Effect of sodium bicarbonate ingestion on measures of football performance - with reference to the impact of training status

Ávirkanin av natriumbikarbonat inntøku á mát fyri kropslig fótbóltsavrik við støði í árinunum av kropsligari venjingarstøðu

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Abstract

Purpose: To investigate effects of acute sodium bicarbonate ($NaHCO_3$) ingestion on performance during a football-specific protocol, with reference to mediating effects of intermittent training status. **Methods**: Ten male university football players (age: 21±1 yrs, height: 180±2 cm, mass: 78.8±2.8 kg) completed two six-a-side football matches after ingesting either 0.4 $g \cdot kg^{-1}$ $NaHCO_3$ (staggered over 90-min) or no supplement (CON) in a randomised, counterbalanced order. Activity profiles, blood [lactate], HR and gastro-intestinal distress determined throughout. Training status was evaluated using Yo-YoIR1, Yo-YoIR2 and repeated-sprint tests. **Results:** Players performed 70.3% more high-speed running (17-21 $km \cdot h^{-1}$) during 0-5min following $NaHCO_3$ ingestion vs. CON (17.9±5.2 vs. 10.5±3.1 m, $P<0.05$). No other significant activity profile differences, including high-intensity running (HIR; $>14km \cdot hr^{-1}$), existed between conditions during any period ($P>0.05$). However, total HIR increased for 70% of participants following $NaHCO_3$ vs. CON ($P>0.05$). Large-very large correlations ($0.5<r<0.9$) existed between training status measures and HIR improvement from CON to $NaHCO_3$ during certain match periods ($P<0.05$). **Conclusions:** Acute $NaHCO_3$ ingestion appears ergogenic for some, but not all, football performance measures. Furthermore, individual variability in HIR response suggests this ergogenic potential is not realised by everyone. Correlational analysis suggests higher intermittent training status may potentiate $NaHCO_3$ efficacy.

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Úrtak

Endamál: At kanna ávirkanina av natriumbikarbonat ($NaHCO_3$) inntøku á kropslig avrik í fótbólti, umframt árinini av kropsligari venjingarstøðu. **Mannagongd**: Tíggju mannligir fótboltsspælarar (aldur: 21±1 ár, hædd: 180±2 cm, vekt: 78,8±2,8 kg) spældu tveir fótbóltsdystir (6 ímóti 6) eftir inntøku av antin 0,4 $g \cdot kg^{-1}$ $NaHCO_3$ (inntikið í tablettformi 90-60 min áðrenn dystirnar) ella uttan inntøku av $NaHCO_3$ (CON). Inntøkan av $NaHCO_3$ var skipað tilvildarliga í dysti 1 og 2. Rennimynstur, blóðmjólkasýra, pulsur og neilig árin á maga-tarm skipanina var mátað undir dystunum. Kropsliga venjingarstøðan var mátað við Yo-Yo IR1, Yo-Yo IR2 og skjótleikatestum. **Úrslit:** Spælarar runnu 70,3% longri við høgari renniferð (17-21 $km \cdot t^{-1}$) í fyrstu 5 min av dystunum eftir $NaHCO_3$ inntøku vs. CON (17,9±5,2 vs. 10,5±3,1 m, P<0,05). Eingin annar signifikantur munur var á skjótari renning (øll renning >14 $km \cdot t^{-1}$) ímillum $NaHCO_3$ og CON (P>0,05), hóast samlaða nøgdin á skjótari renning øktist hjá 70% av leikarunum í $NaHCO_3$ vs. CON (P>0,05). Stórar og sera-stórar korrelatiónir (0,5<r<0.9) vóru ímillum kropsliga venjingarstøðu og framgongd í skjótari renning frá CON til $NaHCO_3$ í serstøkum dystartíðarbilum (P<0,05). **Niðurstøða:** Inntøka av $NaHCO_3$ stimbrar nøkur mát, men ikki øll, fyri kropslig avrik í fótbóltsdystum. Stórir individuellir munir eru á, hvussu stór henda ávirkan er. Korrelatiónsanalysan vísti, at jú betri kropsliga venjingarstøðan er, betri er ávirkanin av $NaHCO_3$ inntøku á kropsligu avrikini.

Keywords: activity profiles, alkalosis, fatigue, intermittent exercise, training status.

Abbreviations

ANOVA:	Analysis of variance
ATP:	Adenosine-tri-phosphate
Ca^{2+}:	Calcium ions
FSG:	Full-Sized Game
GI:	Gastro-Intestinal
GPS:	Global Positioning System
H^+:	Hydrogen ions
HCO^-_3:	Bicarbonate
HIR:	High Intensity Running (>15 $km \cdot h^{-1}$)
HR:	Heart Rate
K^+:	Potassium ions
K_{ATP}:	ATP sensitive potassium channels
LSD:	Least Significant Difference
Na^+:	Sodium ions
$NaHCO_3$:	Sodium bicarbonate
PFI:	Permanent Fatigue Index
PFK:	Phospho-fructo-kinase
RST:	Repeated-Sprint-Test
RPE:	Rate of Perceived Exertion
SEM:	Standard Error of the Mean
SSG:	Small-Sided-Games
TFI:	Temporary Fatigue Index
VAS:	Visual Analogue Scales
Yo-Yo IR:	Yo-Yo Intermittent Recovery test

Introduction

Football is characterised by prolonged high-intensity intermittent exercise (Bangsbo *et al.*, 1991). The ability to repeatedly perform and recover from high-intensity activities is a key performance determinant; validly evaluated using high-intensity running (HIR) distance during match-play, which correlates with performance quality during full- (Mohr *et al.*, 2003) and small-sided games (Dellal *et al.*, 2011). However, this ability declines temporarily during, and towards the end of a game (Mohr *et al.*, 2003). Metabolic perturbations resulting from extensive anaer-

obic energy turnover have been implicated in this match-related fatigue (Bangsbo *et al.*, 2006).

