THE EFFECTS OF TWO DIFFERENT RECOVERY PROTOCOLS ON THE 100-METER FRONT-CRAWL PERFORMANCE OF MALE SWIMMERS

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Abstract:

The aim of this study was to ascertain whether passive recovery with additional sodium hydrogen carbonate ($NaHCO_3^-$) ingestion affected 100-m swimming speed differently from active recovery during a simulation of a typical competition, which consisted of two trials with a 60-min recovery period. Nine male swimmers (mean age 20.9 ±1.6 years, stature 1.81 ± 8.5 m and body mass 78.1 ± 12.9 kg) completed two maximal 100-m front-crawl swims separated by a 60-min active recovery and two maximal 100-m front-crawl swims separated by a 60-min passive recovery with $NaHCO_3$ ingestion on a different test day. The active recovery protocol resulted in lower [LA] than the recovery with $NaHCO_3$ ingestion ($p<.05$), while pH, pCO_2 and [HCO_3^-] were higher in the recovery with $NaHCO_3$ ingestion protocol ($p<.01$). Neither protocol had a significant effect on 100-m swim time. In conclusion, a 60-min active swimming recovery at 65% of maximal velocity on the 100-m crawl or a passive recovery with $NaHCO_3$ intake in the same time interval had no significant effect on the second swimming performance, despite the different effects on pH, [HCO_3^-], pCO_2, and [LA]. In both experimental conditions, no differences in swimming time were found between the first and the second swim.

Keywords*: blood, pH, [HCO_3^-], lactate, $NaHCO_3$ ingestion*

Introduction

International swimming competitions usually extend over several days and include multiple disciplines, each requiring an efficient recovery period for competitors who qualify for the semi-finals and finals. Competitive pool events include races from 50 up to 1,500 m in the time range of about 20 s to 15 min. The corresponding swimming speed decreases inversely hyperbolically with distance (Hill, 1925).

The relative contribution of energy systems during swimming at maximal speeds varies according to the swimming distance. In the shortest distances (50m and 100m), there is a predominance of a combination of phosphate energy and anaerobic glycolysis, while in longer distances, aerobic combustion of carbohydrates, fats, and proteins prevails (Capelli, Termin & Pendergast, 1998). The specific contributions of these systems depend on both the length of the race and the intensity of the pace used.

Anaerobic glycolitic processes typically produce hydrogen ions (H^+), which strongly influence acid-base and electrolyte balance in muscle cells and various other compartments, particularly in the blood (Cairns & Lindinger, 2025; Keyser, 2010). These ions also contribute to or are associated with several causes of fatigue (Cairns & Lindinger, 2025; Keyser, 2010; Lindinger & Heigenhauser, 1991; Sundberg & Fitts, 2019).

The organism needs to activate different buffer systems to counteract acidosis from the cell to the blood compartments, with the strongest blood buffer system being hydrogen carbonate (HCO_3^-) (Kraut & Madias, 2010).

The assumption that ingestion of sodium hydrogen carbonate ($NaHCO_3$) could enhance performance has been widely studied and tested in swimming (Grgic & Mikulic, 2021; Katz, Costill, King, Hargreaves & Fink, 1984; Lindh, Peyrebrune,Ingham, Bailey & Folland, 2008; Mero, et al., 2013; Ušaj, Marčun & Štrumbelj, 2024; Zajac, Cholewa, Poprzecki, Waśkiewicz, & Langfort, 2009) and other types of exercise (Carr, Hopkins & Gore, 2011; Driller, Williams, Gregory & Felt, 2013; Higgins, James & Price, 2013; Horswill, et al., 1988). The aim of this intervention was to potentially help swimmers achieve faster times by compensating for increased acidosis during swimming. However, previous studies have not consist-

ently confirmed this hypothesis. While some studies have reported improvements in swimming performance (Lindh, et al., 2008; Mero, et al., 2013; Zajac, et al., 2009), others have not supported this idea (Grgic & Mikulic, 2021; Ušaj, et al., 2024).

