Kumstát et al., 2018), but these effects are moderated by factors such as event duration, propulsion characteristics, and the extent of underwater phases—dimensions that may differ substantially between finswimming and conventional swimming.

A critical gap is that no study has simultaneously examined acid-base responses, perceptual markers, stroke mechanics, and performance within a unified experimental design. Therefore, the present study aimed to determine whether $NaHCO_3$ ingestion would influence acid-base balance, perceptual responses, stroke mechanics (SR, SL, SI), and overall performance during a simulated 200-m Bi-Fins race. Overall 200-m time was predefined as the primary outcome, with acid-base variables serving as the principal secondary outcomes and biomechanical (SR, SL, SI) and perceptual measures considered supplementary endpoints.

Methods

Participants

Initially, twenty-one elite Czech finswimmers (11 females and 10 males) were recruited for this study through direct contact with competitive finswimming clubs. Two participants (one female and one male) had to withdraw from the study due to acute illness that was unrelated to the experimental protocol. The final sample included nineteen athletes (age: 19.1 ± 5.0 years; height: 173.9 ± 8.8 cm; body mass: 67.2 ± 8.4 kg; 10 females and 9 males), all active members of the junior or senior Czech national finswimming team. Each athlete ranked among the top ten in the Bi-Fins disciplines at the 2024 Czech National Finswimming Championships. Inclusion criteria required regular competitive training, absence of chronic illness or injury, and no current medication or supplement use. All participants were free from increased gastrointestinal discomfort throughout the study. Written informed consent was obtained from all athletes; for minors, also from a parent or a legal guardian. This study was conducted following the principles of the Declaration of Helsinki, and approved by the Ethics Committee of the Palacký University Olomouc, Olomouc, Czech Republic (protocol code: 02/2024; approval date: 04 January 2024). The menstrual cycle phase of female athletes was not controlled or recorded, which represents a methodological limitation but reflects common practice in acute supplementation studies.

Measures

Performance and Stroke Mechanics Analysis

Performance and biomechanical variables were assessed using video analysis. A digital camera (Sony FDR-AX700, Tokyo, Japan) was positioned 4.0 m above the water surface, perpendicularly to the swimming direction, and recorded at full HD resolution (1920 × 1080, 50 fps). Video footage was analyzed with Dartfish software (Live S, Fribourg, Switzerland). A light signal synchronized with the camera indicated the race start, and the first wall contact of the fins determined 25-m split times and final race time (Born et al., 2021). From the video data, the stroke rate (SR), stroke length (SL), and the stroke index (SI) were calculated. Manual split times were simultaneously recorded by three independent timekeepers using stopwatches (Casio, HS-80TW-1EF, Tokyo, Japan) to verify video-derived data, but only video-based measurements were used for statistical analysis.

Capillary Blood Collection

Capillary blood (140 μL) was collected from the lateral surface of the distal phalanx using pre-heparinized glass capillaries (Keraglass, Czech Republic). The site was disinfected, and after discarding the first drop, samples were filled by passive flow and immediately sealed to avoid air contact. All samples were analyzed within 1 hour of collection.

Acid-Base Balance

Acid-base status was assessed using an ABL 825 FLEX analyzer (Radiometer Medical ApS, Denmark), which directly measures blood pH and calculates plasma bicarbonate concentration (HCO_3^-) and actual base excess (ABE). The analyzer was calibrated according to the manufacturer's instructions. Capillary blood lactate concentration was measured using a portable Lactate Scout analyzer (EKF Diagnostics, Germany), which has demonstrated validity and reliability for swimmers (Nikitakis and Toubekis, 2021).

Perceptual Responses Assessment

Perceptual responses were assessed using the Borg 1–10 scale and a 10-cm visual analogue scale (VAS). Perceived exertion (RPE) was evaluated immediately after the 200-m Bi-Fins trial (Ueda and Kurokawa, 1995), while gastrointestinal discomfort was rated ~5 min before the race to detect potential symptoms affecting performance (Jiang et al., 2024). Delayed-onset muscle soreness (DOMS) was measured 3 min post-race using a 10-cm VAS (0 = "no soreness", 100 = "worst imaginable soreness") (Sládečková et al., 2024). Soreness of the lower limbs was rated in a standardized squat position (~90° knee flexion) (Jakeman et al., 2010), and soreness of the upper limbs during maximal voluntary flexion-extension of the elbow (Sakamoto et al., 2009).