Temporary fatigue, as demonstrated by ~50% reduction in HIR in the 5-min following the most intense 5-min period (Mohr *et al.*, 2005), negatively affects tactical (Bradley *et al.*, 2009), technical (Lyons *et al.*, 2006), and repeated-sprint (Krustrup *et al.*, 2006a) abilities. The aetiology of temporary fatigue is complex (Rampinini *et al.*, 2008), but appears unrelated to muscle [phosphocreatine], [ATP], [glycogen] and [lactate] (Mohr *et al.*, 2005). Instead, interstitial potassium (K^+) accumulation, and concomitant muscle cell excitability disturbances appear heavily implicated (Clausen, 2003; Mohr *et al.*, 2005). Furthermore, muscle pH transiently declines below 7.0 during match-play (Krustrup *et al.*, 2006a). Although dismissed as a direct contributor to temporary fatigue development per se (Krustrup *et al.*, 2006a), intra muscular hydrogen ion (H^+) accumulation opens ATP-sensitive K^+ (K_{ATP}) channels, thus potentiating the aforementioned exercise-induced rise in interstitial [K^+], and reducing muscle function (Davies, 1990). Moreover, intramuscular acidosis is also associated with impaired Ca^{2+} release and binding during actomyosin cross-bridge formation, impaired glycolytic enzyme activity (e.g. PFK) and accelerated central fatigue development (Cairns, 2006); all of which are detrimental to high-intensity exercise performance. Accordingly, improving pH (and H^+) regulation during match-play could attenuate these deleterious effects, including match-induced hyperkalaemia, and thus ameliorate temporary fatigue resistance to increase overall HIR, and ultimately improve performance. Sodium bicarbonate ($NaHCO_3$), an alkalinizing agent, may be beneficial in this regard (Peart *et al.*, 2013).

Permitted by the World Anti-Doping Agency and FIFA (Dvorak *et al.*, 2006), $NaHCO_3$ has several mechanisms that may attenuate temporary fatigue development during football match-play. Firstly, $NaHCO_3$ ingestion elevates blood [bicarbonate] (HCO_{3-}) ~2.6-7.3mmol.L^{-1} following 0.3-0.4 g·kg^{-1} body mass dose (Siegler *et al.*, 2010; Krustrup *et al.*, 2015). HCO_3^- provides the predominant extracellular buffering mechanism, sequestering excess H^+ (Bishop, 2010). Accordingly, $NaHCO_3$ ingestion enhances extracellular, as opposed to intracellular, H^+ buffer capacity (Hollidge-Horvat *et al.*, 2000). Specifically, the increased [HCO_3^-] and concomitant reduced extracellular [H^+] creates a large muscle-to-blood [H^+] gradient (Street *et al.*, 2005), which facilitates H^+ efflux from exercising muscle, thus attenuating exercise-induced muscular pH. Therefore, $NaHCO_3$ ingestion can attenuate aforementioned deleterious effects associated with intramuscular acidosis, which also is suggested by recent studies (Marriott *et al.*, 2015; Krustrup *et al.*, 2015). However, it is unknown, how $NaHCO_3$ ingestion affects football match performance.

Few studies have investigated intermittent team-sport players (e.g. football (De Ste Croix & Pope, 2006)), however, their protocols fail to elicit activity profiles and metabolic responses specific to football match-play. Likewise, no study has

used actual match-play as its protocol; problematic due to the task-specific nature of fatigue (Enoka & Stuart, 1992). Therefore, limited ecological validity of previous studies reduces practical applicability of results to football match-play, meaning any potential ergogenic effects of $NaHCO_3$ for football match-play remain to be determined. Furthermore, to the author's knowledge, no research has investigated the effect of intermittent training status on $NaHCO_3$ efficacy; highlighting two gaps in our current knowledge. Such research would allow individual recommendations regarding $NaHCO_3$ efficacy to be made specifically for football players based on football-specific evidence; important because supplement use in football often lacks scientific backing (Taioli, 2007). Thus, the purpose of the current study was to investigate the potential ergogenic effects of acute $NaHCO_3$ ingestion during a protocol specific to football match-play, with reference to the mediating effects of training status; research that is currently unavailable. Specifically, small-sided games (SSGs) were used to elicit match-specific metabolic responses to test the following experimental hypotheses: 1) $NaHCO_3$ will affect distances covered in various activity categories; 2) $NaHCO_3$ will increase match performance as measured by HIR; 3) $NaHCO_3$ efficacy will be mediated by participant training status.

Methods

Participants

Ten male university football players familiar with intense intermittent exercise participated in the study (age: 21±1 years, height: 180±2 cm, body mass: 79±3 kg). Players were fully informed of the experimental protocol, and risks (e.g. GI distress) and benefits associated with participation before freely providing written consent. The study was approved by the University of Exeter Research Ethics Committee.

Experimental design

A fully repeated-measures design was used (condition x time). Participants completed a familiarisation trial, a Yo-Yo Intermittent Recovery test level 1 (Yo-Yo IR1) and level 2 (Yo-Yo IR2) and a repeated-sprint test (RST) separated by ≥48-h to determine intermittent training status. Participants subsequently completed two 6-a-side football matches under two conditions ($NaHCO_3$ and control) in a randomised, counterbalanced, cross-over manner, separated by five days. Activity profile, blood [lactate], heart rate (HR), Gastro-Intestinal (GI) distress and Rate of Perceived Exertion (RPE) data collected throughout match-play. Artificial grass used throughout to ensure standardised surface conditions.

Preparation procedures

Players instructed to refrain from: caffeine (24-h before), strenuous activity (48-h before), alcohol (48-h before), other supplementation (throughout). Prior to all testing sessions, players were fitted with a Global Positioning System (GPS) unit (GPS Sports Systems, SPI Pro X, Australia) and HR chest belt (Polar heart rate monitor, Polar Electro, Finland) to collect activity patterns and HR data, respectively.

Food and fluid intake (type, vol-

ume/mass) recorded prior to match one, and replicated prior to match two to prevent diet-induced changes in acid-base balance (Greenhaff *et al.*, 1988). Participants reported to the football pitch 100-min prior to kick-off to complete supplementation. Both matches played at 1.30 pm; avoiding circadian variation (Reilly, 1986). The same GPS unit was worn by the individual player for both matches to minimise inter-device measurement error (Jennings *et al.*, 2010).

Experimental protocol

Familiarisation. Before testing, participants completed a familiarisation of each performance test to increase test reproducibility (Bangsbo and Mohr, 2012). Body mass (Seca digital scale column SEC-170, Seca, Hamburg, Germany) and height (Seca stadiometer SEC-225, Seca, Hamburg, Germany) were also measured.

Performance testing. The Yo-Yo IR1 and IR2 tests consist of repeated 2x20m shuttles interspersed by 10-s active recovery in 2m wide lanes at progressively increasing speed as controlled by audio cues as described by Krustrup *et al.* (2006b). Yo-Yo IR1 evaluates the ability to repeatedly perform high-intensity aerobic work, while Yo-Yo IR2 simultaneously stimulates aerobic and anaerobic energy turnover, warranting inclusion of both (Bangsbo *et al.*, 2008). Test performance correlates with HIR distance (Krustrup et al., 2006b), thus providing a reproducible, sensitive and valid (external, discriminant) determination of football-specific training status (Ingebrigtsen *et al.*, 2012). Peak HR achieved during Yo-Yo IR1 considered as participant's HR_{max} (Bangsbo *et al.*, 2008).