Given the challenge for swimmers to optimize recovery between two races on the same competition day—often spaced about 60 minutes apart or even less—the idea of $NaHCO_3$ ingestion, among other strategies, was considered (Grgic & Mikulic, 2021; Lindh, et al., 2008; Mero, et al., 2013; Ušaj, Marčun & Štrumbelj, 2024; Zajac, et al., 2009).

There are four known possible recovery practices utilized between races. The first is rest without any active recovery (Toubekis, Smilios, Bogdanis, Mavridis & Tokmadikis, 2006); the second is active recovery on land consisting of low-intensity exercises to remove metabolites (mainly stretching) (Dalamitros, et al., 2018); the third is lactate removal via swimming at a low intensity (Lomax, 2012; Toubekis, et al., 2006); and the fourth is the oral ingestion of sodium bicarbonate to eliminate excessive acidosis (Ušaj, et al., 2024).

A study on the different recovery protocols in swimming suggested that it did not matter whether a self-paced, continuous, steady-rate swimming velocity or swimming recovery consisting of various strokes, intensities, and rest intervals had been adopted as a recovery method. Since both forms of swimming recovery methods reduced more blood lactate than the land-based recovery method, swimmers should be advised to undertake a swimming-based recovery method rather than a land-based recovery method (Lomax, 2012).

Swimming research that focused on passive, out-of-water vs. recovery swimming (active) determined that active recovery was better at reducing blood lactate levels than passive, out-of-water recovery (Greenwood, Moses, Bernardino, Gaesser & Weltman 2008; Neric, Beam, Brown & Wiersma, 2009).

Despite the relatively long recovery period, during which acidosis and metabolites can be largely cleared, passive recovery alone was not considered as a reference test. Instead, low-intensity swimming (Lomax, 2012) and $NaHCO_3$ supplementation (Lindh, et al., 2008; Mero, et al., 2013; Zajac et al., 2009) were examined as promising recovery strategies.

The effects of $NaHCO_3$ ingestion on exercise performance have been extensively studied. A meta-analysis (Carr, et al., 2011) suggests that a dose of 0.3 g/kg of body mass $NaHCO_3$ results in a 1.7 ± 2.0% improvement in exercise performance. However, the evidence remains inconclusive, as several studies have found no effect on physical performance impaired by muscle acidosis (Carr, et al., 2011; Driller, et al., 2013; Grgic & Mikulic, 2021; Higgins, et al., 2013; Horswill, et al., 1988; Mero, et al., 2013; Ušaj, et al., 2024). The findings of Saunders, Sale, Harris, and Saunderland (2014), in which $NaHCO_3$ was administered, indicate that this intervention does not consistently enhance performance in the same individual. This variability highlights the need for caution when interpreting results from a single study. Given these findings, practitioners should conduct repeated tests of $NaHCO_3$ ingestion strategies to differentiate between non-responders and potential responders (Saunders, et al., 2014).

$NaHCO_3$ supplementation in swimmers has been shown to increase [HCO_3^-] and blood pH, which has been associated with improved performance in 50 m (Zajac, et al., 2009), 200 m (Lindh, et al., 2008), and consecutive sets of 100-m freestyle swimming (Mero, et al., 2013), as well as reduced average swim time in a set of four repetitions of 50-m freestyle swimming (Zajac et al., 2009).

The aim of this study was to ascertain whether passive recovery with additional sodium hydrogen carbonate ($NaHCO_3^-$) ingestion affected 100-m swimming speed differently from active recovery during a simulation of a typical competition, which consisted of two trials with a 60-min recovery period.

Materials and methods

Subjects

Nine male subjects (age: 20.9 ± 1.6 years, height: 1.81 ± 8.5 m, body mass: 78.1 ± 12.9 kg), members of two national swimming clubs, voluntarily participated in the study after being informed both verbally and in writing about its nature and risks. The swimmers had varying levels of competitive ability and competition history; however, all had at least eight years of competitive experience. At the time of the study, all swimmers were in the middle of their competitive season, training seven times per week, including three dryland training sessions. All participants signed an informed consent form. The study was approved by the National Medical Ethics Committee of the Republic of Slovenia and complied with the principles of the Declaration of Helsinki (Ethical Code Number: 78/12/13).

