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TITLE PAGE

Sodium bicarbonate supplementation delays neuromuscular fatigue without changes in

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performance outcomes during a basketball match simulation protocol

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ABSTRACT

Purpose: To investigate the development of neuromuscular fatigue during a basketball game simulation and ascertain whether sodium bicarbonate ($NaHCO_3$) supplementation attenuates any neuromuscular fatigue that persists. **Methods:** Ten participants ingested 0.2 $g.kg^{-1}$ of $NaHCO_3$ (or an equimolar placebo dosage of sodium chloride [NaCl]) 90 and 60 minutes prior to commencing a basketball game simulation (ALK-T vs PLA-T). Isometric maximal voluntary contractions of the knee extensors (MVIC) and potentiated high (100 Hz) and low (10 Hz) frequency doublet twitches were recorded before and after each match quarter for both trials. In addition, 15 m sprint times and layup completion (%) were recorded during each quarter. **Results:** MVIC, 100 and 10 Hz twitch forces declined progressively in both trials ($P<0.05$) with a less pronounced decrease in MVIC during ALK-T ($P<0.01$). Both 100 and 10 Hz twitch forces were also significantly greater in ALK-T ($P<0.05$). 15 m sprint time increased over the course of both trials (~2%, $P<0.01$); however, no significant condition or time effect was found for layup completion ($P>0.05$). **Conclusion:** A basketball simulation protocol induces a substantial amount of neuromuscular (reduction in knee extensor MVICs) and peripheral fatigue with a concomitant increase in 15 m sprint time over the protocol. $NaHCO_3$ supplementation attenuated the rate of fatigue development by protecting contractile elements of the muscle fibres. **Practical Applications:** This study provides coaches with information about the magnitude of fatigue induced by a simulated basketball game, and provides evidence of the efficacy of $NaHCO_3$ in attenuating fatigue.

KEY WORDS

Alkalosis; muscular fatigue; peripheral fatigue; team sports

INTRODUCTION

Basketball matches are characterised by a large volume of short duration, high intensity movements as shown via time-motion analysis (25). Simulated games can also raise mean oxygen uptake ($\dot{V}O_2$) and heart rate (HR) values to approximately 65% and 85% of their maximum, respectively (25, 28). Due to the elevated metabolic demand of a basketball game, a build-up of deleterious metabolites (i.e. H^+, Pi) may reduce force producing capacity of the working muscles (2). This is deemed neuromuscular fatigue, and is defined in the present work as any transient, exercise-induced reduction in muscular force generating capacity (42), with underpinning mechanisms of peripheral or central origins. Previous research has shown that explosive power and sprint ability is reduced following basketball-related activity (9). However, to our knowledge, no study has yet investigated the time course of various sites of neuromuscular fatigue during a simulated basketball match, and the efficacy of a potentially ergogenic supplement in ameliorating the aforementioned fatigue by reducing metabolite accumulation.

As a result of high intensity exercise (such as basketball), extra and intra-cellular ionic concentrations are altered within the muscles, causing reduced contractile performance. For a complete review of the processes contributing to peripheral fatigue see Allen et al (2). Examples of factors involved in impairment within the muscular contractile apparatus are reduced intracellular potassium ion (K^+) concentrations caused by efflux into the interstitial spaces, resulting in extracellular accumulation (19). This negatively affects the capacity of the sarcolemma to propagate action potentials (27). This potentially occurs as Na^+, K^+ ATPase activity is inhibited by the increased presence of hydrogen ions (H^+) (acidosis).

Similarly, acidosis inhibits myofibril ATPase activity, leading to reduced calcium ion (Ca^{2+}) reuptake to the sarcoplasmic reticulum (SR) and consequently less Ca^{2+} released from the SR when prompted by an action potential (7, 22).

In this study, contractile function was measured using potentiated, electrically-evoked paired twitches at two different frequencies (10 and 100 Hz) in an attempt to refine the sites of peripheral fatigue development. The mechanical response (twitch) to low frequency doublets (10 Hz) has been shown to be modulated by the extent of Ca^{2+} release from the SR (19, 21). High frequency doublets (100 Hz), and their respective twitch amplitude, have been shown not to be affected by moderate decreases in Ca^{2+}. Therefore decreases in high frequency twitch amplitude reflect attenuated action potential propagation caused by extracellular K^+ accumulation (21). The ratio of low:high frequency twitch forces gives detail about the aetiology of contractile decline during a fatiguing task (27).