Participants completed a 5x30m RST interspersed with 25-s active recovery (jog back to start) (Krustrup *et al.*, 2006a). Tests commenced 5-min after a 10-min standardised warm-up consisting running at increasing intensity, including 2x20m sprints. Starting position was standardised. A 5-s countdown provided for each sprint. Sprint times recorded using wireless timing gates with an accuracy of 0.01-s (Brower TC Timing System, Draper, Utah, USA) adjusted to participant hip height. Fastest sprint time, mean sprint time and fatigue index

$$(\mathrm{FI} = \left(\frac{\text{Last sprint} - \text{Fastest sprint}}{\text{Fastest sprint}}\right) \times 100)$$

were derived from the RST (Bangsbo and Mohr, 2012).

Experimental model. Players completed two 6-a-side matches (one following $NaHCO_3$, one following control) on a half-sized out-door artificial pitch lasting 90-min (2x45-min) after a standardised warm-up. During the first game five players representing both teams received $NaHCO_3$ and during the second game the remaining five players received $NaHCO_3$. Mimicking full-sized game (FSG) match-play was vital to fulfil study aims. SSGs simulate overall activity patterns and closely replicate physiological and technical aspects of FSGs (Dellal *et al.*, 2012), maximising ecological validity and justifying the protocol. A goalkeeper was each team's sixth player, but was not a participant of the study. Blood [lactate] was measured at rest, half-time and full-time via fingertip capillary blood sampling (Lactate Pro, Blood lactate test meter,

Arkray Inc, Kyoto, Japan). HR was collected throughout at 5-s intervals, and recorded as absolute (bpm) and relative (%HR_{max}) values.

GPS analysis used as it provides a time-effective, sensitive and detailed analysis of activity patterns with minimal error (TEM=5.5%) (Aughey, 2010), allowing valid evaluation of fatigue development and supplement efficacy (Randers *et al.*, 2010). The locomotor categories used were modified from Mohr *et al.* (2003): standing (0-1.99km·h^{-1}); walking (2-6.99km·h^{-1}); low-speed running (7-13.99km·h^{-1}); moderate-speed running (14-16.99km·h^{-1}); high-speed running (17-20.99km·h^{-1}); very high-speed running (21-23.99km·h^{-1}); sprinting (>24km·h^{-1}). HIR (considered the most valid physical performance measure) categorised as the sum of moderate-speed running, high-speed running, very high-speed running and sprinting (Mohr *et al.*, 2003). Distance of each recorded at 5-Hz frequency for pre-determined 5-, 15-, 45- and 90-min periods. Total distance (TD) calculated as the sum of all distances. Peak and mean distance covered in HIR in a single 5-min period determined. Sprint frequency and speed data (average and peak) also determined. Two permanent fatigue indexes (PFI1 and PFI2) and one temporary fatigue index (TFI) were calculated in accordance with Mohr *et al.* (2012).

Eleven 100mm visual analogue scales (VAS) validly quantified acute GI distress (Cameron et al., 2010) and ratings of perceived exertion (RPE) (Rebelo *et al.*, 2012) at baseline, half-time and full-time. Participants placed a dash on each VAS to rate; effort, demand, nausea, flatulence, stomach cramping, belching, stomach ache, bowel urgency, diarrhoea, vomiting and stomach bloating. Length of line left of the dash used as the result (0-100mm). Examples below (figure 1).

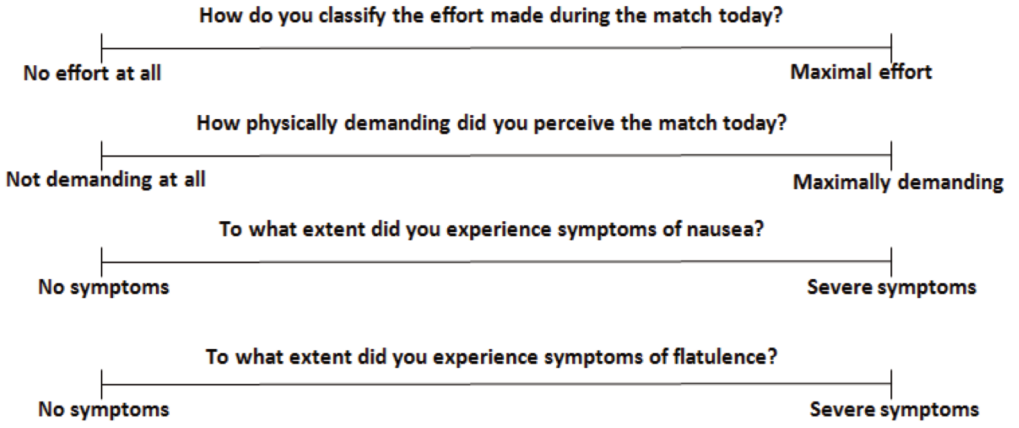

Figure 1. Example VAS scales.

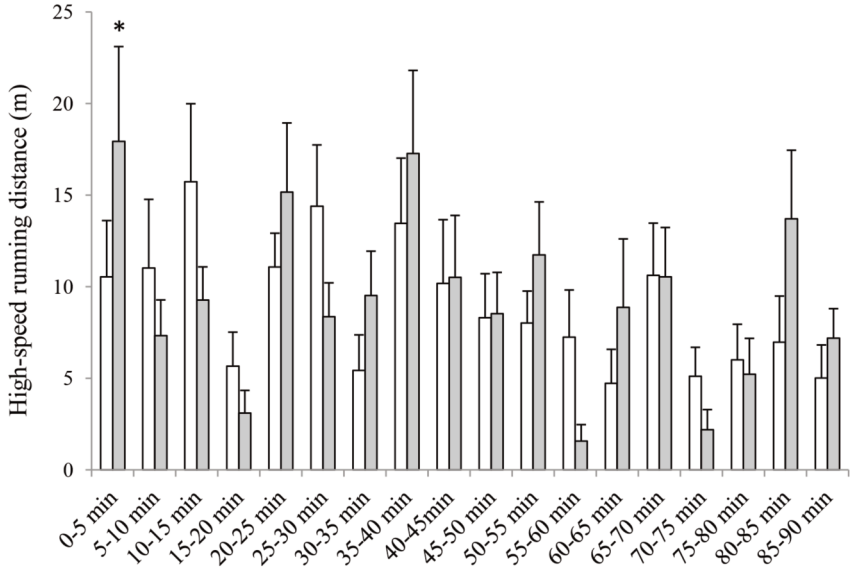

Figure 2. High speed running distance in 5-min intervals. for control □ and $NaHCO_3$ ■ (mean±SEM). *Denotes significant difference compared to control ($P<0.05$).

