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Effect of carbohydrate or sodium bicarbonate ingestion on performance during a validated basketball simulation test.

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ABSTRACT

Current recommendations for nutritional interventions in basketball are largely extrapolated from laboratory-based studies that are not sport-specific. We therefore adapted and validated a basketball simulation test relative to competitive basketball games using well-trained basketball players (n=10), then employed this test to evaluate the effects of two common pre-exercise nutritional interventions on basketball-specific physical and skilled performance. Specifically, in a randomised and counterbalanced order, participants ingested solutions providing either 75 g carbohydrate (sucrose) 45 min before exercise (Study A; n=10) or 2x0.2 $g \cdot kg^{-1}$ sodium bicarbonate ($NaHCO_3$) 90 and 20 min before exercise (Study B; n=7), each relative to appropriate placebos (H_2O and 2x0.14 $g \cdot kg^{-1}$ NaCl, respectively). Heart rate, sweat rate, pedometer count and perceived exertion did not systematically differ between the 60-min basketball simulation test and competitive basketball, with a strong positive correlation in heart rate response (r=0.9, $P<0.001$). Pre-exercise carbohydrate ingestion resulted in marked hypoglycaemia (<3.5 $mmol \cdot l^{-1}$) throughout the first quarter, coincident with impaired sprinting (+0.08±0.05 s; $P=0.01$) and lay-up shooting performance (8.5/11 *versus* 10.3/11 baskets; $P<0.01$). However, ingestion of either carbohydrate or sodium bicarbonate ingestion prior to exercise offset fatigue such that sprinting performance was maintained into the final quarter relative to placebo (Study A: -0.07±0.04 s; $P<0.01$ and Study B: -0.08±0.05 s; $P=0.02$), although neither translated into improved skilled (lay-up shooting) performance. This basketball simulation test provides a valid reflection of physiological demands in competitive basketball and is sufficiently sensitive to detect meaningful changes in physical and skilled performance. While there are benefits of pre-exercise carbohydrate or sodium bicarbonate ingestion, these should be balanced against potential negative side-effects.

Key Words: Intermittent Exercise, Sucrose, Hypoglycaemia, Performance.

INTRODUCTION

Basketball is a team sport involving intermittent periods of sprinting, jogging and walking that act as vehicles for players to gain optimum positions on court from which to exercise a wide range of motor skills. This varied combination of physiological and technical demands presents an opportunity to enhance performance via a variety of training strategies and nutritional interventions. Whilst relevant and specific physical training certainly presents the greatest potential to enhance overall basketball performance, additional performance benefits may be possible via nutritional interventions prior to competition.

Much of what is known about the limitations to physical performance comes from laboratory-based trials involving continuous, often steady-state exercise on a treadmill or cycle ergometer. Consequently, there is a relative lack of information available regarding the efficacy of interventions for intermittent sports such as basketball (Bangsbo et al. 2006). The emergence of intermittent exercise protocols has therefore been invaluable in establishing whether conclusions drawn from steady-state exercise can be confidently generalised to intermittent sports (Welsh et al. 2002; Winnick et al. 2005). An example of one such protocol is the Loughborough Intermittent Shuttle Test (LIST), which provides a credible simulation of the demands of soccer (Nicholas et al. 2000). Importantly, no equivalent protocol has been validated relative to basketball, so the effects of many purportedly ergogenic aids on basketball-specific performance remain to be established (e.g. carbohydrate and bicarbonate).

The ergogenic properties of carbohydrate ingestion have been widely documented across a range of experimental conditions (Jeukendrup & Jentjens 2000). Specific to intermittent exercise, ingestion of carbohydrate-electrolyte solutions extends exercise time to exhaustion and delays the deterioration of both repeated sprint and skilled performance

(Nicholas et al. 1995; Welsh et al. 2002; Winnick et al. 2005; Gant et al. 2007). However, ingestion of carbohydrate within the hour before exercise often produces a transient period of hypoglycaemia (Foster et al. 1979). Although the balance of evidence published to date indicates that endurance performance is ultimately unaffected by any transient hypoglycaemia early in exercise (Jentjens & Jeukendrup 2003; Jentjens et al. 2003; Moseley et al. 2003), it is possible that a brief period of low blood glucose availability may impair high-intensity and/or skilled performance during the initial stages of intermittent sports.

During brief periods of high-intensity exercise there is an increase in the generation of hydrogen ions and this has long been associated with the onset of fatigue (Nakamaru & Schwartz 1972), so it is unsurprising that methods of augmenting buffering capacity have been explored. One such method is to induce metabolic alkalosis via ingestion of sodium bicarbonate ($NaHCO_3$). This approach can provide an ergogenic advantage during high intensity short-duration exercise (Wilkes et al. 1983; Bird et al. 1995) and more recent research has demonstrated improved repeated sprint performance during prolonged intermittent cycling (Bishop et al. 2004; Bishop & Claudius 2005). The latter suggests that pre-exercise sodium bicarbonate ingestion may be a useful supplement for intermittent team sports but this hypothesis is yet to be empirically supported.

We sought to investigate repeated sprint and skilled performance in response to pre-exercise carbohydrate or sodium bicarbonate ingestion, each relative to appropriate placebos. In order to do so, we first validated a novel basketball simulation test, before demonstrating its sensitivity to detect meaningful changes in performance when applied to evaluate these common pre-exercise nutritional interventions. It was hypothesised that pre-exercise carbohydrate ingestion would result in initial hypoglycaemia and thus impaired physical and skilled performance early in exercise, whereas sodium bicarbonate ingestion would enhance these measures of basketball performance.

MATERIALS AND METHODS

Participants and Experimental Design

A cohort of 27 well-trained male basketball players took part in the experimental design illustrated in Figure 1. These individuals were healthy, non-smokers with >4 years competitive experience, ranging from University to International level competition. At weekly intervals, 10 of these participants (age 22±2 years, height 1.86±0.06 m, mass 83.5±6.8 kg, $\dot{V}O_2$ max 51.8±3.5 ml·kg^{-1}·min^{-1}) were monitored during a game situation, then a simulated game (using a modified version of the LIST) and then once more during another game situation (to control for inter-game variation and trial order effects). Having validated the above protocol, a further ten participants took part in an experimental application of that protocol to evaluate the efficacy of pre-exercise carbohydrate ingestion during simulated basketball performance (Study A: n=10, age 20±1 years, height 1.83±0.07 m, mass 84.5±12.8 kg, $\dot{V}O_2$ max 50.7±2.8 ml·kg^{-1}·min^{-1}). The remaining seven participants completed the same procedures but to assess the efficacy of pre-exercise sodium bicarbonate ingestion (Study B: n=7, age 21±2 years, height 1.81±0.10 m, mass 81.0±9.6 kg, $\dot{V}O_2$ max 54.0±3.8 ml.kg^{-1}.min^{-1}). Both intervention studies involved a cross-over design with exposure to treatments in a randomised and counterbalanced trial order. Each participant was briefed regarding the nature of their respective study and provided informed consent both verbally and in writing. The protocol validation was approved by the Loughborough University Ethical Advisory Committee and the experimental applications of that protocol (i.e. Studies A & B) were approved by the University of Bath Research Ethics Approval Panel.

