The Effects of Acute and Chronic Sodium Bicarbonate Supplementation on High-Intensity Intermittent Performance, Recovery and Subsequent Performance in Rugby Union Players

Thesis submitted for the degree of Doctor of Philosophy

Paula Fitzpatrick (BSc)

Student Number: 53642844

School of Health and Human Performance
Dublin City University

Supervisor: Dr. Giles Warrington

July 2012

Abstract:

Exogenous ingestion of alkalising agents, such as sodium bicarbonate (SB), has been shown to enhance muscle buffering capacity, thereby delaying the metabolic acidosis associated with high-intensity exercise and potentially improving performance. **Aim:** The aim of this research was to examine the effects of acute and chronic SB supplementation and a placebo (PLA) on high-intensity intermittent performance, recovery and subsequent performance in trained rugby union players. **Methods:** This aim was achieved through the completion of three interconnected studies. Study 1 examined the effects of acute versus chronic SB supplementation on high-intensity intermittent performance as assessed by 6 x 10s maximal sprint tests on a cycle ergometer. Study 2 investigated effects of chronic SB supplementation on a specifically designed field-based, high-intensity intermittent rugby sevens-specific protocol. Study 3 evaluated the effects of acute SB supplementation on an 80-minute high-intensity intermittent 15-a-side rugby-specific protocol using elite females and sub-elite males. **Results:** In Study 1, acute SB supplementation demonstrated significant elevations in pre-exercise levels for blood bicarbonate ($StdHCO_3^-$), pH and base excess (BE-Ecf) but no significant improvement in peak power output (PPO), mean power output (MPO) or total work (TW). Chronic SB supplementation exhibited a significant increase in $StdHCO_3^-$ following Sprint 1. However, no significant differences in performance parameters were recorded for either acute or chronic SB supplementation when compared to the PLA trial. In Study 2, no significant differences in blood or performance related variables were observed between chronic SB and PLA supplementation trials. In Study 3, pre-exercise alkalosis was induced by acute SB supplementation in both elite females and sub-elite males. However, this did not translate into an ergogenic benefit to rugby union-specific performance. Vertical jump height and passing accuracy were significantly improved with PLA as opposed to SB supplementation in the elite female group. No significant differences in performance were observed between trials in the sub-elite male group. **Conclusion:** The major findings of this work suggest that pre-exercise metabolic alkalosis may be induced following acute but not chronic SB ingestion. However, results are inconclusive regarding the efficacy of acute or chronic SB ingestion to enhance performance in high intensity, intermittent performance indicative of the movement patterns and physiological demands associated with rugby union. Results also appear to indicate a high degree of individual variability, which, in part, may be due to potential gastrointestinal side effects of SB ingestion.

Declaration

I hereby certify that this material, which I now submit for assessment on the programme of study leading to the award of PhD is entirely my own work, that I have exercised reasonable care to ensure that the work is original, and does not to the best of my knowledge breach any law of copyright, and has not been taken from the work of others save and to the extent that such work has been cited and acknowledged within the text of my work.

Signed: _____

Paula Fitzpatrick

ID No.: 53642844

Date: _____

Acknowledgements

To my supervisor Dr. Giles Warrington for his encouragement, support, positivity, patience and generosity with his time and extensive expertise. It has been an absolute privilege to be afforded the opportunity to work with him.

To the instigator of the Postgraduate Metabolism Class for sharing his time, knowledge and passion for physiology. Sincerest thanks for making us think on our feet, encouraging us to realise what it is to be a scientist and for helping us to decide "what we want to be when we grow up".

To IRCSET for funding this project.

To all the staff and postgraduate students in the School of Health and Human Performance, thank you for such an enjoyable, supportive atmosphere over the last few years.

To all of the subjects that participated in this research, thank you for your generosity, reliability and considerable time and effort. Particular thanks must go to the members of the Irish and Leinster women's squads for taking time out of their busy schedules to volunteer as subjects for this study.

Finally, to my parents, my sisters Sarah and Clare, and to my friends, particularly the rugby and hockey crews for the fun, friendship, distraction, stress relief and support.