Procedures

The two experimental sessions were separated by five days.

The sprint trials were conducted in a 25-m indoor swimming pool (water temperature was 27 degrees Celsius, average relative humidity was maintained at 50-55%). All tests were conducted between 9:00 and 11:00 a.m. First, each swimmer completed a standard warm-up session of mixed swimming drills (200-m front crawl, 200-m as 50-m swim–kick–pull–swim, 200 m as 50-m individual medley and 100-m easy swim). The swim-

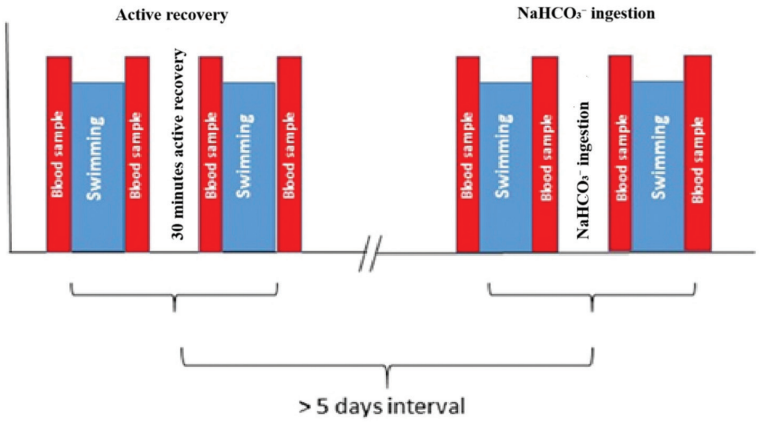

Fig. 1. Design of the experiment.

mers were then assigned to adjacent lanes and then completed 100-m front-crawl sprints with maximum effort. Each sprint was measured individually by two investigators using a standard stopwatch and then recorded. The average times recorded and frequency rate by both examiners were included in the analysis of the results. Both investigators have more than 20 years of experience in swimming research. Although hand timing has been shown to underestimate times compared to electronic timing, the mean differences are consistent and within half a second with good reliability. After each 100-m sprint in both protocols, the swimmers had 60 minutes recovery time before starting the next performance.

Active recovery consisted of 30 minutes of swimming at a speed of 65% achieved during the first lap of the 100-m crawl. The reason for choosing a 65% speed for the 100-m swim for active recovery was the finding that intensities corresponding to 50-60% speeds of the 100-m lap during repeated sprints effectively reduce the swimmer's performance (Toubekis, et al., 2006; Toubekis, Douda & Tokmakidis, 2005).

During passive recovery, a drink in the form of $NaHCO_3$ solution (Lekarna Ljubljana, Slovenia) dissolved in 500 mL of water was taken 15 minutes after the first trial. A standard dose of 0.3 $g \cdot kg^{-1}$ of body mass was used for a single swimmer according to their body mass.

In both experimental cases, the swimmers spent the remaining 30 minutes before the second performance sitting in the stands. Before the second performance, they also performed basic dry-land warm-up exercises.

Blood parameter analysis

Blood samples before and after swimming (obtained in the 1st minute after each trial) were collected at the poolside and kept on ice for 20 minutes until analysis in the laboratory. All blood samples were taken from the hyperaemic earlobe. Whole blood samples were collected in 80 µL blood gas capillary tubes and stored on ice until acid-base balance analysis (pH, $[HCO_3^-]$, pCO and BE) was performed using a blood gas analyser (ABL5, Radiometer, Copenhagen, Denmark). The blood lactate samples were analysed with the LP20 drLANGE photometer (Burladingen, Germany).