The role acidosis plays in the development of neuromuscular fatigue during high intensity exercise (such as basketball) remains under debate (10, 44). However, it is generally agreed that athletes who perform high intensity exercise (such as basketball players) would likely benefit from $NaHCO_3$ supplementation (8) as it attenuates the aforementioned negative effects of acidosis. For instance, a reduction in extracellular accumulation of K^+ during exhaustive exercise has been evidenced following $NaHCO_3$ supplementation (40). $NaHCO_3$ supplementation also attenuates the inhibiting effects of H^+ by increasing the intra – extracellular pH gradient. This allows for greater efflux of deleterious metabolites outside the muscle cells and attenuates their harmful effects on the contractile function (26).

$NaHCO_3$ has been shown to enhance high-intensity performance (5, 6) and delay the development of neuromuscular fatigue during a fatiguing task (38). The ergogenic effects found in the aforementioned laboratory-based studies give rationale for the investigation of $NaHCO_3$ as an ergogenic aid during high-intensity team sport activity such as basketball. Interestingly, only a limited amount of studies have investigated the effect of $NaHCO_3$ supplementation on performance outcomes during simulated game based protocols (1, 23, 31, 34), and neuromuscular function has never been assessed throughout a simulated basketball match. Afman et al (1) recently found a beneficial effect of $NaHCO_3$ supplementation on 15m sprint times, but not layup completions, during a modified Loughborough Intermittent Sprint Test (LIST), which was validated to replicate the demands of a 40-min basketball game. Therefore, the present study aims to investigate the development of neuromuscular fatigue and more specifically, the peripheral mechanisms during a basketball game simulation. The study also aims to ascertain whether $NaHCO_3$ supplementation attenuates this development. It was hypothesised that there would be a significant decline in both voluntary force generating capacity of the knee extensors and the amplitude of evoked paired-twitches. It was hypothesised that this decrease in contractile function would lead to faster 15-m sprint times throughout the protocol and with smaller declines in the supplement (ALK-T) compared to the placebo (PLA-T) trial.

METHODS

Experimental Approach to the Problem

Participants visited the laboratories on three separate occasions. Three and seven days separated familiarisation and 1st fatiguing trial, and 1st and 2nd fatiguing trials respectively, to ensure full washout of the supplement/placebo (5). Familiarisation involved a neuromuscular function assessment performed on an isokinetic dynamometer, followed by one block of the modified LIST protocol. For the two experimental trials, participants performed four blocks of the modified LIST with neuromuscular assessment prior to, and following each block of the LIST (1). Participants were asked to avoid consuming any stimulants or alcohol, and to replicate food intake during a 24-hour period before testing. The study was a double-blind crossover design with exposure to supplements randomized and counterbalanced. Each participant received extensive information, and signed an informed consent form and medical questionnaire after they had the opportunity to ask any questions to researchers. The protocol was approved by the University Ethics committee and adhered to the Declaration of Helsinki.

Subjects

Ten healthy and active male basketball players volunteered to take part in the study (age 21 ± 1 years; height: 182 ± 5 cm; weight: 81.5 ± 8.[illegible] kg). All participants had over 4 years of competitive basketball experience.

Procedures

Neuromuscular Function Assessment

For the neuromuscular assessment of the right knee extensors, participants sat on the Con-Trex Multi-Joint system (Con-Trex, Dubendorf, Switzerland) as per the published reliability study (30) (~85° hip angle; distal dynamometer's shin pad attached 2-3 cm proximal to the lateral malleolus with a strap around the shank; straps were fastened and locked across chest and pelvis; movement resisting pad over the mid-thigh of the contracting leg). Knee angle was kept at 90° for all maximal voluntary isometric contractions (MVICs) and twitches.

Torque measurement was corrected to take gravity effect into account. Participants were instructed to cross their arms across their chest and were provided with visual feedback of force during the protocol.

A 48 mm^2 self-adhesive cathode electrode (CF3200, Nidd Valley Medical Ltd, Harrogate, UK) was placed directly over the femoral nerve in the femoral triangle with the anode placed directly onto the greater trochanter of the femur (Prottens, Bio Protech Inc, Korea).