Validation of Basketball Simulation Test

The exercise intensities employed during the basketball simulation test were calculated relative to maximal oxygen uptake ($\dot{V}O_2$ max), as determined using a progressive shuttle-running protocol to exhaustion (Nicholas et al. 2000). One week later, participants returned to the laboratory in a fed-state having recorded their dietary energy intake and abstained from caffeine and vigorous physical exercise for the preceding 48 h and were provided with 550 ml of plain water. A urine sample was collected for assessment of osmolality using a cryoscopic osmometer (Gonometer 030, Gonotec, Germany), with mean (*s*) values for the group of 639±280 $mOsmol \cdot kg^{-1}$ for game 1, 761±273 $mOsmol \cdot kg^{-1}$ for the basketball simulation and 791±133 $mOsmol \cdot kg^{-1}$ for game 2 (F=1.0, P=0.38). Adequate hydration was assumed for osmolality values below 900 $mOsmol \cdot kg^{-1}$ (Shirreffs & Maughan 1998), with any participants exceeding this value provided with a further 500 ml of plain water immediately before testing. Participants emptied their bladders and were weighed nude using a digital electronic scale to establish pre-exercise body mass. Participants were also fitted with absorbent gauze sweat patches (Tegaderm absorbent dressing 5x7 cm, 3M, Loughborough) placed on the forearm, back (sub-scapular), thigh and chest, along with a chest-worn heart rate monitor (Team Polar, Finland) and a pedometer (Yamax Digi-Walker SW-200).

Participants next underwent a standardised 10-min warm-up before performing the first shooting test as previously described for the control (resting) trial. After this initial test, players were divided into two teams broadly matched for ability/skill level and $\dot{V}O_2$ max and commenced a game of 5-on-5 full court basketball with man-to-man defence, played over four 15-min quarters. After the eleventh minute of each quarter each individual paused to record their rating of perceived exertion (Borg 1973) before repeating their shooting tests at separate baskets between quarters. The game was restarted as quickly as possible after shooting tests, except for at half-time when an additional 10 min break was included. Fluid intake was *ad libitum* during this first game, with participants provided with and encouraged to consume a matched volume during subsequent visits. Following the game, sweat patches were removed before post-exercise body mass was recorded and corrected for actual fluid intake and any urine output to determine sweat losses.

One week later, participants repeated the basketball simulation test having adhered to the same pre-trial procedures/diet as described above for game 1. The same monitors were fitted and pre-exercise measurements made, before participants commenced a modified version of the Loughborough Intermittent Shuttle Test (modified in that only four rather than six 15-min blocks of shuttle-running were performed, with a 10-min break at the halfway point and with the basketball shooting tests interspersed between blocks). Finally, one week after the basketball simulation test, a second game was repeated exactly as was the first. A whirling hygrometer (Zeal, UK) was operated in close proximity to the participants every 15-min during actual and simulated basketball to measure the wet and dry bulb temperatures and hence calculate relative humidity. Environmental temperature and humidity averaged across all participants in each condition were 19.4±0.9°C and 54±4% for actual games and 20.1±1.2°C and 49±6% for simulated games.

Preliminary Testing (Studies A & B)

As described above, all participants completed a multi-stage fitness test to determine $\dot{V}O_2$ max (Ramsbottom et al. 1988) before subsequently visiting the laboratory to complete just 2 blocks of the Loughborough Intermittent Shuttle Test (including the lay-up shooting test) by way of familiarisation with the study procedures (thus minimising learning effects).

Experimental Procedures (Studies A & B)

All participants completed the same pre-trial procedures/diet as described above, except that participants arrived in an overnight fasted state at 0900 h (± 1 h) and those in Study A were formally requested to have performed a standardised 60-min bout of moderate intensity road-running within the 24 h preceding each trial (simply to reduce between-subject variation in carbohydrate availability due to recent training history, which could conceivably interact with the effect of carbohydrate ingestion). Once baseline measures of nude body mass had been recorded, a 20 µl capillary finger prick blood sample was taken for determination of glucose and lactate concentration, before a bolus of the relevant experimental solution was ingested. Participants then rested for either 45 min (Study A) or 90 min (Study B) before another finger prick blood sample was taken immediately pre-exercise in Study A only (i.e. this additional pre-exercise sample is only pertinent to the acute effect of carbohydrate ingestion and not for sodium bicarbonate in Study B).

Following pre-exercise measures, participants commenced four blocks of a modified version of the LIST (Nicholas et al. 2000). As detailed above with regard to our validation procedures, the more standard 5 or 6 block version of the test was instead abbreviated to just 4 blocks, thus more consistent with the duration and work:rest ratios of a typical basketball match. Briefly, each exercise block lasts approximately 15 min (dependent on individual intensity) and comprises 11 cycles, with each cycle including: 3x20 m walking (1.54 $m \cdot s^{-1}$); 1x20 m timed maximal sprint (9.5 s allowed for sprint plus recovery), 3x20 m ‘running’ (~3.49 $m \cdot s^{-1}$) and 3x20 m ‘jogging’ (~2.79 $m \cdot s^{-1}$), with slight inter-individual variability in the latter two due to individually prescribed intensity (Figure 2).

The exercise test validated above was applied with a ‘lay-up’ shot incorporated directly into the sprint cycles of Studies A & B. A lay-up is a whole-body motor skill fundamental to basketball, in which a basketball is shot at close range whilst running towards the hoop, thus combining effective timing, footwork, and hand-eye coordination. Critically, this is a skill which players of any competitive standard should be capable of producing a consistently successful outcome at baseline (i.e. at least 90% success rate), thus providing the opportunity to track the onset of fatigue and potential treatment effects within the context of a demanding exercise protocol.

These lay-ups were simply incorporated into the Loughborough Intermittent Shuttle Test by including pre-recorded verbal instructions between the audible beeps that dictate individualised pace to each participant during the exercise protocol. Specifically, the instruction “sprint in 3, 2, 1” which precedes a beep marking the start of a sprint was almost immediately followed by the instruction “score in 3, 2, 1” followed by another beep 4.5 seconds after the first. The sprint begins from one baseline of a standard basketball court, approximately 1.5 m from the centre of the court, and participants sprint maximally through the usual 20 m distance used in this protocol, at which point they are able to collect a standard men’s basketball from a cylindrical pedestal 0.72 m above the floor and placed, 4.3 m in front and 1.8 m to the right of the basket (reversed for left-handed players). For a successful score to be recorded, the ball must have left the participant’s hand before the second beep (consistent with game clocks in actual basketball). A successful score therefore requires an interaction of both the speed to reach the basket in time and the ability to accurately execute the shot at high-intensity, consistent with game play. All sprints involved maximal effort over the full 20 m distance and were monitored using laser timing gates (Smart Speed, Fusion Sport, Australia) for the full 20 m distance in Study A but only over the first 15 m in Study B (the latter is consistent with the procedures originally described for this protocol; the research team only realised the gates had been placed 20 m apart in Study A after a number of participants had completed testing, so this method was continued for the remainder of the sample in that study).

Other measurements during this protocol included finger-prick capillary blood samples taken prior to supplementation and at the end of each exercise block (with an additional sample 5 min into the first block in Study A given transient hypoglycaemic responses expected early in exercise). Ratings of perceived exertion (Borg 1973) were also recorded immediately following each exercise block, with heart rate (Polar HR monitor, Kempele, Finland) noted during the first shuttle of each walking section. Fluid (water) intake was available *ad libitum* during participants' first trials and was monitored and replicated in subsequent trials.