Statistical analysis

Four data sets collected during the first and second active recovery 100-m tests and during the first and second $NaHCO_3^-$ 100-m tests (Fig. 1) were first tested for normality using the Shapiro-Wilkinson test. The data sets were then compared for differences in front-crawl times, stroke frequency, blood acid-base parameters (pH, pCO_2,$[HCO_3]$, base excess (BE) and [LA]).

The swimming times were compared by the two-factor ANOVA where the comparison between active swimming and $NaHCO_3$ ingestion (RECOVERY as a first factor) and between the 1st and 2nd swims (DELTA as the second factor) was conducted. The alterations of pH (ΔpH), $[HCO_3^-]$ ($\Delta[HCO_3^-]$), pCO_2 (Δ (pCO_2) and [LA] (Δ[LA]) were compared using another two-factor ANOVA, where the comparisons between active swimming and $NaHCO_3$ ingestion (RECOVERY) represented the first factor and ΔpH alterations; $\Delta[HCO_3^-]$, Δ (pCO_2) and Δ[LA] as differences between the 1st and 2nd swims (DELTA) represented the second factor. In all comparisons, a p-value of $<.05$ was considered significant. We estimated that a sample size of nine swimmers per group would be sufficient to obtain statistical power ≥ 0.70 for the assessment of the alterations following the intervention.

Additionally, to ascertain the effect of $NaHCO_3$ ingestion on the blood pH, $[HCO_3^-]$, and [LA], values of pre-swimming for the 1st and 2nd swims and values of post-swimming for the 1st and 2nd swims were compared by paired t-tests. Statistical analyses were completed using SPSS 20 Software (Inc., Chicago, IL, USA).

Results

In both sets of trials of the two repeated 100-m crawl swims, the first with active recovery and the second with passive recovery and $NaHCO_3$ ingestion, the four swim times (in seconds) were similar for the factors RECOVERY ($F = 0.02$; $p = .89$) and DELTA ($F = 0.11$; $p = .74$), and $F = 0.00$; $p = .97$ for their interactions (Table 1). Therefore, $NaHCO_3$ ingestion did not affect swim time in a way that differed from active recovery. No significant differences in the average stroke frequency between four individual swims were found.

Active recovery had no effect on a significant alteration in blood pH and $[HCO_3^-]$, while [LA] decreased ($p < .01$) (Table 2). In contrast, $NaHCO_3$ ingestion increased pH and $[HCO_3^-]$ ($p < .01$) PRE 2 100-m swim, while [LA] remained unchanged during passive recovery (Table 2). POST-PRE differences were calculated to determine possible changes influenced by swimming or $NaHCO_3$ ingestion during recovery intervals. Calculations of the two factors ANOVA for ΔpH revealed no significant differences for the RECOVERY factor ($F = 1.55$; $p = .22$), the DELTA factor ($F = 2.01$; $p = .17$), and the interaction between them ($F = 0.31$; $p = .58$). $\Delta[HCO_3]$ showed no significant differences for the factor RECOVERY ($F = 4.05$; $p = .06$), the factor DELTA ($F = 0.01$; $p = .92$), and the interaction between them ($F = 1.11$; $p = .30$). Compared to the values of the other three 100-m tests, the pCO_2 post value in the blood with $NaHCO_3^-$ ingestion showed a clear tendency to increase by approximately 0.3 kPa ($F = 1.056$; $p = .381$) (Table 2.). However, comparison of the differences in pCO_2 between the two 100-m tests with active recovery (ΔpCO_2 RECOVERY) with the differences in pCO_2post between the two 100-m tests with $NaHCO_3^-$ingestion ($\Delta pCO2NaHCO_3$) revealed a significant change ($t = -2.984$; $p = .011$). The enhancement of Δ[LA] showed a significant difference for the factor DELTA $F = 4.99$, $p = .03$; while the difference for the factor RECOVERY was not significant. There were no interactions between the two factors. Overall, ΔpH and $\Delta[HCO_3^-]$ were not differentially affected by recovery swimming or NaHCO3 intake during the second 100-m crawl, while Δ[LA] were larger due to active recovery (Table 2).