Percutaneous electrical stimulation was delivered by a constant-current stimulator (DS7A, Digitimer, Letchworth Garden City, Great Britain). Stimulations were triggered manually using a PowerLab 15T (Model ML818, AdInstruments Pty Ltd, Dunedin, New Zealand) and force production was recorded using LabChart 7 software (AdInstruments Pty Ltd, Dunedin, New Zealand). Sprint times (15-m) were recorded using wireless electronic timing gates (TC Timing System, Brower, Utah, USA). Participants began each sprint from a standing start 10cm behind the timing gates (see figure 1).

Familiarisation Session

Single electrical 200μs impulses were delivered to the right femoral nerve via the surface electrode. Percutaneous single stimuli were delivered at 10 mA increasing by 10mA until a plateau in twitch force amplitude was reached. This intensity was increased to 130%, to ensure supramaximal stimulations were delivered (mean intensity: 170 ± 35 mA). This process was repeated before each experimental visit. The MVIC familiarisation protocol then consisted of 2 and then 3 × 5-s voluntary contractions performed at 50% and 75% of maximal subjective effort, respectively. The participants then performed 3 × 5-s MVICs. Each maximal contraction was followed by two doublet stimulations (100 Hz and 10 Hz) in 1-s intervals.

Experimental Protocol

Neuromuscular baseline tests were performed followed by a short standardised warm up (a 4length jog of the basketball court). After baseline and warm ups, participants completed four blocks of 11 repetitions of the modified LIST shown in figure 1 (1), meaning 11 sprints and layups were performed per quarter. Participants had 5 minutes rest between quarters, in which neuromuscular fatigue assessment was performed. Three 5 s MVICs with 60 s rest in-between were performed with two doublet stimulations (10 Hz and 100 Hz) following the contractions in 1 s intervals. Due to the time taken to move from basketball court to the dynamometer following each quarter, the timing of the first MVIC was standardised to 75 s.

FIGURE 1 HERE

Supplement

Participants arrived 90 minutes prior to commencement of the protocol in order to consume the first half of either their supplement or placebo; NaHCO$_3$ was delivered in two separate dosages of 0.2 g.kg [illegible] with the second dosage consumed 60 minutes prior. NaHCO$_3$ was dissolved in 500 ml of water and calorie-free cordial each, totalling 0.4 g.kg^{-1}. Sodium chloride (placebo) was composed of two 0.138 g.kg^{-1} dosages dissolved in 500 ml water and cordial each (equimolar amount of sodium to account for alterations in Na$^+$ handling; for more details, see (20)). The same amount of supplement/placebo was consumed 60 minutes prior to exercise. A similar ingestion protocol has been shown to benefit prolonged intermittent activity with no reported incidences of gastrointestinal disturbances following NaHCO$_3$ supplementation, as did the present study (5).

Data Analysis

The maximum 500-ms value was recorded as maximal force for each MVIC plateau, and the peak twitch amplitude was computed for each doublet stimulation. The greatest value over each set of three MVICs and twitches was subsequently recorded for each time point.

Coefficient of variations between the 3 measures were 2.6 ± 2.0% for MVIC, 3.5 ± 2.7% for 100 Hz twitch, and 3.0 ± 2.2% for 10 Hz twitch. Each 15 m sprint time was recorded in seconds (s). Successful completions for the layups were expressed as a percentage of total number of attempts per quarter (out of 11).

Statistical Analysis

Normal distributions were verified (Kolmogorov-Smirnov test) and one-way (1 x 5) repeated measures ANOVAs were run to assess the change in neuromuscular variables (MVC, 100Hz , 10 Hz twitches) and quantify the magnitude of fatigue elicited over the course of the placebo trial (Baseline, Q1, Q2, Q3, Q4). A two-way (2 × 5) repeated measures ANOVAs was performed to test for between condition (ALK-T *vs* PLA-T) and time differences (Baseline, Q1, Q2, Q3, Q4). If sphericity assumption was violated (Mauchly's test) then Fratios were adjusted according to the Greenhouse-Geisser procedure. Significant effects of ANOVAs were followed up using the Bonferroni-corrected pairwise post hoc test.

Significance was accepted at $P \leq 0.05$ and all data is presented as mean ± standard deviation (SD). All statistical analyses were performed using SPSS (version 20, Chicago, USA).