Supplementation protocol

Prior to the two matches, participants ingested either $NaHCO_3$ or no supplement (control) in a randomised, counterbalanced, cross-over manner. $NaHCO_3$ dosing protocol selected in accordance with Marriott *et al.* (2015). Specifically, participants ingested $0.4 g \cdot kg^{-1}$ body mass $NaHCO_3$ prior to one match; selected because $\geq 0.3 g \cdot kg^{-1}$ is required to induce alkalosis (McNaughton, 1992), but $\geq 0.5 g \cdot kg^{-1}$ likely induces significant GI distress (Requena *et al.*, 2005). Indeed, Bishop and Claudius (2005) reported performance enhancement using $0.4 g \cdot kg^{-1}$. Doses administered orally via 20-25 gelatine capsules are minimising GI distress risk vs. solution (Peart *et al.*, 2012). Four-five capsules ingested with water ad libitum at 90-, 80-, 70-, 60- and 50-min before kick-off as staggered dosing gains maximal alkalosis and minimises GI distress (Siegler *et al.*, 2012).

Statistics

Data analysed using SPSS v19.0 for Windows (SPSS Inc., Chicago, IL, USA). Results presented as mean±SEM. Assumptions of normality verified using Kolmogorov-Smirnov test. Sphericity tested using Mauchley's test, with violations corrected using Greenhouse-Geisser ($_{GG}$) correction (controlling type I error rate). Within-within differences in activity profiles, HR, blood [lactate] and VAS data between conditions evaluated using two-way (condition x time) analysis of variance (ANOVA) for repeated-measures. Least

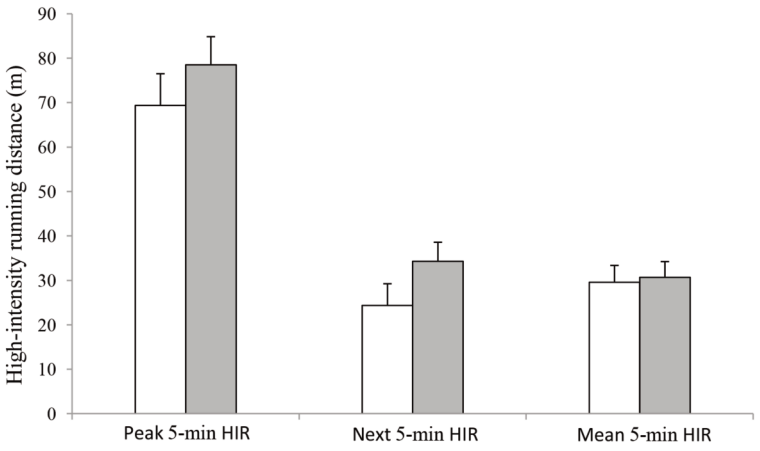

Figure 3. Peak 5-min, following 5-min and average 5-min HIR distance for control and $NaHCO_3$ ■ (mean±SEM)

significant difference (LSD) post-hoc tests located any significant differences. Many time points (up to 18) made Bonferroni inappropriate as type II error risk would be extremely high (Field, 2005); justifying use of LSD. Differences in whole game data between conditions determined using Student's paired t-tests. Relationships between delta increase in HIR distance from control to $NaHCO_3$ and each performance test, as well as between PFI1, PFI2 and TFI with each performance test determined using Pearson's product-moment correlation coefficient. Correlation strengths determined in accordance with Hopkins (2000). Significance accepted at $P<0.05$.

Results

Activity profiles

Significant main effect of time ($P<0.05$), but not condition ($P>0.05$), on distances covered in all activity profile categories (including HIR) during 5-, 15- and 45-min periods, except no main effect of time on very high-speed running and sprinting during 45-min periods ($P>0.05$).

No significant condition x time interaction effects on distances standing, walking, low-speed running, moderate-speed running, very high-speed running, sprinting or HIR (all $P>0.05$), but there was on distance high-speed running ($F_{(17, 153)}=1.764$, $P<0.05$). Specifically, post hoc LSD revealed players covered 70.3% more distance high-speed running following $NaHCO_3$ (18±5 m) compared to control condition (11±3 m) during 0-5 min ($P<0.05$; Figure 2).

No significant difference existed between conditions for peak HIR ($NaHCO_3$:79±7 vs. CON: 69±6 m) or mean 5-min HIR ($NaHCO_3$: 31±4 vs. CON: 30±4 m) HIR distance ($P>0.05$). However, HIR distance during the 5-min interval after peak the 5-min period tended to be higher in $NaHCO_3$ (34±5 m) than in CON (24±4 m) ($t_{(9)}=2.169$, $P=0.058$; Figure 3). Finally, HIR tended (P=0.06) to be lower than mean HIR in this period in CON only.

No significant condition x time interaction on distances standing, walking, moderate-speed running, fast-speed running, very fast-speed running, sprinting and HIR (all $P>0.05$). However, players covered greater low-speed running distance in control (467±36 m) compared to $NaHCO_3$ (370±34 m) during 30-45min ($F_{(5, 45)}=2.417$, $P<0.05$).

No significant condition x time interaction effect on distances standing, walking, low-speed running, moderate-speed running, fast-speed running, very fast-speed running, sprinting, HIR or TD (Figure 4B) (all $P>0.05$). HIR increased from control to $NaHCO_3$ in 60% and 70% of participants in each half and whole game, respectively (Figure 4A).

No significant differences existed between conditions in total distance covered in all activity profile data (including HIR, TD, >17km·h^{-1}, >21km·h^{-1}), PFI1, PFI2 and TFI across the whole match (all $P>0.05$; Table 1).

Table 1. Whole match activity profile, fatigue indexes and HR data (n=10).