Design and Procedures

This randomized, double-blind, placebo-controlled crossover study investigated the effects of sodium bicarbonate ($NaHCO_3$) supplementation on performance, stroke mechanics, as well as selected physiological and perceptual responses in competitive finswimmers. The study adhered to CONSORT guidelines for crossover trials and was preregistered at ClinicalTrials.gov (identifier: NCT07135934). The study comprised three sessions. Session 1 served as a standardized control for baseline and familiarization. Sessions 2 and 3 ($NaHCO_3$ and placebo) were individually randomized using computer-generated allocation to ensure counterbalancing and minimize order effects.

Participants completed three testing sessions, each separated by a 48-hour recovery period during which no training was performed. The first session served as a control condition with no intervention. The second and third sessions involved ingestion of either $NaHCO_3$ (300 mg·kg^{-1} body mass) or a placebo (food-grade starch in gelatin capsules), administered in randomized, counterbalanced order. The $NaHCO_3$ dose was individually adjusted according to body mass on the test day and ingested within a 30-min period, finishing 60–90 min before the simulated race to coincide with the expected time to peak blood bicarbonate concentration (Farias De Oliveira et al., 2020). Water was provided ad libitum during ingestion, and gastrointestinal tolerance was monitored throughout the study. Placebo composition (food-grade starch) has been previously validated as inert in exercise performance trials (Beedie et al., 2007). The use of non-coated gelatin capsules was consistent with earlier swimming research (Gurton et al., 2025).

Neither participants nor investigators could distinguish between the two conditions.

All sessions were conducted in the afternoon under standardized conditions at the Aplikační centrum BALUO, Palacký University Olomouc, using a 25-m indoor test pool. Each session began with a standardized 15–20-min warm-up (1,400 m) performed with identical fins and equipment as in the race simulation; water temperature remained stable (27.6 ± 0.2°C). Immediately afterwards, participants ingested either $NaHCO_3$ or a placebo, and the simulated 200-m Bi-Fins race was conducted 60–90 min later. Blood samples were collected at two time points: 5 min before the race and 3 min post-exercise. All trials were performed in accordance with international CMAS competition rules (Finswimming CMAS Rules, 2023). Data collection took place during the first half of 2025. Participants were instructed to refrain from training on the day of testing, abstain from stimulants (including caffeine), ergogenic supplements and alcohol for 24 h before each session, and maintain consistent eating habits and daily routines throughout the study. All comparisons were conducted within-subject.

Statistical Analysis

Data are presented as mean ± SD. Assumptions were tested with the Shapiro-Wilk test (normality) and the Mauchly's test of sphericity; if sphericity was violated, Greenhouse-Geisser corrections were used. For the pre-post outcomes (acid-base markers and perceptual scales), we used a 3 × 2 repeated-measures ANOVA with condition (Control, Placebo, $NaHCO_3$) and time (pre, post) as within-subject factors, yielding tests of the main effects of time and condition and their time × condition interaction. Simple-effects analyses and Bonferroni-adjusted pairwise comparisons were then used to examine between-condition differences and within-condition pre-post changes. For test performance (finish time, 25–200-m split times) and stroke mechanics (SL, SI, SR), conditions were compared at each split using one-way repeated-measures ANOVA, and changes across distance were examined. Pairwise comparisons between conditions were carried out with Bonferroni adjustment regardless of ANOVA significance (a priori interest in condition differences); all reported pairwise p values are Bonferroni-adjusted (p_{adj}).

Effect sizes were reported as partial eta-squared (ηp^2) for ANOVA effects and bias-corrected Hedges' g for pairwise contrasts. For interpretation we used conventional benchmarks ($\eta p^2 = 0.01/0.06/0.14$ for small/medium/large effects and Hedges' g = 0.20/0.50/0.80 for small/medium/large effects). Statistical significance was set at $\alpha = 0.05$ and all reported p-values were two-sided with Bonferroni adjustment for multiple comparisons. The study was planned a priori for power $1-\beta = 0.80$ to detect $f = 0.30$ ($\eta^2_p = 0.083$; G*Power 3.1). Video-analysis reliability was assessed on ~50% of trials using ICC(3,1) = 0.996, 95% CI 0.993–0.998. Analyses were performed in IBM SPSS Statistics v28 and Microsoft Excel v2110.