Supplement Composition (Study A)

The CHO solution was 75 g of sucrose made up to 500 ml with sugar-free, orange flavoured, artificially sweetened cordial . This absolute quantity has previously been shown to elicit rebound hypoglycaemia (Foster et al. 1979) and broadly meets the maximum rates of exogenous carbohydrate oxidation (Jeukendrup & Jentjens 2000) relative to the duration of exercise performed (i.e. $\geq$60 g glucose$\cdot$h^{-1}). Similarly, sucrose was selected simply as a common carbohydrate source that would elicit the necessary acute effects on metabolism (Wallis & Wittekind 2013). The placebo was matched in volume and composition but without the sucrose. These solutions were provided 45 min prior to exercise at ambient temperature and ingested within 5 min. At exit interview, all participants expressed that the treatments tasted different to one another but none felt able to accurately identify which included carbohydrate.

Supplement Composition (Study B)

Either 0.2 $g\cdot kg^{-1}$ $NaHCO_3$ or 0.14 $g\cdot kg^{-1}$ NaCl was ingested in 500 ml water 90 min prior to the exercise protocol and then an equal dose again 20 min prior to the start of exercise. This dose and timing of sodium bicarbonate supplementation was selected based on previous evidence that dividing the dose across multiple smaller feedings at these times can increase plasma HCO_3 concentrations as effectively as a single dose of 0.3 $g\cdot kg^{-1}$ $NaHCO_3$ but without the adverse gastrointestinal side-effects typically associated with the latter (Bishop & Claudius 2005). Nonetheless, all participants experienced some degree of gastrointestinal disturbance and accordingly were able to identify which treatment was sodium bicarbonate. The dose of NaCl was selected in order to provide an equimolar amount of sodium as the $NaHCO_3$ supplement. This was a precaution to negate the possibility that either removal of H^+ via the Na^+/H^+ exchanger or any more generalised sodium-mediated change in intravascular fluid status may confound the results (Juel 1998).

Sampling and Analysis

Capillary blood samples were drawn using an automatic lancet (Accu-Check, Softclix Pro, Roche Diagnostics LTD., Lewes, U.K.) from the fingertips of the non-dominant hand to reduce any potential impact on lay-up performance. Samples were collected into sealable anti-coagulant (ethylenediaminetetraacetic acid) capillary tubes (Sarstedt, Leicester, UK) and analysed for glucose and lactate concentrations using a YSI 2300 Stat Plus (Yellow Springs, Ohio). Intra-assay coefficients of variation were determined for glucose (1.2%) and lactate (6.4%) based on 10 separate analyses of a single sample.

The volume of sweat absorbed by patches was determined gravimetrically. Samples were then mixed and diluted with di-ionised water and analysed in duplicate. The concentration of sweat sodium and potassium were determined by flame photometry (480 Flame Photometer, Corning, Halstead, UK). Reported data show the arithmetic mean concentration from all four measurement sites.

Statistical Analyses

A two-way general linear model for repeated measures (TreatmentxTime) examined differences over time between experimental conditions, with a Greenhouse-Geisser correction for epsilon <0.75 and a Huynh-Feldt correction adopted for less severe asphericity. The Holm-Bonferroni step-wise method was adopted to determine the location of variance (Atkinson 2002). In Study A a pre-planned contrast focused on block one of the exercise protocol to address the specific research question regarding the potential ergolytic effects of early onset rebound hypoglycaemia. Statistical analyses were performed using the SPSS version 20 (Chicago, USA), statistical significance was accepted at $P \leq 0.05$ and all data are presented as mean±standard deviation (SD).

RESULTS

Validation of Basketball Simulation Test

Heart rate responses to the basketball simulation test showed good agreement with the averaged responses across the two games. Table 1 presents data for each quarter and confirms that absolute heart rates were typically maintained within 4 beats·min^{-1} between conditions. There was therefore no significant difference in the overall mean heart rate between games and simulation ($P=0.1$) and a strong positive correlation in heart rate responses across all participants ($r=0.9$, $P<0.001$). However, while there was no significant difference in either ratings of perceived exertion (*P*=0.1) or pedometer step counts (*P*=0.3) between games and the basketball simulation, these respective variables did not correlate at all across participants (r=0.1, *P*=0.9 & r=0.3, *P*=0.4). The lack of relationship between these variables was due to a relatively homogenous response to the individually prescribed intensity during basketball simulation, relative to relatively greater inter-individual variability during actual games. Similarly, there was no correlation between games and simulation in sweat rates derived from body mass losses (r=0.3, *P*=0.4) and a tendency was apparent for greater sweat losses during the basketball simulation test (-2.3±0.4 l) than the average of the two games (-2.0±0.3 l, *P*=0.06). Although no data are available for the basketball simulation, sweat electrolytes were measured during game situation and revealed absolute sodium losses of 121±22 mol and potassium losses of 11±3 mol (relative losses in Table 1).

Study A

Ingestion of carbohydrate prior to exercise produced a poorer overall lay-up performance than ingestion of the placebo (F=6.2, *P*=0.03; Figure 3A). While there was no time effect considered across both treatments (F=2.3, *P*=0.1), the treatment x time interaction approached statistical significance (F=2.7, *P*=0.067) and the pre-planned contrast during the early hypoglycaemic period revealed that the overall treatment effect was predominantly due to impaired skill performance during the first exercise block (*P*=0.004). Figure 4A illustrates the sprint times during each lay-up attempt; there were no effects of treatment (F=0.4, *P*=0.60) or time (F=0.8, *P*=0.4) but a treatment x time interaction (F=15, *P*=0.002), reflecting ergolytic effects of carbohydrate during the first quarter (*P*=0.01) but an ergogenic effect during the fourth (*P*<0.01).

There were effects of treatment (F=0.29, *P*=0.001), time (F=8, *P*=0.001) and a treatment×time interaction (F=29, *P*<0.001) for blood glucose responses to carbohydrate and placebo ingestion (Figure 5A). Carbohydrate ingestion resulted in blood glucose concentrations that were higher pre-exercise (P<0.01) but lower during the first quarter (P<0.001) than when placebo had been ingested. Blood lactate concentrations displayed a highly consistent response between treatments (treatment×time interaction: F=1.0, *P*=0.4; Figure 6A).

Study B

Lay-up performance exhibited a time effect (F=8.2, *P*=0.02), consistent with a gradual deterioration in performance with the onset of fatigue in both the placebo and sodium bicarbonate trials (Figure 3B). Whilst visual inspection of the data on Figure 3B indicates that this effect was largely driven by the more marked decrement in the placebo trial, there was no treatment×time interaction (F=2.1, *P*=0.2) nor any overall effect of treatment (F=2.8, *P*=0.1). In contrast, the time effect (F=6.2, *P*=0.03) noted for the 15 m sprints leading into each lay-up can be attributed to the progressively increasing sprint times in the placebo trial (treatment×time interaction: F=4.9, *P*=0.03), resulting in an impaired performance relative to the sodium bicarbonate trial by the fourth quarter (*P*=0.02).