RESULTS

TABLE 1 HERE

FIGURE 2 HERE

MVIC force ($F_{(4,36)} = 42.0$, $P < 0.01$), decreased significantly over time but with no significant difference between conditions ($P > 0.05$) (Table 1 and Figure 2). The loss of MVIC was less pronounced during ALK-T as shown by the significant time × condition interaction ($F_{(4,36)} = 6.88$, $P < 0.01$). However, post-hoc tests did not reveal a significant difference between trials at any time points ($P > 0.05$). The one way ANOVA showed that during PLA-T, the decrement in MVIC ($F_{(4,36)} = 36.9$, $P < 0.01$) was progressive from baseline to the 3rd quarter ($P < 0.05$), with a plateau occurring thereafter ($P > 0.05$).

100 Hz twitch ($F_{(4,36)} = 20.25$, $P < 0.01$) and 10 Hz twitch ($F_{(4,36)} = 24.3$, $P < 0.01$) also decreased significantly over time. No time × condition interaction effect was found for either evoked twitches (100 Hz: $F_{(4,36)} = 0.76$, $P = 0.56$; 10 Hz: $F_{(4,36)} = 1.30$, $P = 0.29$). 100 Hz and 10 Hz evoked twitch forces were both greater throughout the protocol in ALK-T (condition effect: 100 Hz: $F_{(1,9)} = 11.8$, $P < 0.01$; 10 Hz: $F_{(1,9)} = 8.77$, $P < 0.05$). The one way ANOVA showed that during PLA-T, 100 and 10 Hz twitches were not different from baseline ($P > 0.05$) until after the second quarter from which time point a reduction was significant ($P < 0.05$). No time or condition effect was observed for 10:100 Hz twitches ratio ($P > 0.05$).

No condition or interaction effect were found for either of the performance variables ($P > 0.05$) but the 15-m sprint times became significantly slower over both trials (time effect: $F_{(3,27)} = 9.39$, $P < 0.01$). The participants' sprints in both trials were systematically slower from one quarter to the next ($P < 0.05$, Table 1). When comparing first vs last quarter sprint times, both ALK-T and PLA-T were significantly longer (ALK-T: -1.7%, $F_{(3.7)} = 4.3$, $P < 0.01$; PLA-T -2.4%, $F_{(3.7)} = 9.3$, $P < 0.05$).

DISCUSSION

To our knowledge this is the first study reporting development of neuromuscular fatigue during simulation of a basketball match. Maximal force production of the knee extensors (MVIC) during PLA-T reduced throughout the first three quarters of the simulated match (Figure 2; Table 1; ~5% loss per quarter) with no further reduction in the final quarter. Peripheral fatigue was evident from the 2nd quarter of the protocol with disturbances of contractile properties. The ~15% reduction in MVIC torque recorded post 3rd and 4th quarter in this study is similar to the ~15% reduction reported after a 60-min squash match (12), and within the ~11% (11, 13, 29) to ~20% range (16, 17) reported for laboratory based studies investigating repeated sprint activity (4-to 10-s sprints, 8-12 repetitions, 10- to 30-s passive recovery).

To our knowledge, this is also the first study applying femoral nerve stimulations to assess mechanisms of peripheral fatigue during a basketball game simulation. The ~15% reductions in evoked twitch forces from baseline for both doublet stimulations are similar to those previously reported in laboratory-based studies following repeated sprint exercise (9-15%; (29, 32)). In the present study, changes in the several mechanisms of contractile function impairment seem to adopt a similar time course with a decrease after the 2nd quarter, and no further decrease thereafter (apart from one occurrence: 10 Hz twitch between quarter 2 and 3, $P = 0.01$). Perrey et al (29) found decreases of 15% in low frequency (20 Hz) twitch forces but of only 8% in the high frequency (80 Hz) twitch forces following repeated sprints. Their decreased ratio of low:high frequency evoked twitches (−9%) suggested that muscle fibre excitability was the predominant cause for the impairment of the contractile function. In contrast, the present study found decreases of similar extent in low and high frequency evoked twitch forces, suggesting that a basketball simulation protocol affects both excitation and contraction mechanisms to a similar degree.