Table of Contents

List of Tables

List of Figures

List of Illustrations

Glossary of Terms

Absolute power output:
Power output expressed in Watts.

Acute SB supplementation:
Ingestion of sodium bicarbonate between 1 and 3 hours prior to exercise.

Chronic SB supplementation:
Ingestion of sodium bicarbonate over a period of days prior to exercise.

Extracellular base excess:
An in vivo expression of base excess. Base excess is the deviation in mmol/L of the buffer base from the normal level in the blood.

Relative power output:
Power output produced relative to body mass, expressed in Watts per kilogram.

Standard bicarbonate:
The concentration of bicarbonate in plasma which is equilibrated with a gas mixture with $PCO_2 = 40mmHg$ (5.3kPa) and $PO_2 \geq 100mmHg$ (13.3kPa) at 37ºC.

Total work:
The product of the mean power output and the duration of the sprint on the cycle ergometer.

List of Abbreviations

ACTH: Adrenocorticotropic Hormone

ANOVA: Analysis of Vvariance

ATP: Adenosine Triphosphate

ATP-PCr: Adenosine Triphosphate-Phosphocreatine

BE-Ecf: Base Eexcess in Eextracellular Ffluid

BM: Body Mmass

BPM: Beats Per Minute

CNS: Central Nervous System

CODS: Change of Direction Speed

CSF: Cerebrospinal Fluid

DCU: Dublin City University

DDS: Differential Descriptor Scale

EPRH: Exercise Physiology Research Hall

GAS: General Adaptation Syndrome

GI: Gastro Intestinal

GPS: Global Ppositioning Ssystem

Hb: Haemoglobin

kJ: Kilojoules

kPa: kilopascal

MJ: Millijoules

MPO: Mean Ppower Ooutput

$NaHCO_3$: Sodium Bbicarbonate

OTS: Over Training Syndrome

PCO_2: Partial Pressure of Carbon Dioxide in Blood

PLA: Placebo

PO_2: Partial Pressure of Oxygen in Blood

PPO: Peak Ppower Ooutput

RAS: Reactive Agility Speed

RPE: Rating of Perceived Exertion

RPM: Revolutions Per Minute

SB: Sodium bicarbonate

SPSS: Statistical Package for the Social Sciences

$StdHCO_3^-$: Standard Bicarbonate

W: Watts

Chapter 1

Introduction

1.1. Background Information and Justification

The professionalism now associated with the game of rugby union has brought with it a more scientific approach to preparation for, and recovery from, competition and training. One key area that has developed as a result of the increasing demands placed on players is nutrition along with the use of ergogenic aids. Nutritional interventions are widespread in sport, with the majority of athletes incorporating some form of nutritional strategy into their training to optimise performance. This may range from simple healthy dietary choices for the amateur athlete to supplementation with ergogenic aids, such as creatine, for elite competitors. Adequate nutrition between bouts of high-intensity exercise is essential to optimise performance, recovery and subsequent exercise performance. In rugby union, the incentive to obtain even the most marginal advantage over opponents has lead to a growing culture of supplement use both among professionals and in the amateur game. However, particularly in an amateur rugby union environment, sports nutrition information is often delivered by an unqualified source. Information on supplement use is often based on anecdotal evidence enforced by collective team practices and not on definitive scientific evidence (Burke, 2003).

Sodium bicarbonate ($NaHCO_3$ or SB) is a buffer which occurs naturally in the body. Exogenous supplementation with SB prior to exercise has been shown to increase pH levels and standard blood bicarbonate ($StdHCO_3^-$) concentrations, thereby enhancing the extracellular buffering capacity (Requena et al., 2005). During high intensity exercise, hydrogen ions (H^+) are formed as a by-product of anaerobic glycolysis (Medbo et al., 1985). This increase in $[H^+]$, and consequential decrease in pH, leads to inhibition of calcium (Ca^{2+}) release from the sarcoplasmic reticulum (Favero et al., 1995), inhibition of the binding of actin and myosin (Fabiato and Fabiato, 1978) and inhibition of enzymatic activity involved in energy production (Trivedi et al., 1966). This inhibition of cellular processes results in a diminished capacity for muscular force production leading to fatigue (Hawley and Reilly, 1997). By creating a state of pre-exercise alkalosis within the body, SB is purported to have the capacity to delay acute fatigue caused by exercise-induced metabolic acidosis (McNaughton, 1992; Jones et al., 1977; Wilkes et al., 1983).