Glucose concentrations were broadly similar between the sodium bicarbonate and placebo treatments (treatment×time interaction: F=0.8, *P*=0.5; Figure 5B). However, blood lactate concentrations were higher throughout exercise following sodium bicarbonate rather than placebo ingestion (treatment: F=19, *P*<0.01) and diverged further towards the end of exercise (treatment×time interaction: F=4.1, *P*=0.04) such that the treatments differed significantly by the end of the fourth quarter (*P*=0.01).

DISCUSSION

This study sought to validate a novel basketball simulation test and simultaneously demonstrate its sensitivity to meaningful performance effects, thus providing novel insight via the evaluation of common pre-exercise nutritional interventions (i.e. carbohydrate or sodium bicarbonate ingestion). At a group level, the basketball simulation protocol elicited similar absolute physiological responses as did actual game play, although the range of responses observed understandably exhibited less inter-individual variation during the standardised protocol than the freely-paced game situation. The lay-up shooting performance test incorporated directly into the sprint cycles of the exercise protocol revealed both repeated sprint and skill performance to be impaired during the early stages of exercise by pre-exercise carbohydrate ingestion but unaffected by pre-exercise sodium bicarbonate ingestion. However, both these nutritional interventions offset fatigue in that sprinting ability was maintained during the latter stages of exercise, although this did not translate into improved skill performance.

To our knowledge, no previous study has examined the effect of carbohydrate ingestion within the hour prior to exercise involving skilled performance. Consistent with the 'rebound' hypoglycaemia described by Foster et al. (1979), the carbohydrate ingested shortly before exercise in the current study did indeed produce a transient period of reduced blood glucose availability. This is most likely a consequence of decreased hepatic glucose output combined with high rates of both insulin- and contraction-mediated glucose uptake into skeletal muscle. Previous literature has reported blood glucose concentrations below fasted levels to result in more erratic motor control of movement during exercise (Brooke et al. 1982), at least partly due to attenuated activation of muscle contraction by the central nervous system when hypo- *versus* eu-glycaemic (Nybo 2003). It is therefore possible that the changes in carbohydrate availability induced by supplementation may have directly impacted motor control, co-ordination and thus skilled performance.

While every participant in the present study experienced rebound hypoglycaemia to some extent after carbohydrate ingestion, the magnitude of this effect varied substantially between individuals (range 2.4-3.5 $\text{mmol}\cdot\text{l}^{-1}$). This inter-individual susceptibility to rebound hypoglycaemia (and/or the symptoms thereof) is well documented and systematic investigation has neatly revealed how the specific quantity (Jentjens et al. 2003), timing (Moseley et al. 2003) and type (Jentjens & Jeukendrup 2003) of carbohydrate feeding may predict the response within but not between individuals. The precise reason why certain individuals may be more pre-disposed to rebound hypoglycaemia remains largely unclear.

It is interesting that the universal hypoglycaemia ($<3.5\ \text{mmol}\cdot\text{l}^{-1}$) apparent for every basketball player in the present study is somewhat more severe and thus relatively more consistent than the aforementioned reports involving endurance-trained cyclists (Jentjens & Jeukendrup 2003; Jentjens et al. 2003; Moseley et al. 2003). In view of training- and therefore sport-specific adaptations, it is conceivable that the prevalence of rebound hypoglycaemia may vary between different sports. In practical terms, however, any differences in the incidence of rebound hypoglycaemia between sports is more likely to depend on whether a given event provides opportunity for continued carbohydrate supplementation during exercise. Indeed, previous studies into basketball-type activities would advocate carbohydrate ingestion during exercise (Welsh et al. 2002; Winnick et al. 2005). The results of the present study should therefore be interpreted with care, with practical application based on recognition of individual preferences and context (e.g. ingestion of carbohydrate at a different time-point and/or in an alternative form may not elicit the same effects as reported here). Furthermore, this study merely provides proof of concept that supplementation in a fasted state can impact basketball-related outcomes; having now established this in principle, further research is required to examine whether such effects persist within various more ecologically valid conditions as relevant to individual context (e.g. in the fed-state and/or with additional supplementation during exercise).

The intermittent nature of competitive basketball requires a capacity for frequent bursts of intense physical exertion, interspersed with active recovery during lower intensity periods of play. Sodium bicarbonate is therefore a logical supplement to facilitate the removal or buffering of the metabolic by-products that can accumulate during repeated sprints and has indeed previously been shown to improve repeated sprint performance during prolonged intermittent cycling (Bishop et al. 2004; Bishop & Claudius 2005). Here we provide the first evidence that pre-exercise sodium bicarbonate can improve repeated sprint performance during intermittent running, in this case during the final quarter of a simulated basketball game. It is interesting that this effect did not translate into a difference in skilled performance, although the large standard deviation for the placebo group during the fourth quarter (Fig. 2B) reflects the fact that not all participants' performance had begun to deteriorate by that stage, so there was not yet any fatigue to postpone. While it might therefore be speculated that sodium bicarbonate would have improved lay-up shooting performance had the protocol been of increased relative intensity or duration, the validation data also reported here attest that those conclusions would not then apply to basketball.

One practical issue to consider when evaluating sodium bicarbonate supplementation is the potential for adverse gastrointestinal side-effects. As noted, all participants in the present study experienced some degree of gastrointestinal disturbance associated with sodium bicarbonate ingestion and, understandably given that dosing was relative to body mass, these symptoms were most notable amongst larger participants. This issue is highly relevant to how the present results may be generalised across various athletic populations. Specifically, while the present cohort were well trained and highly skilled basketball players, the single factor that best distinguishes this cohort from those at the very highest level is an average height 15-20 cm shorter and mass 15-20 kg lower than an average NBA player (~2.0 m and ~100 kg). It is therefore doubtful that many players at the most elite level could attain the ergogenic benefits reported here without these being outweighed by severe gastrointestinal distress.

Although not a primary focus of this work, an interesting result arising from the monitored basketball game *per se* was the sweat electrolyte losses, which we believe is the first report of this variable during competitive basketball. Notably, salt losses (NaCl) of 7.1±1 g are close to the highest rates reported during outdoor soccer of similar duration (Shirreffs et al. 2006) and this is despite lower environmental temperature and/or humidity during the indoor basketball reported here (i.e. 19.4±0.9°C and 54±4% for games in this study) than for the relevant outdoor comparisons cited above.

In conclusion, the basketball simulation test described here provides a valid reflection of the absolute physiological demands of competitive basketball and also a sufficiently sensitive measure of small but worthwhile changes in both physical and skilled performance. Within the context of this design, ingestion of carbohydrate and/or sodium bicarbonate shortly before basketball has the potential to offset fatigue and thus improve aspects of performance late in exercise, although both supplements require balanced consideration of individual tolerance prior to competition to minimise acute negative side-effects.

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Figure 1 Schematic of the experimental design. LIST denotes a modified version of the Loughborough Intermittent Shuttle Test (LIST) that incorporates a lay-up shot.

Figure 2 Schematic of the exercise pattern (cycle) repeated eleven times during each quarter of the simulated basketball test.

Figure 3 Mean (*s*) lay-ups scored during each 15-min quarter of simulated basketball and mean scores for the entire protocol. Upper panel (A) illustrates data for sucrose (CHO) *versus* placebo (H_2O) and lower panel (B) illustrates data for sodium bicarbonate ($NaHCO_3$) *versus* placebo (NaCl).*denotes differences relative to placebo ($P \leq 0.03$).