15-m sprint times increased by ~2% in PLA-T following the basketball simulation protocol, compared to Afman et al (1) who reported a ~5% increase during the placebo trial of the modified LIST. This lies within the 2-10% decrease in sprint times of short distances (≤ 20 m) typically reported for team sport activities (3, 4, 18, 24). These reductions in running performance are greater than losses in 'pure' strength measurements such as MVICs mentioned earlier (~10-20%). This could be explained by a possible change in sprint mechanics in a fatigued state, affecting speed production as a consequence (33). For instance, in the present study, participants were tightly secured on the dynamometer to avoid any extra bodily movements other than the knee extensors, so that MVIC forces could not be affected by a change in technique. Interestingly however, both evoked twitches were significantly greater in ALK-T, demonstrating the protective effect increased extracellular buffering agents have on both potassium and calcium ion-related contractile properties of the muscle fibres of the knee extensors. This protection of the muscle force-generating capacity is further illustrated in the present study by the attenuation in the continuous development of neuromuscular fatigue (MVIC torque) during the protocol under the $NaHCO_3$ supplementation.

Several studies have to date reported the effect of $NaHCO_3$ on neuromuscular fatigue. This was following submaximal isometric calf muscles contractions (36), a 2-min voluntary knee extension (35), tetanic stimulation (39), and high-intensity repeated sprint cycling (38). In agreement with our findings, all found no condition effect on MVIC forces from pre- to postexercise. In contrast with the present results however, the force decline was similar in both alkalosis and placebo trials (35). The differences in the fatiguing protocols and measurement methods might explain these discrepancies. Our basketball simulation protocol engaged a greater muscle mass and was of longer duration so that a time × condition interaction effect was more likely to occur. Stimulations were applied to the posterior tibial nerve to evoke force in the calf muscles in Siegler et al (36), and as suggested by the authors themselves, the relatively low task demand coupled with the small muscle group might have contributed to the lack of pH effect.

The 15-m sprint times were on average 0.2% faster, and MVIC torque 3.3% greater in ALKT (5 measures, $n = 10$). Whilst 7 out of 10 participants recorded lesser decreases in 100 and 10 Hz twitch amplitudes in ALK-T compared to PLA-T, the two-way ANOVA did not depict any interaction effect. A meta-analysis reported for an ergogenic effect of only 1.7% on some performance indicators such as mean power during repeated sprint exercise (8). Several studies also reported a lack of condition effect on 15-m sprint times following ingestion of a buffering agent compared to a placebo (1, 34). Whilst the present study focussed on mechanisms affecting the contractile apparatus, it should be noted that alterations within the CNS may also be responsible for declines in voluntary force. Team sport activity has been shown to induce substantial decreases in voluntary activation of quadriceps muscles(14, 43). $NaHCO_3$ may also attenuate afferent feedback associated with metabolite accumulation (37).

Therefore, the ergogenic effects demonstrated in the present study (i.e. attenuated MVIC force reduction) might not be purely due to protection of contractile mechanisms. Furthermore, NaHCO$_3$ supplementation in the present study was limited by the absence of blood gas measurements; however there is evidence that a similar supplementation protocol to the one used in this study raises [HCO$_3^-$] levels by ~5mmol.L^{-1} and sustains elevated blood pH and HCO$_3^-$ during a prolonged intermittent sprint protocol _ENREF_17(5). Factors such as time to peak [HCO$_3^-$] and [pH] also show high degrees of inter and intra-individual variability (15, 41). Therefore, it is possible that the ergogenic effect seen in the present study may not have been maximal as the ingestion times were standardised to 90 and 60 minutes.

In conclusion, the present study shows a two-phase response in the development of fatigue over time during a simulated basketball match, with an initial early development of neuromuscular and peripheral fatigue after just two quarters of simulated match. Beyond which no further deleterious effect on the neuromuscular function can be seen. This occurred alongside a slowing down of 15-m sprint times while layup scores remained unchanged. The second major finding is that ingestion of sodium bicarbonate 90 and 60 minutes priorexercise attenuates the above-mentioned development of neuromuscular fatigue. Maximal force production can be preserved until the 3rd quarter of the match. The supplementation preserved both potassium and calcium ion-related contractile properties of the knee extensors so that a greater muscular force generating capacity was possible in the alkaline condition when twitches were evoked using paired stimulations of the femoral nerve. This could be the reason maximal force production was preserved during the protocol.

The present findings should be interpreted with caution due to a small sample size weakening overall statistical power. For example, there was a condition effect for MVIC torque alongside a condition *x* time interaction effect, but with no post-hoc difference depicted. This is not uncommon in the literature surrounding $NaHCO_3$ supplementation (36, 38). Another limitation in this study refers to the lack of electromyography (EMG) measurements. The intensity for electrical simulation was therefore based on a plateau of twitch force with increasing current, rather than based on a plateau identified for the compound muscle action potential (M wave). As a result, there is no absolute confidence that all motor units were innervated by the electrical stimulation. However, the mean intensity in the present study (170 mA) is comparable to that of studies using plateaus in twitch and M-wave amplitudes for the determination of stimulation threshold in the knee extensors in similar populations (80-170 mA,(12); ~190 mA, (14)).