SB ingestion has been shown to improve performance in high intensity exercise of 1-7 minutes duration (McNaughton, 1992; Goldfinch et al., 1988). However, little is known regarding the effect of SB on repeated sprint-ability and high intensity intermittent activity comparable to the type of movement, duration and intensity necessary in the performance of team field sports such as rugby union. Therefore, the aim of this research is to investigate the effects of nutritional supplementation with SB on high-intensity intermittent sprint performance indicative of the physiological demands of rugby union. The research will also aim to examine the effects of SB on recovery from exercise and subsequent high-intensity repeat-sprint performance.

1.2. Purpose of the Research

In modern sport, there is widespread use of nutritional supplements in team field sports, including rugby union. However, there is a dearth of scientific evidence to verify the efficacy of all supplement use in promoting performance enhancements, physiological recovery after exercise and improvements in subsequent performance. Much of the current research in the area of SB supplementation concerns single bout events or laboratory based experiments with little emphasis on the response to SB supplementation in a team field-based sport-specific setting. In addition, the investigation of the effect of SB on recovery from exercise within the literature rarely extends beyond post-exercise monitoring of blood parameters, such as $StdHCO_3^-$, pH, base excess (BE-Ecf) and lactate, and seldom expands to examination of subsequent performance. Few studies have considered pre-exercise SB ingestion as a potential recovery modality capable of enhancing subsequent performance (Siegler et al., 2010).

The purpose of this research, therefore, is to enhance the limited body of knowledge concerning SB supplementation in high intensity intermittent exercise indicative of team sport performance. In addition, this investigation aims to provide coaches and athletes with a fact-based approach to SB supplementation regarding its capacity to improve

performance and optimise recovery for enhanced subsequent performance. As part of this research, three studies were conducted and are outlined below:

Study 1: The effects of acute versus chronic SB supplementation on high-intensity intermittent sprint performance.

Study 2: The effects of chronic SB supplementation on a high-intensity intermittent rugby sevens-specific protocol.

Study 3: The effects of acute SB supplementation on an 80-minute high-intensity intermittent 15-a-side rugby-specific protocol using elite females and sub-elite males.

1.3. Research Aims and Objectives:

To examine the effects of acute and chronic sodium bicarbonate ($NaHCO_3$ or referred to as SB when referring to ingestion) supplementation on high-intensity intermittent performance, recovery and subsequent performance in rugby union players.

Aims:

1. To examine the effects of acute versus chronic SB supplementation on a controlled laboratory experiment involving high-intensity intermittent sprint cycling performance.
2. To investigate the effects of chronic SB supplementation on a field-based high-intensity intermittent 7s rugby match-specific protocol.
3. To analyse the effects of acute SB supplementation on a field-based 80-minute high-intensity intermittent 15s rugby match-specific protocol involving elite females and sub-elite males.

Overall Objectives:

1. To examine the effects of acute and chronic SB supplementation on group and individual performance indices.
2. To examine the effects of acute and chronic SB supplementation on the various biochemical parameters including $StdHCO_3^-$, pH, BE-Ecf and lactate.
3. To investigate the potential relationship between the aforementioned biochemical parameters and performance.
4. To analyse the impact of acute and chronic SB supplementation on heart rate and subjective ratings of perceived exertion, muscle soreness and gastro-intestinal discomfort and their potential relationship to performance.

Overall Hypothesis:

Acute and chronic SB supplementation will significantly enhance high intensity intermittent performance, recovery and subsequent performance in both laboratory and field-based tests indicative of the movement demands observed in rugby union when compared to a placebo (PLA).