Discussion and conclusion

Our study investigated the effects of two recovery protocols on swimming performance during a simulated competition day, which required swimmers to complete multiple trials within the same day. Indeed, the recovery interval of one hour or less may be important for maintaining or even improving performance during successive swims. The results showed that neither active recovery with moderate swimming intensity nor consuming 0.3 $g \cdot kg^{-1}$ of sodium hydrogen carbonate ($NaHCO_3$)

Table 1. Basic physical characteristics of 100-m swimming trials with thte selected recovery protocol

	Active recovery trials		$NaHCO_3$ trials	
	Trial 1	Trial 2	Trial 1	Trial 2_{NaHCO3}
Time 100m (s)	57.9±3.15	58.24±3.2	57.73±2.85	58.13±2.98
f strokes (min^{-1})	46.9±2.4	46.2±2.8	46.7±2.3	45.9±2.7

Note. * – p<.05, different from other trials (linear mixed-effects model), f strokes – strokes frequency.

Table 2. Blood gas, acid-base, and lactate values of 100-m swimming trials

	Active recovery trials		$NaHCO_3^-$ trials	
	Trial 1	Trial 2	Trial 1	Trial 2_{NaHCO3}
pCO_2 pre (kPa)	5.3±0.5	5.2±0.3	5.2±0.4	5.6±0.3*
pCO_2post (kPa)	5.1±0.6	5.2±0.4	5.0±0.5	5.5±0.4*
pHpre	7.40±0.03	7.40±0.01	7.43±0.02	7.50±0.02*
pHpost	7.19±0.04	7.22±0.06	7.23±0.05	7.33±0.02*
[]pre (mmol·L-1)	23.9±1.4	23.6±1.6	25.4±1.3	31.9±0.8*
[]post (mmol·L-1)	14.3±2.1	15.3±2.1	15.1±1.4	20.9±0.8*
BEpre	-0.3±1.5	-0.9±1.5	1.4±1.2	8.1±0.6*
BEpost	-12.6±2.6	-11.1±2.6	-11±1.8	-4.3±0.7*
[LA]pre (mmol·L-1)	2.0±0.6	1.7±0.5	2.0±0.6	2.3±0.4
[LA]post (mmol·L-1)	15.8±2.9	12.6±2.2	14.5±3.4	14.4±1.7

Note. * – p<.05, different from other trials (linear mixed-effects model)

affected the second maximal swimming velocity in a way to be higher than the previous one. Active swimming recovery may reduce [LA] more than passive recovery with $NaHCO_3$ ingestion, but without significant influence on subsequent swimming performance. In contrast, $NaHCO_3$ ingestion influenced the typical alkaline response with an increase in blood pH and [HCO_3^-] in addition to a reduction in [LA], but again swimming velocity was not affected.