Results

Split Times and Final Time

A significant condition × section interaction was observed ($p < 0.001$), with slower split times under the sodium bicarbonate supplementation condition at 25- and 50-m splits compared with control and placebo conditions ($p_{adj} < 0.05$). No further differences were detected in later splits or in overall 200-m performance time (all $p > 0.10$; Table 1).

Stroke Length (SL), the Stroke Rate (SR), and the Stroke Index (SI)

Across all 25-m splits of the 200-m trial, a three-level repeated-measures ANOVA (condition: control, placebo, $NaHCO_3$) revealed no main effect of the condition for stroke length (SL), the stroke index (SI) or the stroke rate (SR) (all $p > 0.05$; partial η^2 small). Bonferroni-adjusted pairwise contrasts showed trivial-to-small differences ($|g|$ generally ≤ 0.3) and were non-significant, with one isolated exception at 150 m for the SI, where the placebo condition led to lower values compared to the $NaHCO_3$ supplementation condition ($g \approx -0.27$, $p_{adj} \approx 0.019$). The race profiles of the SR (progressive decrease) and SL (slight increase) were highly similar across conditions, indicating no meaningful ergogenic effect of $NaHCO_3$ on stroke mechanics over 200 m.

Physiological Responses: Acid-Base Variables and Lactate

Sodium bicarbonate supplementation

induced a clear systemic alkalosis, with significantly higher pre- and post-exercise HCO_3^-, pH, and ABE values under the $NaHCO_3$ supplementation condition compared with control and placebo conditions (all $p < 0.001$; $\eta^2p \geq 0.73$). Complete descriptive and inferential statistics for acid-base variables are provided in Table 3, and post-exercise distributions are visualized in Figure 1. Baseline lactate did not differ among conditions. Following testing, lactate was statistically higher after $NaHCO_3$ compared with placebo supplementation ($p_{adj} = 0.030$; $\eta^2p = 0.18$), although the absolute difference (<1 mmol·L⁻¹) was small and unlikely to be physiologically meaningful.

Ratings of Perceived Exertion (RPE) and Muscle Soreness (VAS)

A significant main effect of condition was observed for the RPE ($p = 0.002$; $\eta^2p = 0.20$), with lower values following $NaHCO_3$ compared with placebo supplementation ($p_{adj} = 0.002$; $g = 1.10$), and a trend toward reduction compared with the control condition ($p_{adj} = 0.057$). Post-exercise RPE distributions are illustrated in Figure 1. VAS scores for upper and lower limbs showed no effect of condition (all $p > 0.65$), with full statistics presented in Table 4.

Repeated-measures ANOVA showed robust time × condition interactions for all acid-base variables and blood lactate (all $p < 0.001$, partial $\eta^2 \approx 0.92–0.97$), indicating that pre- to post-test changes differed substantially among control, placebo and $NaHCO_3$ supplementation conditions. Significant main effects of condition were present for HCO_3^-, pH and ABE (all $p < 0.001$), whereas for LAC the condition effect was only a trend ($p = 0.084$). Across conditions, blood lactate concentration increased and pH and ABE decreased over time (all $p < 0.05$), while the overall time effect for HCO_3^- was not significant ($p = 0.104$).

Table 1. Split times (s) and total 200-m performance under control, placebo, and $NaHCO_3$ conditions: descriptive statistics and repeated-measures ANOVA with post-hoc comparisons.

Splits	Control (M ± SD)	Placebo (M ± SD)	Soda (M ± SD)	ANOVA F(df)	*p*	partial η^2	C–P: g (p_{adj})	C–S: g (p_{adj})	P–S: g (p_{adj})
25 m	12.336 ± 0.921	12.347 ± 0.987	12.564 ± 1.026	7.377	0.002	0.291	−0.01 (1.000)	−0.22 (0.041)	−0.21 (0.023)
50 m	13.783 ± 1.207	13.749 ± 1.168	13.976 ± 1.218	4.792	0.014	0.21	+0.03 (1.000)	−0.15 (0.069)	−0.18 (0.043)
75 m	14.389 ± 1.200	14.322 ± 1.223	14.577 ± 1.222	3.201	0.053	0.151	+0.05 (1.000)	−0.15 (0.147)	0.20 (0.103)
100 m	14.771 ± 1.172	14.753 ± 1.195	14.877 ± 1.989	2.143	0.132	0.106	+0.01 (1.000)	−0.06 (0.502)	−0.07 (0.158)
125 m	14.975 ± 1.251	14.949 ± 1.306	14.958 ± 1.235	0.726	0.491	0.039	+0.02 (1.000)	+0.01 (1.000)	−0.01 (1.000)
150 m	15.308 ± 1.262	15.285 ± 1.409	15.216 ± 1.257	0.678	0.514	0.036	+0.02 (1.000)	+0.07 (1.000)	+0.05 (1.000)
175 m	15.260 ± 1.228	15.254 ± 1.306	15.167 ± 1.323	0.678	0.514	0.036	+0.00 (1.000)	+0.07 (1.000)	+0.06 (1.000)
200 m	14.579 ± 1.131	14.599 ± 1.262	14.423 ± 1.297	1.978	0.153	0.099	−0.02 (1.000)	+0.12 (1.000)	+0.13 (1.000)
Finnish time	115.401 ± 8.941	115.321 ± 9.493	115.713 ± 9.378	0.709	0.499	0.038	+0.01 (1.000; 95% CI −0.74 to 0.90)	−0.03 (1.000; 95% CI −1.28 to 0.66)	−0.04 (0.881; 95 % CI −1.35 to 0.56)