1.4. Study One: The Effects of Acute versus Chronic SB Supplementation on High-Intensity Intermittent Sprint Performance

Aim:

The aim of this study was to examine the effects of both acute and chronic SB supplementation versus PLA on high-intensity intermittent sprint performance in sub-elite male rugby players.

Hypothesis:

Acute and chronic SB supplementation will induce sufficient elevations in $StdHCO_3^-$, pH and BE-Ecf to delay fatigue and result in enhanced performance of high intensity intermittent sprints on a cycle ergometer when compared to a PLA trial.

1.5. Study Two: The Effects of Chronic SB Supplementation on Simulated Rugby Sevens Performance

Aim:

The aim of this study was to examine the effects of chronic five-day supplementation with SB on a specifically designed test protocol developed to replicated the specific physiological demands associated with a typical rugby sevens match.

Hypothesis:

Chronic SB supplementation, using a specifically designed loading protocol, will stimulate metabolic alkalosis and result in enhanced performance, recovery and subsequent performance of high-intensity intermittent performance when compared to a PLA trial.

1.6. Study Three: The Effects of Acute SB Supplementation on High-Intensity Intermittent Performance Using a Simulated 80 minute Rugby-Specific Test Protocol in Elite Females and Sub-Elite Male Rugby Players

Aim:

The aim of this study was to examine the effects of acute SB supplementation versus a PLA on high-intensity intermittent performance using a specifically designed test protocol developed to simulate typical demands of 80-minute rugby union match.

Hypothesis:

Acute SB supplementation will cause a significant increase in $StdHCO_3^-$, pH and BE-Ecf and result in enhanced high intensity intermittent performance, recovery during the 10 minute half time interval and subsequent performance when compared to a PLA trial.

1.7. Delimitations

- All tests were administered by the author in order to avoid inter-tester error.
- Subjects were restricted to male or female rugby union players aged 18-35 years.
- Male subjects were required to be current club rugby union players of level J2 or above. Female subjects were required to be current interprovincial or international rugby union players.
- Subjects were excluded from the study if they were suffering from any cardiovascular, pulmonary or metabolic diseases; if they were smokers; or if they had any injury or illness that may have affected their performance, as determined by the general health questionnaire (*Appendix B*) completed by each subject prior to testing.

1.8. Limitations

- Subjects were under specific instruction to maintain their normal diet; not to consume any form of performance drinks, ergogenic aids or alcohol and to avoid strenuous physical exercise (described as exercise requiring great effort, energy or exertion) for the 48 hours prior to each test. However, these criteria may not have always been adhered to.
- Despite the familiarisation protocol, possible learning effects may have affected behaviour in the experimental conditions due to the repeated testing involved in this research. However, a randomised experimental design was employed to negate these effects as much as possible.
- The limited number of subjects (10 subjects for Study 1 and 2, 20 subjects for Study 3) involved in each study may have diminished the statistical power of the study and therefore reduced the likelihood of significant findings.
- Rugby union is a high-impact, collision sport with a high incidence of injury resulting in a number of subjects having to withdraw from the study.
- Arterialised capillary blood samples were used to measure blood gases indicative of arterial blood. However, due to field based testing and an inability to accurately heat the earlobe to a designated temperature, the sample obtained may have been composed of a mixed arterial-venous sample and as a result the PO_2 values may have been slightly compromised.
- Measurements for $StdHCO_3^-$, pH, BE-Ecf, PO_2, PCO_2 and lactate were obtained from the blood and not the muscle. Intracellular measurements may have provided more accurate results relevant to the working muscle. However, it was deemed that muscle biopsy was impractical in a field based setting.