During the recovery phase between two 100-m swims, which lasts about half hour, as is the case on a typical competition day, the organism tries to restore homeostasis. According to the predominant energetic processes during the 100-m swim, the accumulation of metabolic products such as hydrogen ions (H^+) and free phosphate (Pi) seems to play an important role in the development of fatigue. Therefore, restoring acid-base and electrolyte balance during the recovery interval seems to be very important to reduce fatigue and restore performance. We have assumed that one hour of even passive recovery will allow for a complete rebalancing of acid-base status. The only possible reason for the consumption of $NaHCO_3$ could therefore be the possibility of an increased hydrogen carbonate buffering capacity in the blood (Kraut & Madias, 2010). This could provide some advantage in buffering metabolic acidosis during the final 100-metre swim and consequently improve performance. In the study by Katz et al. (1984) it was concluded that the ingestion of $NaHCO_3$ prior to exercise does not help maintain an intense workload, possibly because it is unable to raise the intracellular pH or alkaline reserve of the muscle. Indeed, the increased blood alkalosis must be transferred to the intramuscular space to allow an effect on muscle performance. The study conducted by Horswill et al. (1988) concluded that incremental $NaHCO_3$ doses of 0.20 $g.kg^{-1}$ and less resulted in a gradual increase in blood [HCO_3^-] levels, but did not improve performance in a 2-minute sprint for cyclists. The results of another study also indicate that $NaHCO_3$, administered at the same dosage as in our study, has no ergogenic benefit in performances of 10 s or 30 s duration, whereas in activities of 120 s and 240 s duration, performance was significantly higher after $NaHCO_3$ ingestion than in the control and placebo conditions (McNaughton, 1992). On the other hand, a study by Lindh et al. (2008) concluded that $NaHCO_3$ supplementation could improve the 200-m freestyle performance time of elite male competitors, most likely by increasing the buffering capacity of the blood. Zajac et al. (2009) also demonstrated that $NaHCO_3$ ingestion is an effective buffer in adolescent athletes during high-intensity interval swimming and suggested that such a procedure could be used in adolescent athletes to increase training intensity as well as swimming performance during competitions over the 50- to 200-m distance. In a similar study (Mero, et al., 2013), where the time between two maximal swimming trials was 12 minutes, it was found that the swim time on the second attempt was improved significantly after acute $NaHCO_3$ supplementation compared to the placebo group. However, in both groups, the second swimming trial were slower than the first. Gough, Newbury, and Price (2023) demonstrated that ingesting 0.3 $g{\cdot}kg^{-1}$ of sodium bicarbonate improved performance in the latter stages of repeated 50-m sprint-interval swimming, likely due to enhanced extracellular buffering capacity. Finally, a review of nine selected studies on the ergogenic effects of $NaHCO_3$ supplementation on swimming performance concluded that $NaHCO_3$ intake improved performance in 200-m and 400-m swimming events. However, there was no significant difference between placebo and $NaHCO_3$ when data from all included studies on 91.4 m and 100-m swimming distances were considered (Grgic & Mikulic, 2021). Newbury, Cole, Kelly, and Gough (2024) found that neither individualised nor standardised sodium bicarbonate supplementation improved performance in repeated high-intensity swimming or a subsequent 200-m time trial in highly trained female swimmers. Domínguez et al. (2025) reported in a review that, unlike creatine, sodium bicarbonate supplementation did not show significant pooled effects on swimming performance, highlighting the need for further high-quality, swimming-specific research on its ergogenic potential. The results of our study also showed no significant influence on performance, despite a significant increase in blood $NaHCO_3$ and pH.

One of the reasons for the different results is the $NaHCO_3$ dosing protocol chosen. A dose in the form of a powder dissolved in water can directly influence the change in acid-base balance in the digestive flora. This was observed in our subjects, three of whom showed signs of indigestion in the form of stomach cramps and diarrhea. The side effects can be limited by a different dosing method. In the study by Saunders et al. (2014), subjects took part of the dose of sodium hydrogen carbonate (0.2 $g{\cdot}kg^{-1}$ body mass) in the form of gelatin capsules with water) with a predominantly carbohydrate breakfast. Another part of the dose (0.1 $g{\cdot}kg^{-1}$ body mass in the form of gelatine capsules) was taken two hours before the test, which further reduced the occurrence of gastrointestinal problems before and during the trials.

The findings of Ušaj et al. (2024) further support our results regarding the lack of ergogenic effects of $NaHCO_3$ supplementation on swimming performance. In their study examining the effects of sodium hydrogen carbonate ingestion during recovery between two 200-m front-crawl time trials, they found no significant improvement in