Figure 4 Mean (*s*) sprint times during each 15-min quarter of simulated basketball and mean sprint times for the entire protocol. Upper panel (A) illustrates data for sucrose (CHO) *versus* placebo (H_2O) and lower panel (B) illustrates data for sodium bicarbonate ($NaHCO_3$) *versus* placebo (NaCl).*denotes differences relative to placebo ($P \leq 0.02$).

Figure 5 Mean (*s*) whole blood glucose concentrations prior to and at the end of each 15-min quarter of simulated basketball. Upper panel (A) illustrates data for sucrose (CHO) *versus* placebo (H_2O) and lower panel (B) illustrates data for sodium bicarbonate ($NaHCO_3$) *versus* placebo (NaCl).*denotes differences relative to placebo ($P<0.01$).

Figure 6 Mean (*s*) whole blood lactate concentrations prior to and at the end of each 15-min quarter of simulated basketball. Upper panel (A) illustrates data for sucrose (CHO) *versus* placebo (H_2O) and lower panel (B) illustrates data for sodium bicarbonate ($NaHCO_3$) *versus* placebo (NaCl).*denotes differences relative to placebo ($P=0.01$).

TABLE 1 Heart rate, ratings of perceived exertion, pedometer counts and sweat losses and sweat-electrolyte concentrations during the basketball simulation relative to games 1 & 2, along with ratings of perceived exertion for studies A & B (values are means ± SD).

	1st Quarter	2nd Quarter	3rd Quarter	4th Quarter	Overall	r (simulation *versus* games correlation) *P* (simulation *versus* games difference) SEM = Standard Error of Measurement
Protocol Validation						
Heart Rate (beats·min^{-1})						
Game 1	168 ± 12	174 ± 13	171 ± 14	171 ± 13	170 ± 12	0.9 (P<0.001)
Game Simulation	169 ± 13	173 ± 14	170 ± 14	171 ± 15	171 ± 14	P=0.1
Game 2	176 ± 13	171 ± 12	170 ± 12	178 ± 12	174 ± 12	SEM = 4.8 beats·min^{-1}
RPE (6-20 scale)						
Game 1	11 ± 2	13 ± 2	14 ± 2	16 ± 2	14 ± 2	0.1 (P=0.9)
Game Simulation	11 ± 1	14 ± 1	15 ± 2	16 ± 2	14 ± 2	P=0.1
Game 2	12 ± 2	12 ± 2	12 ± 3	14 ± 2	12 ± 1	SEM = 1.4 (6-20 scale)
Pedometer Count (steps)						
Game 1	-	-	-	-	8022 ± 886	0.2 (P=0.7)
Game Simulation	-	-	-	-	8045 ± 247	P=0.3
Game 2	-	-	-	-	7841 ± 682	SEM = 345 steps
Body Mass (kg)	Pre			Post	Sweat Loss (l)	
Game 1	84.0 ± 7.6	-	-	83.3 ± 7.7	-2.1 ± 0.3	0.3 (P=0.4)
Game Simulation	83.0 ± 8.0	-	-	82.2 ± 8.1	-2.3 ± 0.4	P=0.06
Game 2	82.6 ± 9.1	-	-	82.2 ± 8.6	-1.8 ± 0.4	SEM = 0.76 kg
Sweat Na (mmol·l^{-1})						
Game 1	-	-	-	-	59.4 ± 16.1	-
Sweat K (mmol·l^{-1})						
Game 1	-	-	-	-	5.2 ± 1.1	-

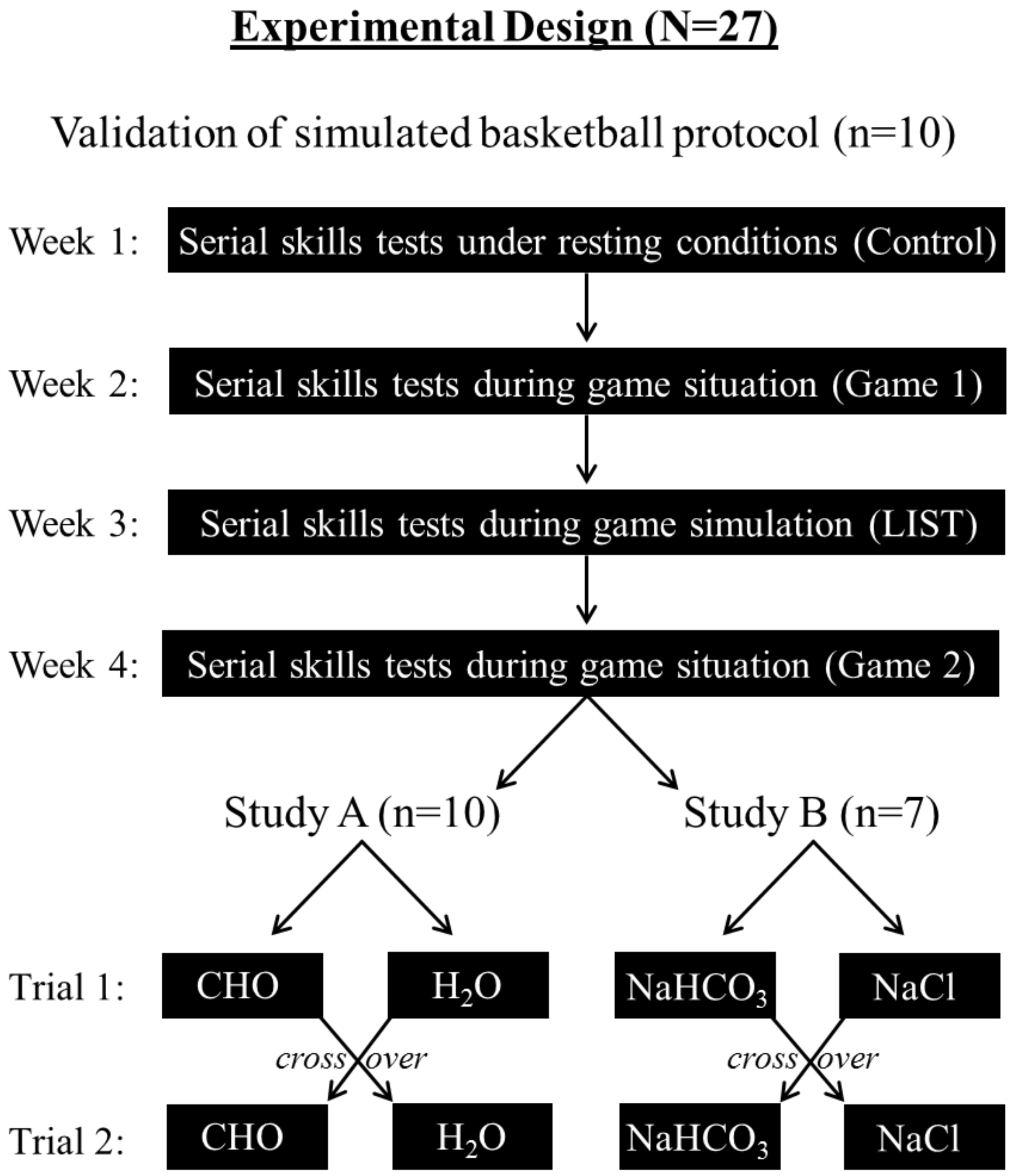
Experimental Design (N=27)
Validation of simulated basketball protocol (n=10)
Week 1:
Serial skills tests under resting conditions (Control)
Week 2:
Serial skills tests during game situation (Game 1)
Week 3:
Serial skills tests during game simulation (LIST)
Week 4:
Serial skills tests during game situation (Game 2)
Study A (n=10)
Study B (n=7)
Trial 1:
CHO
H2O
NaHCO3
NaCl
cross over
cross over
Trial 2:
CHO
H2O
NaHCO3
NaCl