Chapter 2

Review of Literature

2.1. Introduction

Sports performance is affected by many factors, of which nutrition may be one of the most influential (Maughan and Burke, 2011). Adequate nutrition between bouts of high-intensity exercise is essential to optimise subsequent exercise performance. In order to meet the physiological demands of exercise combined with the challenges of modern living, there is a trend towards the use of various ergogenic aids by both recreational and elite athletes. Sodium bicarbonate (SB) is one such nutritional ergogenic aid that has the potential to enhance high-intensity anaerobic exercise performance. However, little is known regarding the effect of SB on repeated sprint-ability comparable to the type of movement patterns, duration and intensity of exercise necessary in the performance of team field sports such as rugby union. The aim of the current research, therefore, is to investigate the effects of sodium bicarbonate supplementation on high-intensity, intermittent sprint performance, recovery and subsequent repeat sprint performance in rugby union players. This review of literature will provide an overview of the research in the area of SB supplementation, with a specific focus on its effect on high-intensity intermittent exercise, recovery and subsequent exercise performance. The evidence surrounding the various mechanisms and supplementation protocols in terms of dosage and timing of ingestion of SB will also be presented, as demonstrated in the current scientific literature.

2.2. Physiological Demands of Rugby Union

Due to the growing culture of supplement use in the sport at present, rugby union was chosen as the selected field-based team sport for this research. With the advent of professionalism in rugby, a more scientific approach to performance has been adopted. Increases in the volume of matches, along with the demands and intensities of those games, culminate in escalating physiological stress being placed on elite players. As a result, optimal nutrition, both in the preparation for and recovery from exercise, is considered as important to performance as the training stimulus itself. The use of various

supplements, including SB_4 among individuals, and in particular in both amateur and elite team settings is becoming increasingly prevalent. However, there is a lack of comprehensive research to justify this practice (Burke, 2003).

Rugby union is contact, field-based team sport which consists of two 40-minute halves separated by a 10-minute half-time period. Fifteen players from each team contest play on a field approximately 100m long and 70m wide. The fifteen players on each team are divided into eight forwards and seven backs, with each of the fifteen positions possessing distinct roles, physical characteristics and physiological attributes. This heterogeneous feature of the sport is unique to rugby union in comparison to most other field-based team sports. However, from movement analysis, measurement of physiological parameters such as heart rate, blood lactate, blood glucose and muscle glycogen, the physiological responses to rugby union are typical of many team sports with a range of work intensities, durations and recovery periods (Quarrie et al., 1996).

According to match analysis by Nicholas (1997), "rugby is an interval or intermittent sport and players must be able to perform a large number of intensive efforts of 5 to 15 seconds duration with less than 40 seconds recovery between each bout of high intensity activity." Research by Deutsch et al. (1998) purported that rugby players can cover 6km during a game. The mean distance that a player will sprint is 15m, and the total distance sprinted during competition ranges from 100m (forwards) to 350m (backs). Players run at near-maximal pace for an additional total of 370m (forwards) and 550m (backs). Although there is a considerable variation in demands relative to each position on the field, all rugby players require high levels of strength, speed, power and endurance (Gabbett, 2006; Gabbett, 2007; Duthie et al., 2003). As a result, demands are placed on both the anaerobic and aerobic systems.

The work to rest ratios of 1:5.7 reported by Cunniffe et al. (2009) were based on GPS tracking of just two players (one loose forward and one inside back) during one professional Celtic league rugby union match. Time motion analysis of the demands of rugby union by McLean et al. (1992) argued the most frequent work to rest ratios

observed measured during professional rugby union over five games throughout the 1989-1990 Five Nations Championship were 1:1-1.9 and 1-1.9:1. Deutsch et al. (1998) substantiated this to some extent, identifying a mean work to rest ratio of 1:1.4 and 1-2.7 for under-19 forwards and backs, respectively. However, the latter two studies were conducted using time motion analysis and as a result may not be comparable to the global positioning systems (GPS) based work of Cunniffe et al. (2009). It would appear that the work to rest ratios performed during rugby union remain disparate depending on the method of assessment. Time motion analysis is a valuable tool used to quantify the physiological demands of sport. However, due to the dynamic, open nature of rugby union, the simplification of movements into categories may prevent accurate comparisons with other method such as GPS analysis.