Values are mean ± SD; ANOVA = repeated-measures analysis of variance; η²p = partial eta squared; g = Hedges' effect size; p_adj = Bonferroni-adjusted p-value

Table 2. Stroke length (SL), stroke rate (SR), and stroke index (SI) across the 200-m Bi-Fins race under control, placebo, and $NaHCO_3$ conditions.

Splits	Outcome	Control (M ± SD)	Placebo (M ± SD)	Soda (M ± SD)	ANOVA F(df)	*p*	partial η^2	C–P g (p_{adj})	C–S g (p_{adj})	P–S g (p_{adj})
25 m	SL	2.640 ± 0.239	2.607 ± 0.221	2.626 ± 0.246	0.689	0.509	0.037	0.14 (0.935)	0.06 (1.000)	−0.08 (1.000)
	SI	4.610 ± 0.553	4.548 ± 0.537	4.515 ± 0.539	2.149	0.131	0.107	0.11 (0.522)	0.17 (0.138)	0.06 (1.000)
	SR	39.904 ± 5.007	40.395 ± 5.229	39.514 ± 5.299	1.24	0.301	0.064	−0.09 (1.000)	0.07 (1.000)	0.16 (0.347)
50 m	SL	2.770 ± 0.277	2.734 ± 0.249	2.756 ± 0.259	0.787	0.463	0.042	0.13 (0.868)	0.05 (1.000)	−0.08 (1.000)
	SI	4.653 ± 0.601	4.603 ± 0.553	4.569 ± 0.568	1.652	0.206	0.084	0.08 (0.934)	0.14 (0.260)	0.06 (1.000)
	SR	36.588 ± 4.760	37.089 ± 4.597	36.269 ± 4.698	1.119	0.338	0.059	−0.10 (1.000)	0.07 (0.403)	0.17 (0.403)
75 m	SL	2.759 ± 0.269	2.736 ± 0.221	2.771 ± 0.277	0.622	0.543	0.033	0.09 (1.000)	−0.04 (1.000)	−0.14 (0.626)
	SI	4.568 ± 0.630	4.420 ± 0.541	4.429 ± 0.605	0.337	0.716	0.018	0.25 (1.000)	0.22 (1.000)	-0.02 (1.000)
	SR	35.240 ± 4.310	35.462 ± 3.825	34.765 ± 4.148	1.034	0.366	0.054	−0.05 (1.000)	0.11 (0.354)	0.17 (0.354)
100 m	SL	2.721 ± 0.221	2.700 ± 0.185	2.718 ± 0.240	0.45	0.641	0.024	0.10 (1.000)	0.01 (1.000)	−0.08 (1.000)
	SI	4.271 ± 0.512	4.236 ± 0.426	4.248 ± 0.520	0.402	0.672	0.022	0.07 (1.000)	0.04 (1.000)	−0.02 (1.000)
	SR	34.630 ± 3.651	34.919 ± 3.694	34.536 ± 3.553	0.6	0.554	0.032	−0.08 (1.000)	0.03 (1.000)	0.10 (0.812)
125 m	SL	2.710 ± 0.232	2.688 ± 0.190	2.711 ± 0.216	0.534	0.591	0.029	0.10 (1.000)	−0.00 (1.000)	−0.11 (0.732)
	SI	4.212 ± 0.539	4.180 ± 0.472	4.222 ± 0.495	0.526	0.596	0.028	0.06 (1.000)	−0.02 (1.000)	−0.09 (1.000)
	SR	34.435 ± 3.639	34.742 ± 3.845	34.487 ± 3.532	0.305	0.739	0.017	−0.08 (1.000)	−0.01 (1.000)	0.07 (1.000)
150 m	SL	2.682 ± 0.236	2.641 ± 0.181	2.695 ± 0.209	1.621	0.214	0.084	0.19 (0.812)	−0.06 (1.000)	−0.27 (0.019)
	SI	4.071 ± 0.565	4.007 ± 0.407	4.119 ± 0.486	1.77	0.198	0.090	0.13 (0.985)	−0.09 (1.000)	−0.24 (0.010)
	SR	33.927 ± 3.300	34.549 ± 4.132	34.003 ± 3.253	1.22	0.307	0.063	−0.16 (0.727)	−0.02 (1.000)	0.14 (0.440)
175 m	SL	2.674 ± 0.200	2.643 ± 0.157	2.659 ± 0.193	0.784	0.464	0.042	0.17 (0.834)	0.07 (1.000)	−0.09 (1.000)
	SI	4.090 ± 0.487	4.038 ± 0.394	4.099 ± 0.479	0.838	0.441	0.044	0.11 (0.857)	−0.02 (1.000)	−0.14 (0.613)
	SR	34.328 ± 3.377	34.678 ± 3.752	34.695 ± 3.494	0.814	0.452	0.043	−0.10 (1.000)	−0.10 (0.755)	−0.00 (1.000)
200 m	SL	2.736 ± 0.193	2.751 ± 0.199	2.738 ± 0.168	0.213	0.809	0.012	−0.07 (1.000)	−0.01 (1.000)	0.07 (1.000)
	SI	4.423 ± 0.427	4.464 ± 0.484	4.501 ± 0.489	1.083	0.349	0.057	−0.09 (1.000)	−0.17 (0.308)	−0.07 (1.000)
	SR	36.572 ± 3.860	35.585 ± 4.136	36.136 ± 3.942	1.115	0.339	0.058	0.24 (1.000)	0.11 (0.551)	−0.13 (0.605)