Figure 1

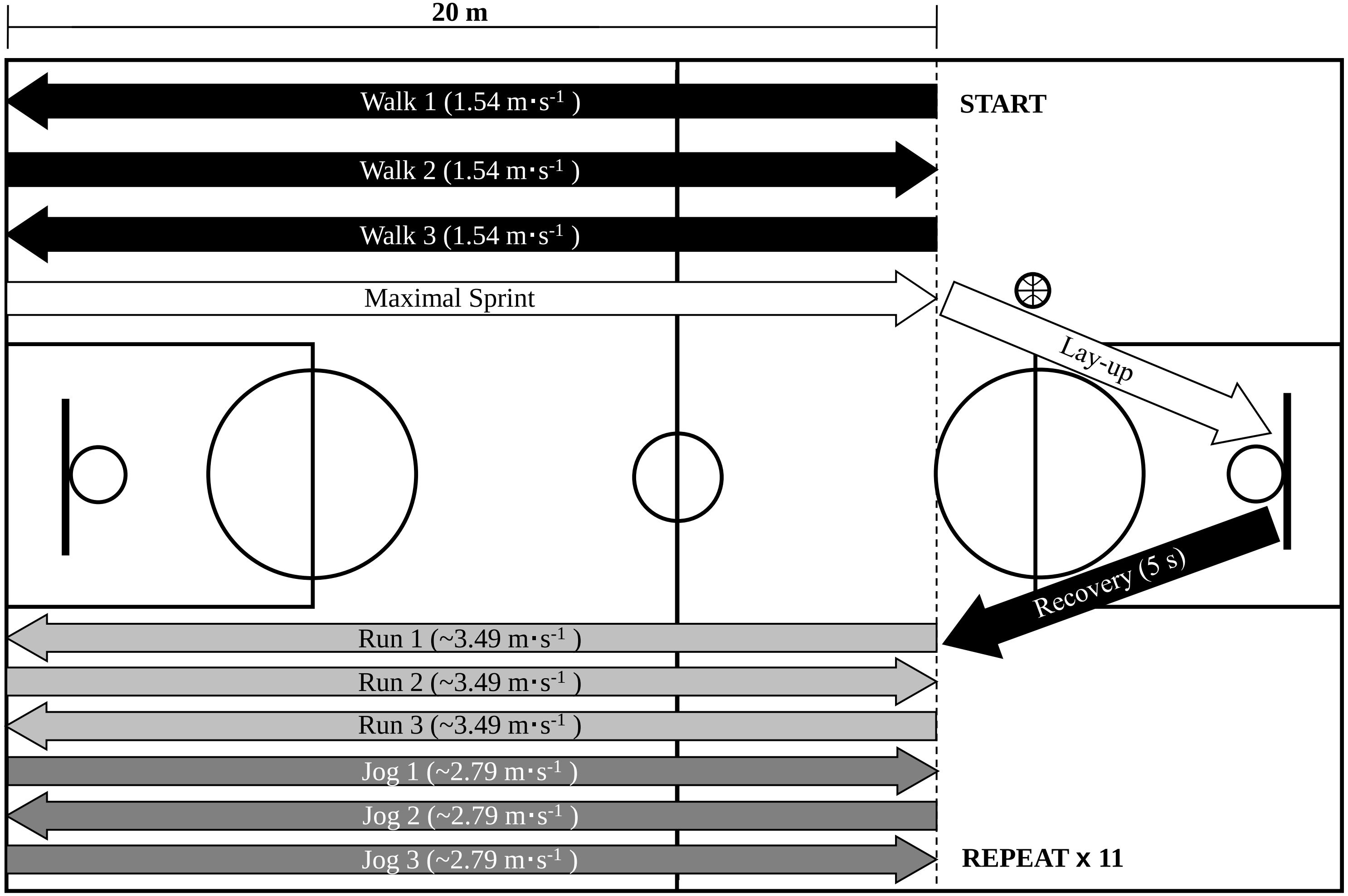
20 m
Walk 1 (1.54 m·s-1)
START
Walk 2 (1.54 m·s-1)
Walk 3 (1.54 m·s-1)
Maximal Sprint
Lay-up
Recovery (5 s)
Run 1 (~3.49 m·s-1)
Run 2 (~3.49 m·s-1)
Run 3 (~3.49 m·s-1)
Jog 1 (~2.79 m·s-1)
Jog 2 (~2.79 m·s-1)
Jog 3 (~2.79 m·s-1)
REPEAT x 11

Figure 2

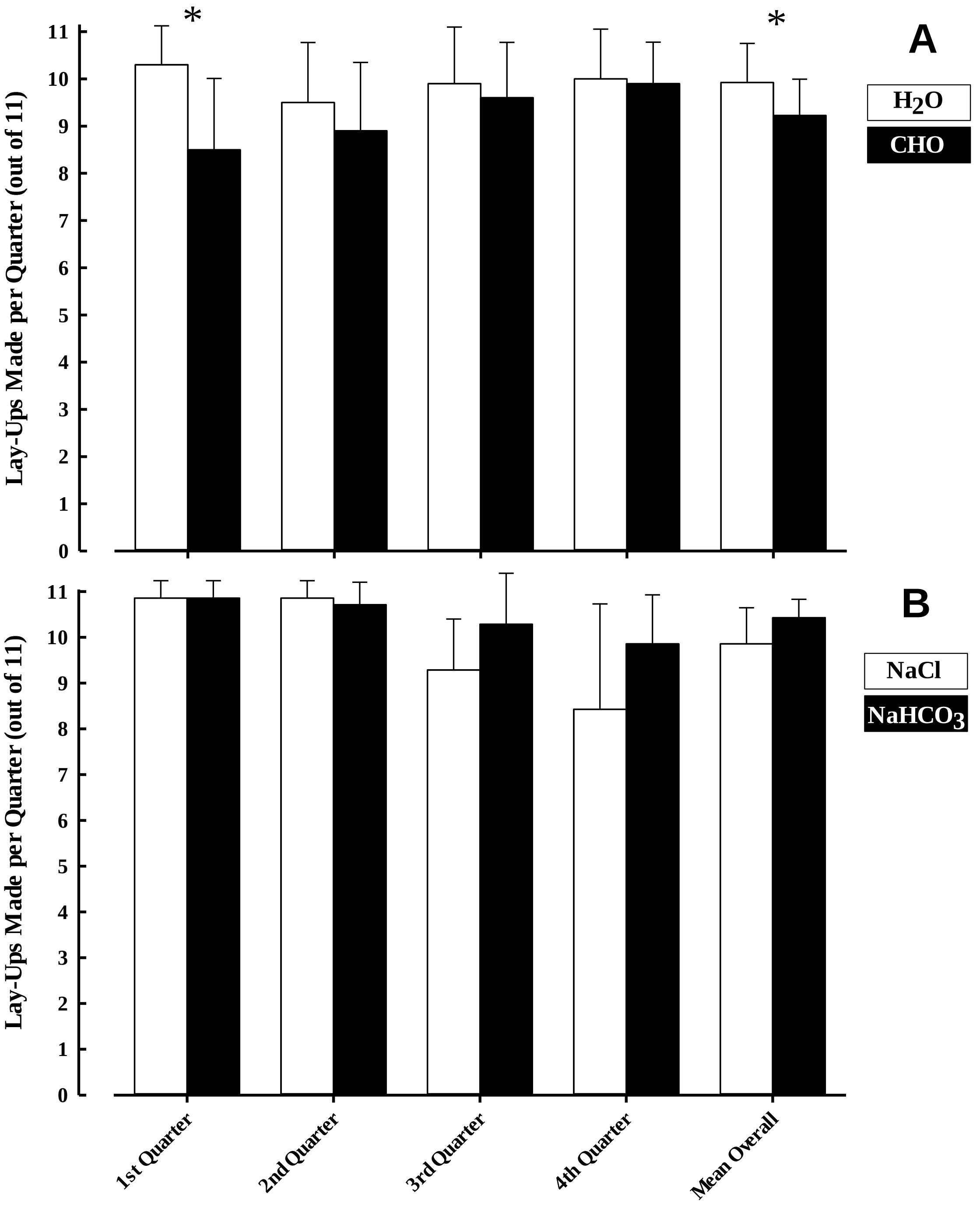
A
B
Lay-Ups Made per Quarter (out of 11)
0
1
2
3
4
5
6
7
8
9
10
11
*
*
H2O
CHO
NaCl
NaHCO3
1st Quarter
2nd Quarter
3rd Quarter
4th Quarter
Mean Overall