GPS analysis by Cunniffe and colleagues (2009) also indicates that backs travel greater distances (7.6% further) during a game than their forward counterparts. However, both forward and back position players recorded greater running distances in the second half of the game. This would suggest that depletion of energy reserves was not a concern. However, the half-time recovery practices undertaken were not outlined in this article. Match analysis reported by Deutsch et al. (1998) has shown that the mean distance that rugby players sprint is between 10 and 20 metres. Therefore, acceleration speed over 10-20m will indicate a player's sprint ability specific to the demands of a typical game. From a review of the demands on New Zealand rugby players, it has also been suggested that metabolic demands on the players may differ depending on the environment, the level of play, the style of referee and the tactical component of the game (Quarrie et al, 1995). Given these considerable demands being placed on rugby players, pre-exercise nutrition is an evolving area viewed as a key component of performance.

2.3. Rugby Sevens

Rugby sevens is a seven-a-side variation of rugby union, played under essentially the same laws and on a playing field of the same dimensions as 15s rugby. Rugby sevens matches consist of two seven-minute halves with a half-time period of 2 minutes. Sevens tournaments typically involve five or six matches and are usually completed over the course of one day or a weekend. Sevens rugby is a highly popular version of the game worldwide and is now recognised as an Olympic sport making its debut in the 2016 Summer Olympic Games in Rio de Janeiro, Brazil. Only limited research relating to rugby sevens currently exists, with the majority of studies focusing on injury incidence. Prior to Olympic inclusion, just one journal article existed relating to the physiological demands of rugby sevens (Rienzi et al., 1999). However, the 2009 announcement of upcoming Olympic inclusion has prompted three more scientific articles to be published analysing the movement patterns, physiological demands and anthropometric profiles of rugby sevens players.

Using GPS analysis, Higham et al., (2012) conducted a characterisation and review of the movement patterns of 19 international level male rugby sevens players. From comparison of this analysis with GPS analysis from an 83 minute 15-a side rugby union game (Cunniffe et al., 2009a), rugby sevens was found to be played at a substantially greater running intensity. The rugby sevens players covered approximately 45% greater total distance per minute at a significantly greater velocity (135% greater distance covered at >5m/s). Unsurprisingly, Higham et al., (2012) also observed that in relative terms international sevens matches required greater maximum running velocities, greater distances covered at high velocity and a greater number of changes in velocity when compared to domestic sevens matches. Given the greater relative running loads associated with sevens rugby when compared to its 15-a-side rugby counterpart, and with international sevens as opposed to domestic sevens, the necessity to maintain performance and resist fatigue is seemingly most pronounced in international sevens rugby. As a result, this environment may provide the greatest scope for investigation of the effects of SB on performance.

A combination of GPS analysis and heart rate responses were used to assess running demands on seven male domestic sevens players by Suarez-Arrones et al. (2011). It was found that that during the course of a 14 minute game, players covered a mean distance of 1580±146.3m resulting in a mean running velocity of 6km/h. This is in contrast to a 15-a-side rugby game, over 80 minutes, which has reported mean distances covered of 6953m at an mean velocity of 4.2km/h (Cunniffe et al., 2009). Of this mean distance of 1580m observed for sevens rugby (Suarez-Arrones et al., 2011), standing and walking, jogging and cruising comprised 34.8% (549.7±79.1 m), 26.2% (414.8±105.1 m) and 9.8% (154.6± 53.5 m) respectively. The remainder of the distance was covered by 15.5% striding (244.5±80.1 m), 5% high-intensity running (79.5±37.2 m) and 8.7% sprinting (137.7±84.9 m). Suarez-Arrones et al. (2011) also found that seven players' work to rest ratio was 1:0.5 and players spent approximately 75% of the 14 minute game at a heart rate greater than 80% of their maximal heart rate.

Suarez-Arrones et al., (2011) also investigated the running demands and heart rate responses in elite female international sevens players with similar findings. The female sevens players also operated at an intensity greater than 80% of their maximal heart rate for approximately 75% of a 14 minute game. Female players covered an mean distance of 1556.2+/-189.3 m per game, which was composed of 29.7% (462.6+/-94.6 m) standing and walking, 33.2% (515.9+/-88.6 m) jogging, 11.6% (181+/-61.4 m) cruising, 16.4% (255.7+/-88.3 m) striding, 3.7% (57.1+/-40.8 m) high-intensity running, and 5.4% (84.0+/-64.8 m) sprinting. The work to rest ratio for female international sevens players was 1:0.4, which along with the data revealed for the male players, highlights the difference in physiological demands of rugby sevens compared to other rugby codes such as 15-a-side rugby where work to rest ratios of 1:1-1.9 (Duthie et al., 2003) and 1: 5.7 (Cunniffe et al., 2009) have been previously reported.