Values are mean ± SD. SL = stroke length (m); SR = stroke rate (cycle/min); SI = stroke index

Table 3. Acid-base variables and blood lactate concentrations under control, placebo, and $NaHCO_3$ conditions.

Outcome	Control (M ± SD)	Placebo (M ± SD)	Soda (M ± SD)	ANOVA F(df1.df2)	*p*	partial η²	C–P: *g* (p_{adj}; 95% CI)	C–S: *g* (p_{adj}; 95% CI)	P–S: *g* (p_{adj}); 95% CI)
HCO3 pre	22.91 ± 1.64	23.25 ± 2.22	30.14 ± 1.82	207.529	**<0.001**	0.92	−0.17 (1.000; −1.44 to 0.75)	−4.00 (<0.001; −8.15 to −6.33)	−3.27 (<0.001; −8.05 to −5.74)
HCO3 post	10.363 ± 2.045	10.016 ± 1.922	14.484 ± 3.099	89.839	**<0.001**	0.833	+0.17 (0.802; −0.45 to 1.15)	−1.53 (<0.001; −5.22 to −3.03)	−1.71 (<0.001; −5.49 to −3.45)
pH pre	7.408 ± 0.023	7.414 ± 0.041	7.484 ± 0.021	49.324	**<0.001**	0.733	−0.17 (1.000; −0.032 to 0.021)	−3.34 (<0.001; −0.089 to −0.063)	−2.19 (<0.001; −0.097 to −0.045)
pH post	7.162 ± 0.066	7.157 ± 0.071	7.261 ± 0.078	78.462	**<0.001**	0.813	+0.08 (1.000; −0.013 to 0.024)	−1.32 (<0.001; −0.128 to −0.070)	−1.35 (<0.001; −0.130 to −0.079)
ABE pre	-0.85 ± 1.39	-0.48 ± 2.38	6.47 ± 1.37	175.391	**<0.001**	0.907	−0.18 (1.000; −1.65 to 0.92)	−5.06 (<0.001; −8.15 to −6.48)	−3.55 (<0.001; −8.26 to −5.65)
ABE post	-17.521 ± 3.563	-18.084 ± 3.681	-11.463 ± 4.600	100.919	**<0.001**	0.849	+0.15 (0.577; −0.54 to 1.66)	−1.42 (<0.001; −7.62 to −4.50)	−1.53 (<0.001; −8.02 to −5.23)
LAC pre	1.200 ± 0.438	1.047 ± 0.331	1.058 ± 0.339	1.229	0.305	0.064	+0.38 (0.598; −0.15 to 0.46)	+0.35 (0.689; −0.16 to 0.44)	−0.03 (1.000; −0.27 to 0.24)
LAC post	13.947 ± 2.408	13.416 ± 2.024	14.268 ± 2.216	3.937 (2.36)	**0.028**	0.179	+0.23 (0.269; −0.25 to 1.31)	−0.136 (1.000; −1.19 to 0.54)	−0.39 (0.030; −1.63 to −0.07)