Figure 3

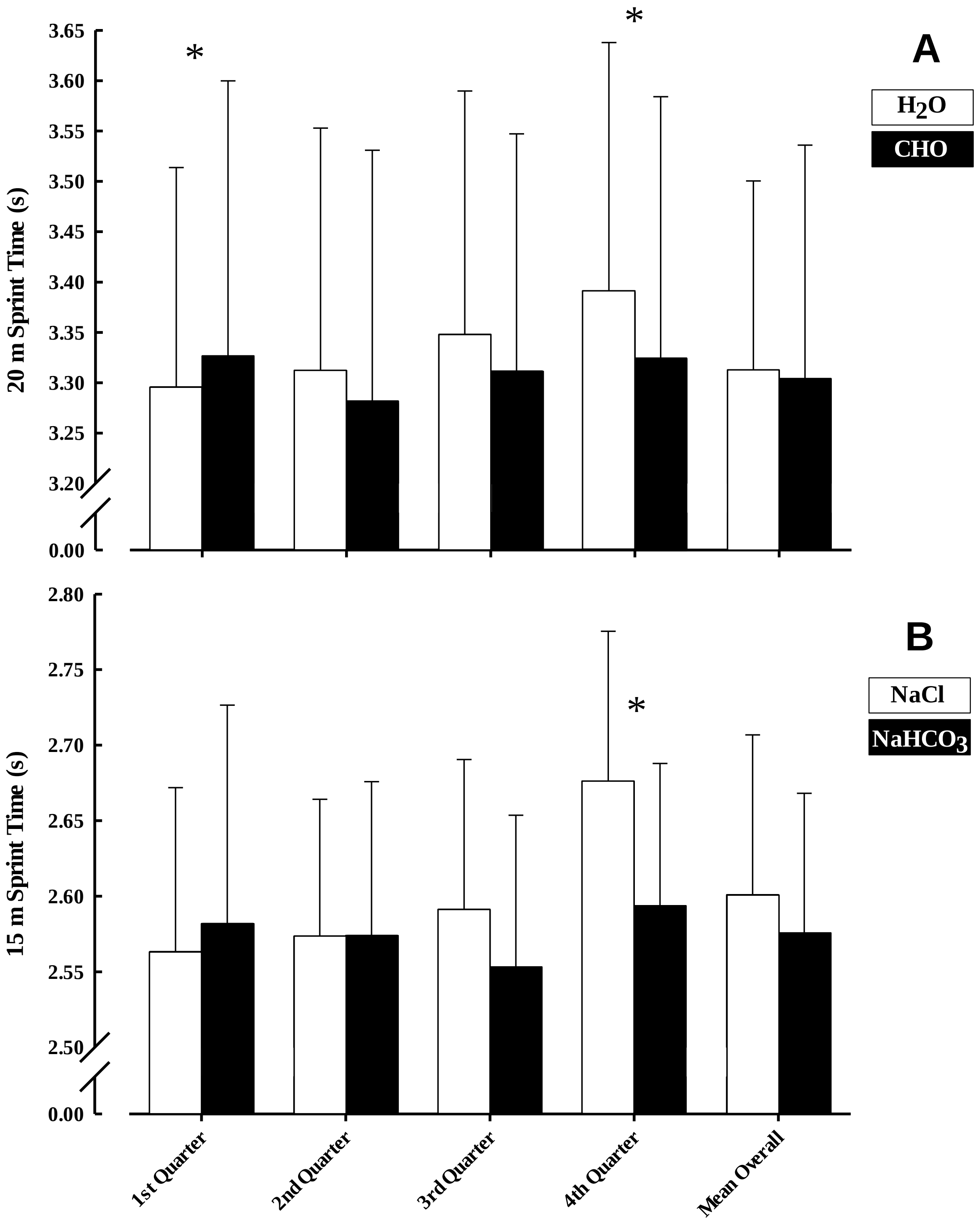
A
H2O
CHO
20 m Sprint Time (s)
3.65
3.60
3.55
3.50
3.45
3.40
3.35
3.30
3.25
3.20
0.00
*
*
B
NaCl
NaHCO3
15 m Sprint Time (s)
2.80
2.75
2.70
2.65
2.60
2.55
2.50
0.00
*
1st Quarter
2nd Quarter
3rd Quarter
4th Quarter
Mean Overall