2.4. Fatigue and High Intensity Intermittent Exercise

Fatigue is defined as an inability to maintain a given force or power output (Maughan et al., 1997) and is a multifactoral process (Juel and Pilegaard, 1998). Several components may contribute to fatigue including substrate availability, metabolic by-products, muscle fibre contractile properties and nervous system alterations (McArdle et al,. 2010). High intensity intermittent exercise requires a high turnover of skeletal adenosine triphosphate (ATP). Given that intramuscular ATP stores are capable of sustaining muscular contraction for only a few seconds, ATP must be resynthesised through anaerobic glycolysis and phosphocreatine degradation (Hollidge-Horvat et al., 2000). During a rugby union game there is a contribution to muscular activity from all three energy systems – 1. ATP-PCr system, 2. Anaerobic glycolysis and 3. Oxidative phosphorylation (Nicholas, 1997). The relative contributions of each energy system are dependent upon both exercise intensity and duration at any given time point within a rugby game. Typically, the contribution of the adenosine triphosphate-phosphocreatine (ATP-PCr) system may be greater during high intensity, short duration bursts of play. However, energy during low intensity periods of the game may be supplied predominantly by the oxidative system.

Primarily characterised by short-duration, high intensity efforts interspersed with low intensity active recovery and rest periods, rugby union relies heavily on both the ATP-PCr system and anaerobic glycolysis for energy production (Deutsch et al., 1998). Spencer et al. (2005) reported that following 10 second maximal sprint activity, the initial source of ATP is predominantly supplied by ATP degradation and glycogenolysis, particularly in type II muscle fibres. In addition, it is purported that the depletion of PCr within these fibres it is a main contributing factor to fatigue in high-intensity, short duration exercise (Spencer et al., 2005). Resting intramuscular ATP stores are usually reported to be approximately 20-25mmol/kg dry mass (Gaitanos et al., 1993). During 30 second sprint exercise, muscle ATP depletion appears restricted to within approximately 45% of resting levels (Boobis et al., 1982; Cheetham et al., 1986). The fact that muscle

ATP levels are mostly maintained during maximal exercise results in significant PCr depletion and illustrates the importance of PCr as an energy buffer (Medbo et al., 1999).

It has been reported that following 30 seconds of maximal exercise, resting PCr stores are depleted by up to 60-80% (Medbo et al., 1999). PCr depletion during 2.5 seconds of electrical stimulation was shown to be only 26% (Hultman and Sjoholm, 1983). As a result, sprinting specific to team field sports (2-3 second in duration) is likely to involve anaerobic glycolysis along with PCr degradation. The PCr recovery rate is dependent, in part, on the kinetic mechanisms of the catalytic enzyme, mitochondrial creatine kinase (Malucelli et al., 2011). Low ATP concentration and reduced intramuscular pH following high intensity exercise is believed to contribute to fatigue by slowing the rate of PCr resynthesis (Spriet et al., 1989). According to Bogdanis et al. (1996), the ability to produce high power outputs is directly correlated to the resynthesis of PCr. During team sport performance involving typical recovery periods of between 20 and 30 seconds, full PCr resynthesis is not possible. Therefore, there is an increasing reliance on anaerobic glycolysis and the aerobic energy system (Spencer et al., 2005). Furthermore, creatine supplementation has been shown to induce an increased rate of PCr resynthesis and therefore, delayed fatigue. This is attributed to a reduction in inorganic phosphate and an increased intramuscular pH (Yquel et al., 2002).