Values are mean ± SD. HCO_3^- *= bicarbonate (mmol/l); ABE = actual base excess (mmol/l); LAC = blood lactate (mmol/l); Bold values (e.g., <0.001) indicate statistical significance*

Table 4. Ratings of perceived exertion (RPE) and muscle soreness (VAS) under control, placebo and $NaHCO_3$ conditions.

Outcome	Control (M ± SD)	Placebo (M ± SD)	Soda (M ± SD)	ANOVA F(df1.df2)	*p*	partial η²	C–P: *g* (p_{adj})	C–S: *g* (p_{adj})	P–S: *g* (p_{adj})
Borg post	8.68 ± 0.67	9.00 ± 0.82	8.05 ± 0.91	6.81 (2.54)	**0.002**	0.201	−0.41 (.697; 95% CI −0.67 to 0.04)	0.77 (.057; 95% CI 0.13 to 1.14)	1.10 (.002; 95% CI 0.36 to 1.54)
VAS upper limbs	49.47 ± 16.08	54.21 ± 18.51	51.47 ± 23.71	0.277 (2.54)	0.759	0.01	−0.27 (1.000)	−0.10 (1.000)	0.13 (1.000)
VAS lower limbs	58.21 ± 18.71	61.16 ± 22.22	55.00 ± 21.32	0.416 (2.54)	0.662	0.015	−0.14 (1.000)	0.16 (1.000)	0.27 (1.000)

Values are mean ± SD. RPE = rating of perceived exertion (Borg scale 1–10); VAS = visual analogue scale (0–100 mm); Bold values (e.g., <0.001) indicate statistical significance

Table 5. Repeated-measures ANOVA results for time, condition and time × condition effects on acid-base variables and blood lactate.

Outcome	Effect	F(df1. df2)	*p*	partial η^2
HCO_3	Time	2.93 (1.18)	0.104	0.14
	Condition	113.75 (2.36)	**<0.001**	0.86
	Time × Condition	594.75 (2.36)	**<0.001**	0.97
pH	Time	10.70 (1.18)	**0.004**	0.37
	Condition	35.25 (2.36)	**<0.001**	0.66
	Time × Condition	219.74 (2.36)	**<0.001**	0.92
LAC	Time	592.55 (1.18)	**<0.001**	0.97
	Condition	2.65 (2.36)	0.084	0.13
	Time × Condition	658.53 (2.36)	**<0.001**	0.97
ABE	Time	F(1.18) = 8.02	**0.011**	0.308
	Condition	F(2.18) = 82.55	**<0.001**	0.821
	Time × Condition	F(2.18) = 397.70	**<0.001**	0.957

Time = pre- vs post-test; Condition = control, placebo and $NaHCO_3$ *trials,* HCO_3^- *= blood bicarbonate; pH = blood pH; LAC = blood lactate concentration; ABE = actual base excess; Bold values (e.g., <0.001) indicate statistical significance*