	0-90 min	
	$NaHCO_3$	Control
Activity profile distance (m)		
Standing	195±7	195±9
Walking	4086±116	4096±98
Low-speed running	2572±103	2626±152
Moderate-speed running	413±45	388±41
High-speed running	168±24	159±24
Very high-speed running	18±4	23±5
Sprinting	0.3±0.1	1.3±1
Cumulative distances (m)		
HIR	600±70	572±65
TD	7453±193	7489±236
>17km·h^{-1}	187±27	184±27
>21km·h^{-1}	19±4	25±5
Fatigue index (%)		
PFI1	-3.4±20.7	29.1±14.9
PFI2	-13.3±17.8	24.2±9.0
TFI	54.4±8.0	63.8±5.5
Absolute HR (bpm)		
Average	151±5	152±5
Peak	186±4	186±5
Relative HR (%HR_{max})		
Average	78.0±2.5	78.6±2.5
Peak	96.3±2.0	96.0±2.4

Game variable and heart rate during a game in $NaHCO_3$ and control condition. Values are mean±SEM.. *Significant difference between conditions ($P<0.05$).

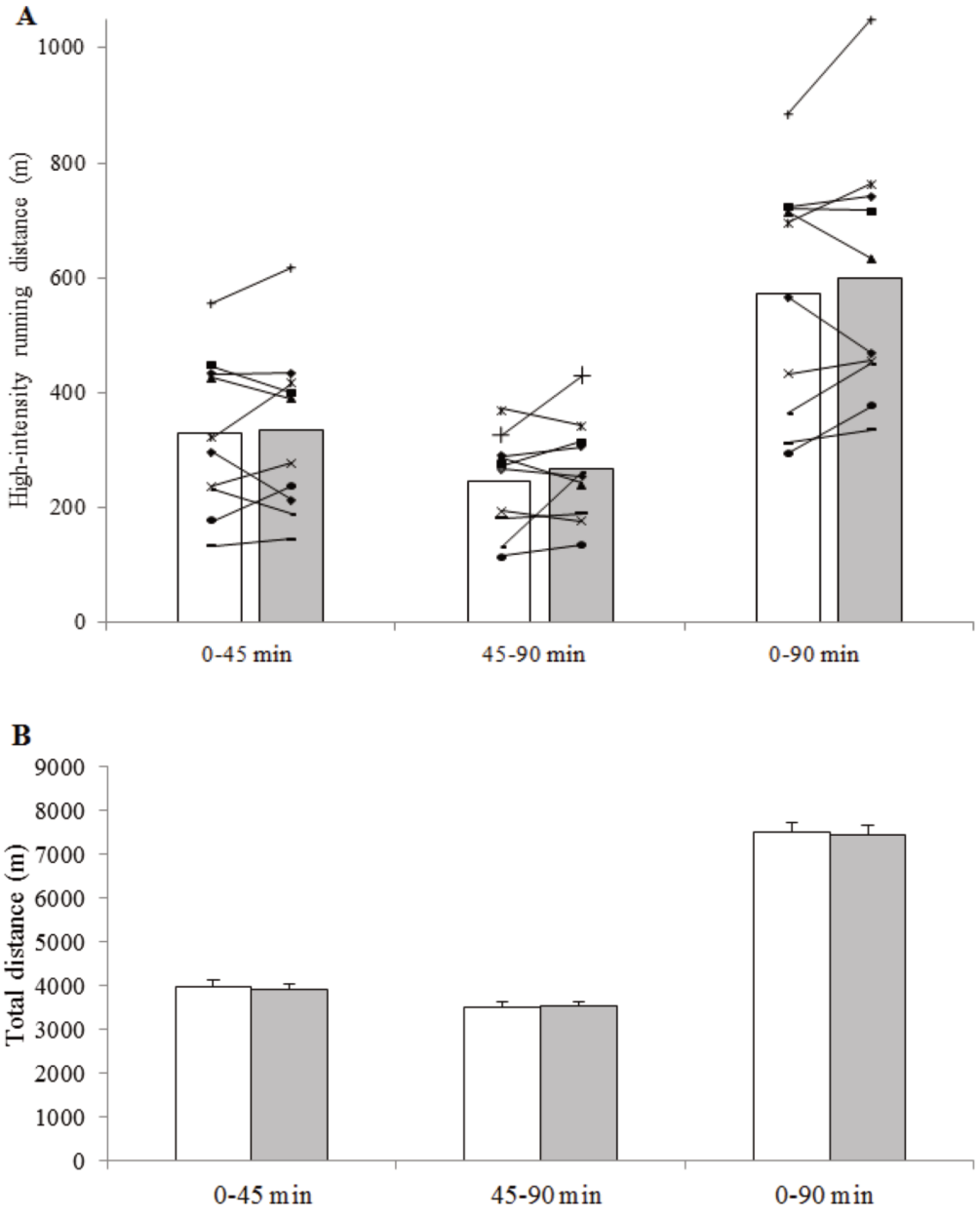

Figure 4. Mean and individual scores for HIR (A) and mean±SEM for TD (B) in each half and overall for control □ and $NaHCO_3$ ■.

Table 2. Blood [lactate] data (n=10).

	Baseline	Half-time	Full-time			
	$NaHCO_3$	Control	$NaHCO_3$	Control	$NaHCO_3$	Control
Blood [lactate]	1.9±0.2	1.6±0.3	4.5±0.7	4.8±1.2	3.6±1.0	5.0±1.1

Data are in $mmol \cdot l^{-1}$. Values are mean±SEM.

No significant main or interaction effects on average and peak speed during any match-play period (P>0.05).

Cardiovascular loading and blood lactate

Average HR values were higher (P<0.05) in the first vs. second half for absolute (157±4 vs. 146±4 bpm, respectively) and relative HR (81.1±1.9 vs. 75.4±1.8 $\%HR_{max}$, respectively) HR. No condition x time interaction on all HR measures (P>0.05). No significant differences in all HR measures between conditions over whole match (P>0.05; Table 1).

Blood [lactate] was significantly higher at half-time (4.7±0.6 $mmol.L^{-1}$) and full-time (4.3±0.8 $mmol.L^{-1}$) vs. baseline (1.8±0.2 $mmol.L^{-1}$) (P<0.05), with no significant differences between halves (P>0.05). No condition x time interaction effect existed for blood [lactate] (P>0.05; Table 2).

Table 3. Participant training status data (n=10).

Performance measure	Test result/score
Yo-YoIR1 (m)	1324±122
Yo-YoIR2 (m)	524±52
RST fastest sprint (s)	4.64±0.07
RST mean sprint (s)	4.77±0.09
RST fatigue index (%)	5.2±1.0

Values are mean±SEM.