swimming performance despite observing expected physiological changes in acid-base status and pCO_2. Their research used a similar protocol with $NaHCO_3$ ingestion (0.3 g·kg^{-1} body mass) during a 60-minute recovery period between maximal swimming efforts. Importantly, their study extended the investigation to a longer swimming distance (200 m), which theoretically should be more responsive to bicarbonate supplementation due to greater contribution of anaerobic glycolysis and subsequent acidosis. Despite this theoretical advantage, no performance benefits were observed, which aligns with our findings for 100-m swimming and suggests that the lack of ergogenic effect may be consistent across different sprint swimming distances. This consistency across studies using similar methodologies but different swimming distances strengthens the evidence that $NaHCO_3$ supplementation during recovery periods may not provide the expected performance benefits in competitive swimming scenarios. The reason could be, according to the results in our and Ušaj's study, that the pCO_2 levels in both studies increased towards hypercapnic values, which additionally stimulates breathing. This is already restricted during the maximum front-crawl and cannot be increased by increasing the breathing rate, as the mechanical work of the respiratory muscles increases considerably. Thus, the increase in pCO_2 becomes an important limiting factor that could have a negative impact on performance, in contrast to what would be expected with the $NaHCO_3$ ingestion.

What was the reason for the active recovery (swimming at 65% of maximal velocity during 100-m) is not clear. One possible explanation is a faster and greater decrease in [LA] due to the use of lactate as fuel for aerobic processes (Greenwood, et al., 2008; McMaster, Stoddard & Duncan, 1989; Toubekis, et al., 2005). Lactate has not been identified as a source of fatigue during exercise (Brooks, 2001). Nevertheless, this type of active recovery is a common practice in swimming. McMaster et al. (1989) showed that 15 min of swimming between 55-75% of competitive speed after a 200-metre competitive swim resulted in a return of [LA] to baseline (~2 mmol·L^{-1}). Greenwood et al. (2008) demonstrated that active recovery at the speed associated with the lactate threshold was more effective in lowering [La] than speeds above or below the lactate threshold. However, despite the clear finding that active recovery by swimming reduced [LA] faster than passive recovery, no effect on swimming velocity or performance was reported. Faghy, Lomax, and Brown (2019) showed that physiologically guided active recovery, such as swimming at the lactate threshold velocity, enhanced lactate clearance in elite swimmers, which may complement strategies like sodium bicarbonate supplementation to manage acidosis during repeated high-intensity efforts.

Due to uncertainty what the real causes of fatigue are in the 100-m swim and which strategy is successful in reducing fatigue during recovery between two swims, we tested both strategies to find the most efficient one. Our results show that the active recovery strategy was not successful in increasing maximal swimming performance during the second (final) trial when simulating a day of competition consisting of a semi-final and a final. Although this strategy reduced the [LA] before the second 100-metre swim, this goal can also be achieved with only passive recovery. Even when the active recovery strategy [LA] resulted in the smaller values, the difference between this and passive recovery with $NaHCO_3$ ingestion was too small to have any significant physiological significance for the 100-m swim.

The competition schedule at swimming events often requires swimmers to complete multiple disciplines in one part of the competition, with short breaks in between. It is believed that the main aim of this break is to reduce or eliminate the severe metabolic acidosis caused by the accumulation of H^+ ions. Therefore, increasing the capacity of the hydrogen carbonate system during the recovery phase could be an important goal of $NaHCO_3$ intake, which subsequently improves swimming performance by enhancing blood alkalosis. Unfortunately, this idea was only partially realized in our study. It can also be gleaned from about half of the reference literature used in this study. Instead of increasing blood pH and [HCO_3^-], swimming performance was not changed. The other idea, that active, low velocity swimming during recovery would reduce the volume of lactate in the blood, did not increase performance.

To solve the main limitation: reaching maximal effort during the two swimming events, we propose the simulation of an official competition outside the official national competitions. This could not only generate adequate motivation for maximum effort, but also the necessary intensity of anxiety before the trials. In future research, the results of the study could be improved by including larger samples, using more precise electronic timing, implementing strict pre-test dietary and hydration controls, and employing more tolerable forms of $NaHCO_3$ administration to minimize gastrointestinal discomfort.

In conclusion, a 60-min active swimming recovery at 65% of maximal velocity on the 100-m crawl or a passive recovery with $NaHCO_3$ intake in the same time interval had no significant effect on the second swimming performance, despite the different effects on pH, [HCO_3^-], pCO_2 and [LA]. In both experimental conditions, no differences in swimming time were found between the first and the second swim.

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