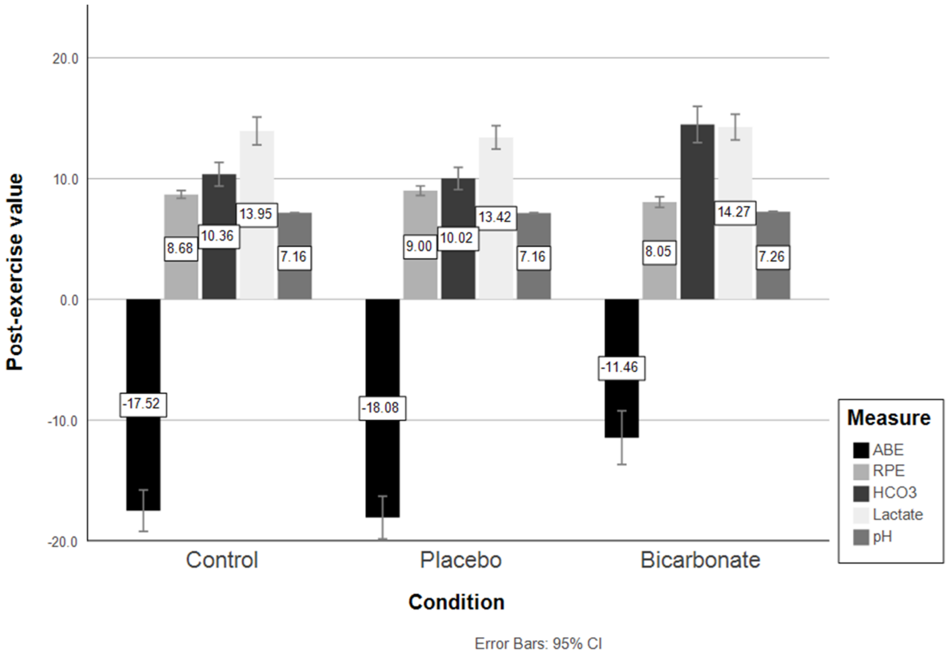

Figure 1. Post-exercise acid-base, blood lactate, and perceptual responses across experimental conditions.

Bars represent mean post-exercise values with 95% confidence interval error bars. ABE = actual base excess (mmol·L^{-1}*); RPE = rating of perceived exertion (Borg scale 1–10);* HCO_3^- *= blood bicarbonate (mmol·L*$^{-1}$*); Lactate = blood lactate concentration (mmol·L*$^{-1}$*); pH = venous blood pH*

Discussion

The aim of this study was to examine the acute ergogenic effects of sodium bicarbonate ($NaHCO_3$) supplementation on 200-m Bi-Fins performance and related biomechanical variables. Contrary to our hypothesis, ingestion of 300 $mg \cdot kg^{-1}$ $NaHCO_3$ did not improve overall race time, aligning with previous studies reporting inconsistent or null effects in middle-distance swimming (Gurton et al., 2025; Joyce et al., 2012). Our split-time analysis revealed a significant condition × section interaction, with slower 25- and 50-m splits following $NaHCO_3$ supplementation. Since no differences were observed in surface stroke mechanics (SR, SL, SI), these early decrements likely reflected either pacing adjustments or subtle alterations in start and underwater execution, which strongly shape early split performance in 25-m pools. In addition, acute alkalosis may transiently disrupt glycolytic flux through pH-sensitive enzymatic pathways (Lancha Junior et al., 2015), contributing to the slower initial pacing before compensation later in the race.

The effectiveness of $NaHCO_3$ supplementation appears highly dependent on methodological factors. Lindh et al. (2008) reported performance benefits with 14–21-day intervals between trials, which may have enabled training-related adaptations, whereas studies with tighter control of training load and shorter wash-out periods (Gurton et al., 2025; Joyce et al., 2012) observed no ergogenic effect. Considerable inter-individual variability in time to peak blood bicarbonate (TTP) further complicates interpretation (Farias De Oliveira et al., 2020). Because TTP was not individualized in our study, some trials may have occurred outside the optimal alkalosis window.

Despite the absence of performance benefits, systemic alkalosis was clearly induced, as shown by elevated plasma HCO_3^- (30,14 ± 1,82 $mmol \cdot L^{-1}$), increased pH (7.484), and an alkalotic shift in ABE (+6,47 ± 1,37 $mmol \cdot L^{-1}$). These values exceeded the commonly cited ergogenic threshold of +5–6 $mmol \cdot L^{-1}$ above baseline (Carr et al., 2011), confirming that the supplementation protocol successfully achieved bicarbonate saturation. Post-exercise metabolic acidosis developed across all conditions; however, the smaller decline in pH and ABE after $NaHCO_3$ supplementation indicates preserved buffering capacity (Chiron et al., 2024; McNaughton et al., 2008). Although this buffering advantage did not translate into improved single-bout performance in the present 25-m pool setting, it may hold greater relevance in competitive scenarios requiring repeated efforts (e.g., heats and finals), where faster restoration of acid-base balance could meaningfully influence performance stability and recovery (Gough et al., 2023; Gurton et al., 2024).