VAS ratings

There were no condition x time interactions on perceived effort, perceived demand, nausea, stomach ache, bowel urgency, diarrhoea and vomiting (all P>0.05), but there was on flatulence ($F_{GG(1.299,\ 11.690)}$=4.471, P<0.05), stomach cramping ($F_{(2,\ 18)}$=10.214, P<0.05), belching ($F_{GG(1.184,\ 10.654)}$=6.048, P<0.05) and stomach bloating ($F_{GG(1.108,\ 9.970)}$=8.889, P<0.05). Post hoc LSD located these differences (Figure 5). No significant VAS differences between conditions at kick-off (P>0.05).

Correlations

Delta HIR increase (m) from control to $NaHCO_3$ represents $NaHCO_3$ HIR-control HIR (positive=$NaHCO_3$>control; negative=$NaHCO_3$<control). Training status data presented below (Table 3).

Significant correlations between Yo-Yo IR1 (Figure 6A), Yo-Yo IR2 (Figure 6B and 6C) and RST measures (Table 4) with delta HIR increase during different match-play periods presented below. PFI1 (r= -0.58) and PFI2 (r= -0.76) significantly correlated with Yo-Yo IR2 performance in control condition only (P<0.05). TFI was not significantly correlated with any performance test in either condition (P>0.05).

Yo-Yo IR1 was not correlated with total HIR in either condition (P>0.05). Yo-

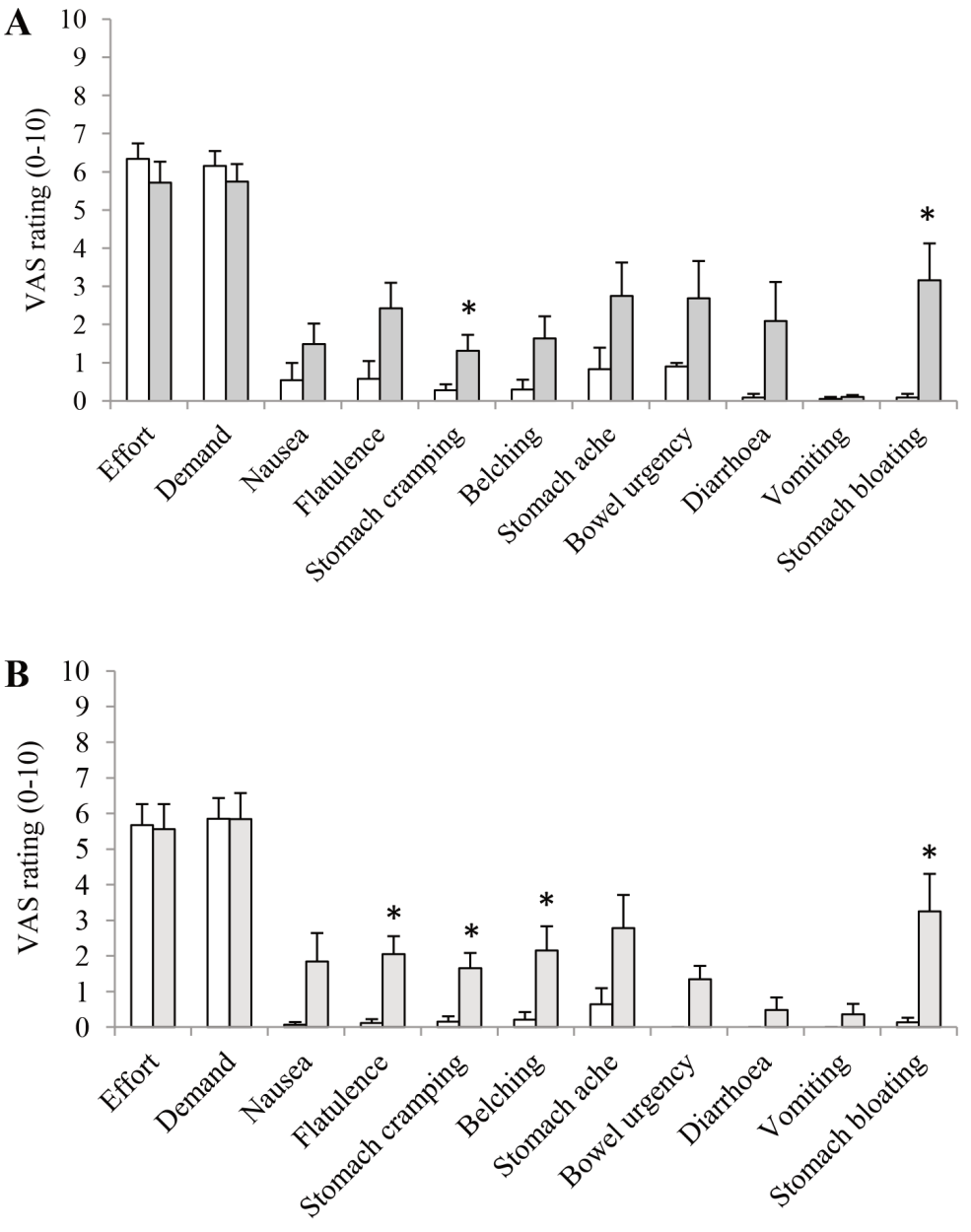

Figure 5. VAS rating in first- (A) and second-half (B) for control □ and $NaHCO_3$ ■ (mean±SEM). *Significantly higher in $NaHCO_3$ (P<0.05).