Figure 4

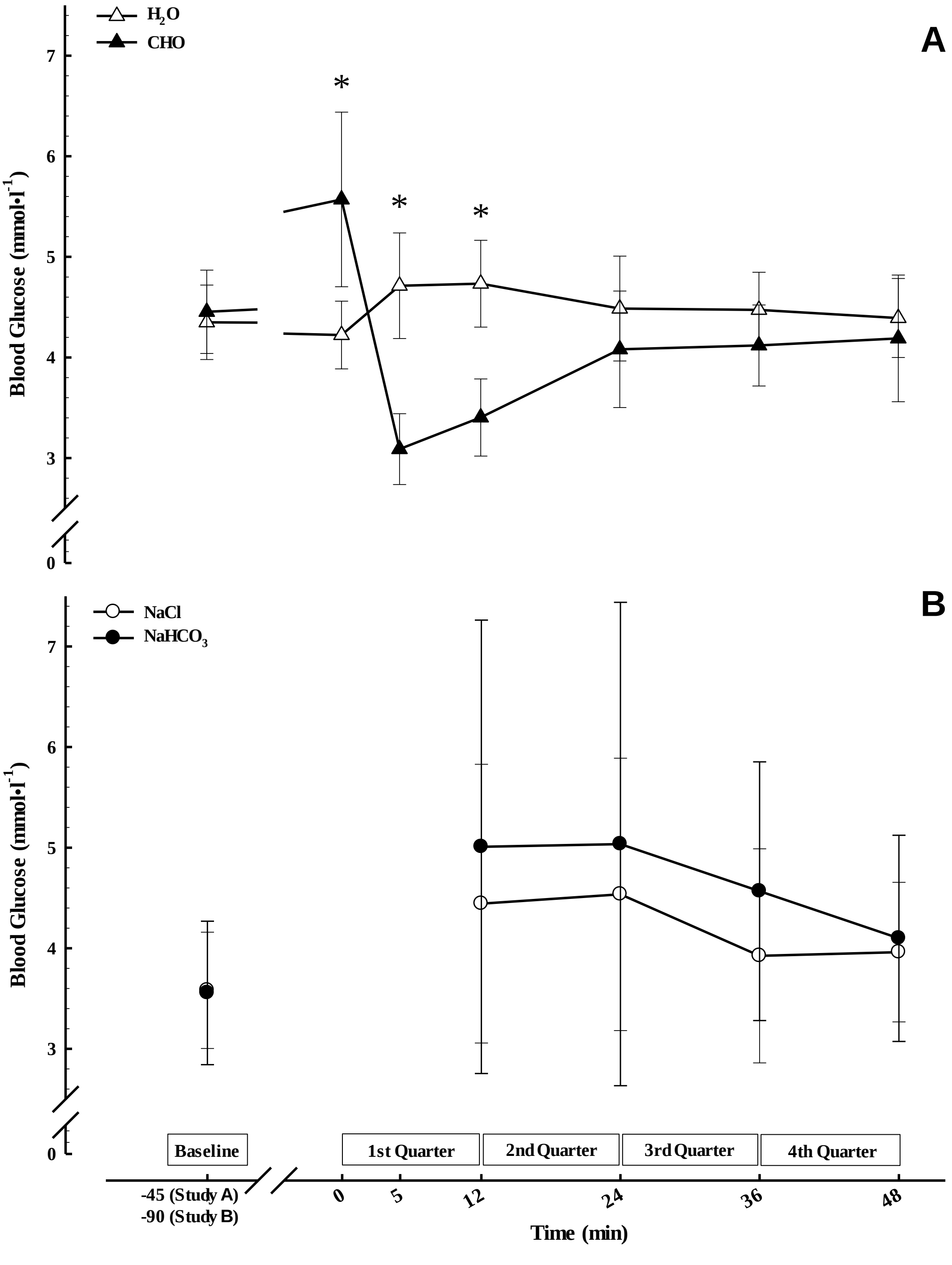
H₂O
CHO
A
*
*
*
Blood Glucose (mmol•l⁻¹)
7
6
5
4
3
0
NaCl
NaHCO₃
B
Baseline
1st Quarter
2nd Quarter
3rd Quarter
4th Quarter
-45 (Study A)
-90 (Study B)
0
5
12
24
36
48
Time (min)

Figure 5

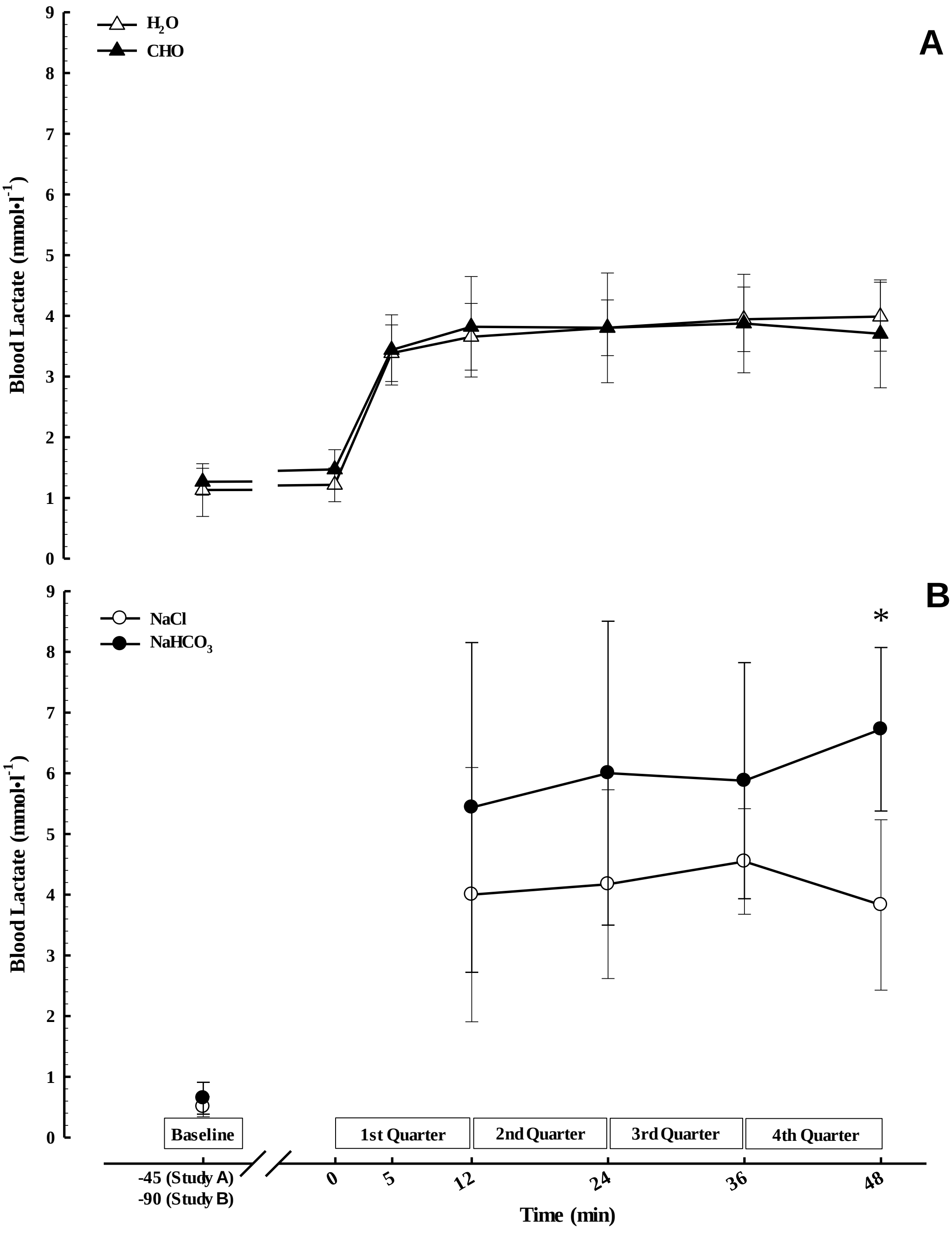
A
B
H_2O
CHO
NaCl
$NaHCO_3$
Blood Lactate (mmol•l^{-1})
0
1
2
3
4
5
6
7
8
9
*
Baseline
1st Quarter
2nd Quarter
3rd Quarter
4th Quarter
-45 (Study A)
-90 (Study B)
0
5
12
24
36
48
Time (min)

Figure 6