Despite confirmed alkalosis, post-test blood lactate was only slightly higher under the $NaHCO_3$ condition compared with the placebo condition (14.3 vs. 13.4 $mmol \cdot L^{-1}$; $p_{adj} = 0.030$), with a moderate ANOVA effect ($F(2,36) = 3.94$, $p = 0.028$; $\eta^2p = 0.18$). However, the absolute difference (<1 $mmol \cdot L^{-1}$) remained below the ≥2 $mmol \cdot L^{-1}$ threshold generally considered physiologically meaningful (Messonnier et al., 2007). This modest elevation contrasts with studies reporting more pronounced increases in blood lactate after $NaHCO_3$ ingestion (Gurton et al., 2025; Lindh et al., 2008), commonly attributed to enhanced lactate/H^+ efflux through monocarboxylate transporters (Bishop et al., 2004).

The limited impact of $NaHCO_3$ on lactate responses may be linked to the whole-body demands of Bi-Fins swimming, where extracellular buffering capacity must be distributed across multiple active muscle groups (Nakashima et al., 2019; Sostaric et al., 2006). Consistently, perceived muscle discomfort (VAS) did not differ among conditions, indicating that bicarbonate did not meaningfully alter subjective muscular strain (Yilmaz et al., 2026). The multi-segmental nature of Bi-Fins swimming may therefore attenuate localized bicarbonate-related effects that have been reported in disciplines with predominantly lower-limb involvement, such as monofin swimming (Kunitson et al., 2015).

Interestingly, athletes reported lower RPE values after $NaHCO_3$ supplementation, which is consistent with earlier findings (Gough et al., 2023; Gurton et al., 2025). In our study, RPEs were significantly reduced compared with the placebo condition ($p_{adj} = 0.002$; large effect) and showed a trend toward reduction compared with the control condition ($p_{adj} \approx 0.057$). This aligns with theories of centrally mediated fatigue modulation, whereby reduced intramuscular H^+ lowers afferent feedback from group III/IV muscle receptors, diminishing inhibitory input to the CNS and helping to sustain motor output (Gurton et al., 2023; Siegler et al., 2016). Although this perceptual advantage did not translate into acute performance improvement, it may enhance tolerance to high-intensity training sessions and facilitate greater training load over time—an aspect particularly relevant for multi-bout or tournament-style finswimming formats.

The stroke rate, length, and index were unaffected by the condition. The only split-level difference occurred at 150 m, where the SI was slightly higher following $NaHCO_3$ supplementation compared to the placebo condition (small effect; $g \approx 0.27$; $p_{adj} = 0.019$), but this pattern did not appear in other splits or in SR/SL. These findings, however, must be interpreted in the context of a 25-m pool, where CMAS rules permit up to 15 m underwater per length (Finswimming CMAS Rules, 2023), leaving limited surface-swimming distance for stroke mechanics to manifest and imposing considerable hypoxic and hypercapnic strain that may mask subtle technical effects of alkalosis (Michalica et al., 2024; Veiga et al., 2022). The combination of slower early splits and unchanged surface-mechanics variables indicates that start mechanics, underwater execution, and pacing strategy likely played a more decisive role than any direct effect on stroke technique. Future research should therefore quantify underwater performance both biomechanically (distance, speed, breakout quality) and physiologically (O_2 saturation, heart-rate dynamics), and examine 50-m pools, where reduced underwater contribution may increase sensitivity to $NaHCO_3$-related changes in stroke efficiency.

Conclusions

In summary, acute ingestion of 300 $mg \cdot kg^{-1}$ sodium bicarbonate induced clear systemic alkalosis but did not improve overall 200-m Bi-Fins performance. Supplementation was accompanied by slower early splits without corresponding changes in surface stroke mechanics, suggesting influences from pacing or underwater-phase execution rather than biomechanical disruption. Although buffering capacity was preserved and perceived exertion was reduced, these advantages did not translate into measurable performance gains in this single-bout, 25-m pool context. From an applied standpoint, sodium bicarbonate may still offer benefits in competitive settings involving repeated efforts (e.g., heats and finals), where faster restoration of acid-base balance and reduced perceptual load could support performance maintenance. Future research should also investigate whether $NaHCO_3$ supplementation-related effects become more pronounced in 50-m pools, where longer surface-swimming segments may increase sensitivity to technical or metabolic improvements.

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