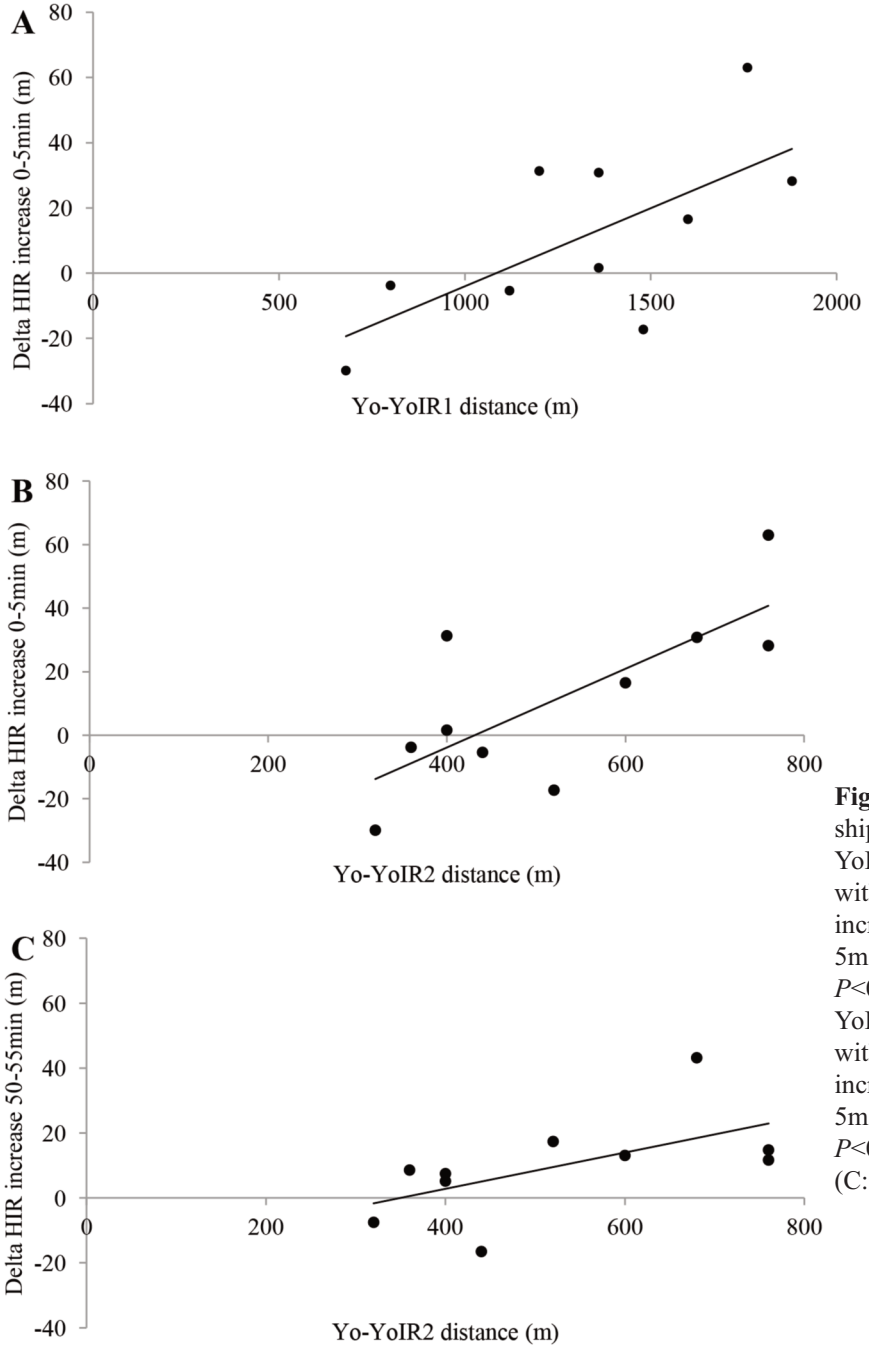

Figure 6. Relationships between: Yo-YoIR1 performance with delta HIR increase during 0-5min (A: r= 0.75; P<0.05) and Yo-YoIR2 performance with delta HIR increase during 0-5min (B: r= 0.67; P<0.05) and 50-55min (C: r= 0.59; P<0.05).

Table 4. Relationship between delta HIR increase and RST performance data

Performance measure	Time period			
	15-20 min	0-45 min	60-75 min	0-90 min
RST fastest sprint (s)		*r*= -0.75*		*r*= -0.63*
RST mean sprint (s)		*r*= -0.74*	*r*= -0.58*	*r*= -0.69*
RST FI (%)	*r*= -0.57*		*r*= -0.59*	*r*= -0.67

**Statistical significance ($P<0.05$).

Yo IR2 correlated with total HIR during 0-45, 45-90 and 0-90 min in both $NaHCO_3$ (*r*= 0.65, 0.57 and 0.65 respectively; $P<0.05$) and control (*r*= 0.57, 0.58 and 0.61 respectively; $P<0.05$).

Discussion

The current study is the first to use football match-play to investigate the ergogentic effects of oral $NaHCO_3$ supplementation. Major findings shows that players covered greater high-speed running (17-21 $km \cdot h^{-1}$) distance during 0-5 min of match-play in the $NaHCO_3$ vs. control condition. However, all other activity profiles, including HIR, were unchanged by $NaHCO_3$ during all match-play periods. Furthermore, $NaHCO_3$ efficacy was extremely individual, potentially mediated by training status. Therefore, results support experimental hypotheses one and three, but not two.

Players performed 70% more high-speed running following $NaHCO_3$ vs. control during the initial 5-min of the first half, which on average wass the most intense 5-min interval of both game-trials (Figure 2). The first part of a game is usually the period with most high intensity running (Mohr *et al.*, 2003) and may therefore be the period challenging the acid-base homeostasis to the highest degree. Thus, the potential effect of $NaHCO_3$ supplementation may be greatest in the initial phase of a game. No previous study has used game activity pattern as dependent variable, making direct comparisons impossible. However, corroboratory evidence has been reported during repeated-sprint (Bishop *et al.*, 2004) and intense intermittent exercise (Krustrup *et al.*, 2015; Marriott *et al.*, 2015); exercise protocols relevant to football match-play. Additionally, HIR during the 5-min interval following the peak 5-min period was lowered below mean (tendency; P=0.06) for control, as also shown by others (Mohr *et al.*, 2003), but not in the $NaHCO_3$-trial (non-significant), and HIR in this period tended (P=0.058) to be higher (~41%) in $NaHCO_3$ than control; possibly representing attenuated temporary fatigue development following $NaHCO_3$ intake. These performance enhancements are valuable and may result from reduced efflux and increased reuptake of K^+ in the exercising muscles via the Na^+-K^+ ATPase, and resultant attenuated exercise-induced rise in muscle interstitial [K^+] associated with alkalosis (Street *et al.*, 2005); important given the likely role of interstitial K^+ accumulation in temporary fatigue development (Mohr *et al.*,

2005). Indeed, reducing interstitial [K^+] has been associated with improved intermittent exercise performance (Mohr *et al.*, 2011). However, high-intensity repeated /intermittent-sprint performance is not always improved after $NaHCO_3$ intake despite inducing alkalosis (De Ste Croix & Pope, 2006; Price & Simons, 2010; Tan *et al.*, 2010), thus contradicting current findings. The different doses used in these studies and the use of solution (increases GI distress risk vs. capsules; Peart et al., 2012) may contribute to the lack of performance improvements in these studies. Furthermore, staggering ingestion as in the present study has been associated with improved performance (Bishop & Claudius, 2005; Marriott *et al.*, 2015; Krustrup *et al.*, 2015), whereas single bolus was not (e.g. Tan *et al.*, 2010). Varying exercise modality, physiological demands, circadian variation and participant training status may also contribute to aforementioned discrepancies (Douroudos *et al.*, 2006).