PRACTICAL APPLICATIONS

A simulated basketball match protocol causes a significant amount of overall and peripheral fatigue from the 1st and 2nd quarter, respectively, as quantified by neuromuscular assessments. Sprint times are also slower throughout a simulated basketball match. The employment of intelligent substitution timings and tactics should be used to negate this effect.

Supplementing two dosages of [illegible] $g.kg^{-1}$ $NaHCO_3$ 90 and 60 minutes prior to a basketball simulated match protocol can significantly delay the rate of development of neuromuscular fatigue by protecting contractile properties of muscle fibres.

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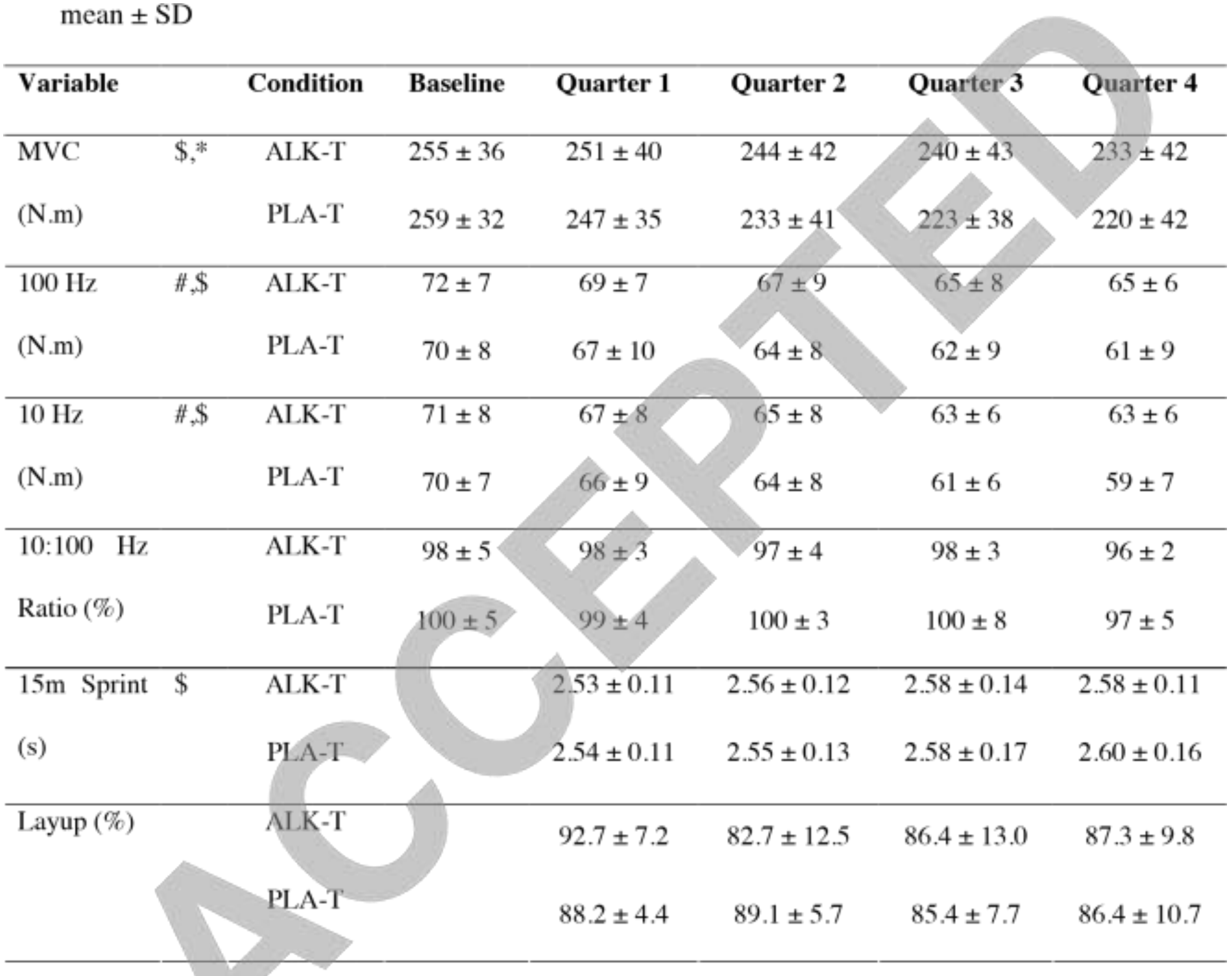
Figure Legends:

Figure 1: Schematic of one quarter of the modified Loughborough Intermittent Sprint Test (LIST).