Paradoxically, all other activity data, including HIR, were unaffected by $NaHCO_3$ throughout. Lack of performance effect supports some (e.g. Tan *et al.*, 2010), but not all (Bishop *et al.*, 2004) previous investigations. Peak speed was also unaffected by $NaHCO_3$, agreeing with Zinner *et al.* (2011). Furthermore, non-significant tendency for less high-speed running following $NaHCO_3$ during 25-30 and 55-60 min could suggest an ergolytic effect. Frequent lower-intensity periods during match-play would be expected to be conducive to $NaHCO_3$ efficacy (Siegler *et al.*, 2008). However, no significant TFI improvement suggests temporary fatigue was not attenuated with $NaHCO_3$, possibly contributing to lack of HIR change. Lack of HIR increase could also be ascribed to failure of supplementation to induce the increases in pH (0.05-0.1unit) and [HCO_{3-}] (~5.0mmol.L^{-1}) generally required for improved repeated/intermittent-sprint performance (Bishop, 2010), as this would nullify aforementioned ergogenic mechanisms associated with $NaHCO_3$. However, these targets are usually exceeded following staggered 0.4 g·kg^{-1} $NaHCO_3$ ingestion (Bishop & Claudius, 2005). Therefore, factors other than H^+ accumulation, such as interstitial [K^+] (temporary fatigue) and glycogen depletion in individual muscle fibres ('permanent' fatigue), may have limited performance (Krustrup *et al.*, 2006a), meaning additional extracellular buffer capacity afforded by $NaHCO_3$ would provide limited performance benefits. This may contribute to the lack of performance improvement, and further dismiss H^+ accumulation as a major contributor to football-specific fatigue. Indeed, alkalosis can occur without concomitant performance enhancements (Tan *et al.*, 2010).

Alternatively, average match intensity (~80% HR_{max}) may not have provided sufficient metabolic stress (including [H^+]) to benefit from additional extracellular buffer capacity afforded by $NaHCO_3$, further contributing to lack of HIR improvement. Additionally, consensus suggests $NaHCO_3$ is ergogenic during exercise of 1-15 min (Bellinger *et al.*, 2012), possibly explaining why high-speed running only improved during the first 5-min interval of the game, and why HIR over 90-min was

not improved. Nevertheless, HIR was non-significantly higher during the first-half (1.9%), second-half (8.9%) and whole match (4.9%) following $NaHCO_3$ and may have failed to reach statistical significant due to the small sample size. However, such improvements may have practical significance.

Perceived effort and demand were not significantly affected by $NaHCO_3$, agreeing with some (Zabala *et al.*, 2011), but not others who report reduced RPE following $NaHCO_3$; discrepancies partly attributable to varying protocol metabolic requirements (Marriott *et al.*, 2015; Krustrup *et al.*, 2015). This lack of effect may further contribute to lack of HIR improvement. Furthermore, $NaHCO_3$ significantly increased rating of flatulence, stomach cramping, belching and stomach bloating, possibly attributable to increased small intestine [Na^+] (Heigenhauser, 1991). GI distress is frequently reported, and may have negated potential ergogenic effects of $NaHCO_3$, possibly contributing to lack of HIR increase.

$NaHCO_3$ had no significant effect on blood [lactate], contrasting increased concentrations reported previously (Hollidge-Horvat *et al.*, 2000). This result suggests glycolytic turnover and/or lactate efflux were not enhanced following $NaHCO_3$, possibly further explaining the lack of HIR increase (Juel, 1998). However, it is likely that the blood lactate levels have been higher early in the first half, where the effects of the $NaHCO_3$ are likely to peak, which also is indicated by the great amount of high intensity running during the first 5 min of the game (Figure 2).

HIR increased from control to $NaHCO_3$ in 60% and 70% of participants in each half and whole game, respectively (figure 4A), reiterating the low vs. high-responder effect frequently reported following drug manipulations (Price & Simons, 2010). This large individual variability may have prevented statistical significance for HIR. Interestingly, large-very large significant correlations ($0.5<r<0.9$) exist between training status and improvement in HIR from control to $NaHCO_3$; suggesting fitness level may mediate this inter-individual variability. Specifically, higher Yo-Yo IR1 and IR2 performances were related to greater HIR increases following $NaHCO_3$ vs. control during 0-5 and 50-55min (Yo-Yo IR2 only). Additionally, lower values for RST FI, fastest and mean sprint time (greater training status) related to greater HIR gains during 0-45 (fastest and mean), 0-90 (all), 15-20 (FI) and 60-75min (mean and FI). Collectively, these results suggest higher training status (high Yo-Yo IR values, low RST values) may potentiate $NaHCO_3$ efficacy, at least within this specific sample of sub-elite footballers. In support of this notion greater performance gains with higher training status have been reported previously (Carr *et al.*, 2012); apparently particularly true during repeated-sprint exercise (weighted effect size: trained=0.18 vs. untrained=0.05) (Peart *et al.*, 2012), as observed during football.

Inherent match-to-match variation in activity patterns (Gregson *et al.*, 2010) and relatively small sample size possibly reduced statistical power (increasing type II error risk), thus preventing statistical sig-

nificance. Furthermore, considerably lower training status of current sub-elite participants vs. elite players (Mohr & Krustrup, 2013), may make direct extrapolation of results to elite players erroneous. To overcome these limitations, future research could investigate $NaHCO_3$ efficacy using a large sample of preferably elite players during the Copenhagen Soccer Test; a controlled, reproducible protocol that closely relates to match-play and effectively evaluates technical ability (not assessed in the current study but contributes to overall performance and nutritional interventions (Bendiksen *et al.*, 2012). Additionally, components of an optimal dosing strategy using $0.4g \cdot kg^{-1}$ (e.g. timings, capsules vs. solution) should be identified; research that is lacking for this dose. Further improvements to the current study include use of a double-blind, placebo-controlled design, and analysis of blood samples for pH, $[H^+]$ and $[K^+]$ to allow a more mechanistic explanation of results to be provided.

In conclusion, despite no significant effect on total match HIR, the significant increase in high-speed running in the first 5-min vs. control suggests $NaHCO_3$ may be an effective ergogenic aid for football players, with no significant ergolytic effect observed in any activity profile. Furthermore, results suggest individuals with higher training status may gain greater ergogenic benefits from $NaHCO_3$ ingestion. Finally, individual variability in response reiterates the importance of evaluating performance changes on an individual basis before making recommendations regarding $NaHCO_3$ intake.

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