Figure 2: Neuromuscular function assessment by play ing quarter. Data presented in mean [illegible] SD. A: MVIC force throughout the protocol; B: 100 Hz twitch force; C: 10 Hz twitch force [illegible] Significant group effect; $ Significant time effect; *Significant interaction effect

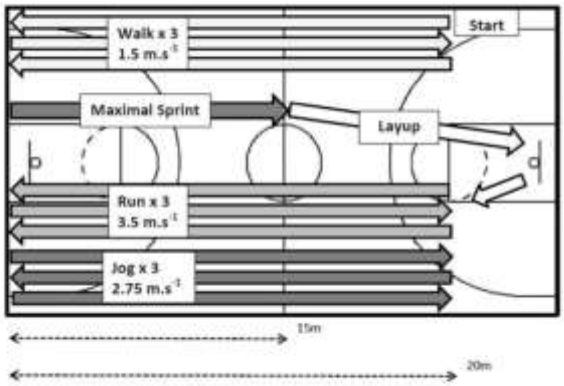
TABLES

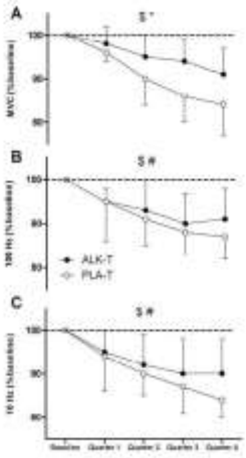
Table 1: Assessment of neuromuscular fatigue and performance indicators at all stages of both conditions (ALK-T: Alkalosis trial; PLA-T: Placebo trial). # Significant group effect; $ Significant time effect; * Significant interaction effect. All P<0.05. All data is presented in



mean ± SD

Variable		Condition	Baseline	Quarter 1	Quarter 2	Quarter 3	Quarter 4
MVC	$,*	ALK-T	255 ± 36	251 ± 40	244 ± 42	240 ± 43	233 ± 42
(N.m)		PLA-T	259 ± 32	247 ± 35	233 ± 41	223 ± 38	220 ± 42
100 Hz	#,$	ALK-T	72 ± 7	69 ± 7	67 ± 9	65 ± 8	65 ± 6
(N.m)		PLA-T	70 ± 8	67 ± 10	64 ± 8	62 ± 9	61 ± 9
10 Hz	#,$	ALK-T	71 ± 8	67 ± 8	65 ± 8	63 ± 6	63 ± 6
(N.m)		PLA-T	70 ± 7	66 ± 9	64 ± 8	61 ± 6	59 ± 7
10:100 Hz		ALK-T	98 ± 5	98 ± 3	97 ± 4	98 ± 3	96 ± 2
Ratio (%)		PLA-T	100 ± 5	99 ± 4	100 ± 3	100 ± 8	97 ± 5
15m Sprint	$	ALK-T		2.53 ± 0.11	2.56 ± 0.12	2.58 ± 0.14	2.58 ± 0.11
(s)		PLA-T		2.54 ± 0.11	2.55 ± 0.13	2.58 ± 0.17	2.60 ± 0.16
Layup (%)		ALK-T		92.7 ± 7.2	82.7 ± 12.5	86.4 ± 13.0	87.3 ± 9.8
		PLA-T		88.2 ± 4.4	89.1 ± 5.7	85.4 ± 7.7	86.4 ± 10.7


Start
Walk x 3
1.5 m.s^{-1}
Maximal Sprint
Layup
Run x 3
3.5 m.s^{-1}
Jog x 3
2.75 m.s^{-1}
15m
20m


A
$ *
100
90
80
MVC (%baseline)
B
$ #
100
80
60
100 Hz (%baseline)
ALK-T
PLA-T
C
$ #
100
90
80
10 Hz (%baseline)
Baseline
Quarter 1
Quarter 2
Quarter 3
Quarter 4