Glycolysis is associated with hydrogen ion (H^+) accumulation which causes both muscle and blood pH to decrease (Medbo et al., 1985). This increased acidosis (decreased pH) causes a deceleration in glycolysis due to impaired enzyme activity (Trivedi et al., 1966; Ui, 1966) and interference with calcium release from the sarcoplasmic reticulum, leading to fatigue (Favero et al., 1995; Nakamura and Schwarz 1970). In order to maintain homeostasis and potentially delay the onset of fatigue, there must be a balance between the formation and removal of H^+. The ingestion of sodium bicarbonate prior to exercise may increase blood buffering capacity and delay the accumulation of H^+ (Peart et al., 2012).

2.5. Acid-Base Balance

Acid-base balance refers to the state of equilibrium between acidity and alkalinity in the blood (Filley, 1971). Hydrogen ions (H^+) play a central role in maintenance of the acid-base balance. Acids dissociate in solution, donating H^+, whereas bases accept the H^+ to form hydroxide ions (OH^-). pH is used as the measure to express the activity of H^+ in the blood and, therefore, the acid-base balance (Aerenhouts et al., 2011). Due to the fractional numbers associated with hydrogen ion concentration (0.00000004 equivalents/L), pH is defined as minus the decimal logarithm of the hydrogen ion concentration $[H^+]$ in a solution (Guyton and Hall, 1996):

$pH = \log(1/[H^+]) = -\log[H^+]$
$pH = -\log[0.00000004]$
$pH = 7.4$

$[H^+]$ and pH are inversely related, therefore an increased $[H^+]$ in the blood corresponds to a decrease in pH. Conversely, a decreased $[H^+]$ will equate to an increase in pH. The $[H^+]$ or arterial blood pH must be maintained near to or within the normal range of 7.35-7.45 pH units. This is the pH at which the chemical reactions in the cells of the body may optimally occur. Even minimal deviations from normal arterial blood pH will cause marked alterations in enzymatic activity, leading to acceleration or deceleration of various chemical reactions and, ultimately, impairing cell function (McArdle, et al., 2010). As a result, pH plays a key role in the maintenance of homeostasis within the body. Below this normal arterial pH range (a pH of <7.35) is considered a state of increased acidity in the body, known as acidosis, while an extracellular pH of greater than 7.35 is indicative of alkalosis. Humans are unable to sustain a pH of below approximately 6.8 or above approximately 8.0 for more than a few hours. Acidosis and alkalosis may be caused by either respiratory or metabolic mechanisms (Muthayya, 2002).

The regulation of pH and maintenance of acid-base balance is controlled by the following mechanisms:

1. Buffer systems in body fluid
2. Respiratory mechanisms
3. Renal mechanisms

2.5.1. Buffering Systems in the Body

Buffers consist of a mixture of a weak acid with its conjugate base or a weak base with its conjugate acid. Buffer systems in the body react chemically, within a fraction of a second, to changes in the [H^+] and therefore minimise the change in pH (Martini, 2006). Rather than eliminating or introducing H^+ to the body, buffer systems keep the H^+ engaged until the acid-base balance can be restored. The body has a large buffering capacity as illustrated by Pitts (1952) following infusion of dilute hydrochloric acid into dogs. The authors found that infusion of 14mmol/L of H^+ caused a decrease in pH from 7.44 ([H^+]=36nmols/L) to a pH of 7.14 ([H^+]=72nmols/L). As a result, an increase of only 36nmols/L was observed in the blood, suggesting that the other 13,999,964nmols/L infused had been buffered effectively by the body's buffering systems. In humans, approximately 80milliequivalents of hydrogen is ingested or produced by metabolism per day. In contrast, the [H^+] of the body fluids contains only approximately 0.0004 milliequivalents/L (Guyton and Hall, 1996). This large discrepancy between the quantity of acid introduced to the body and the amount that is subsequently visible in the body fluids demonstrates the importance and effectiveness of buffering. As a result of these buffering systems, the body avoids exposure to the huge changes in [H^+] that would otherwise occur. The main buffering systems of the blood and other body fluids include the phosphate buffering system, the protein buffering system and the bicarbonate buffering system (Boning et al., 2007).

2.5.1.1. Phosphate Buffering System

The concentration of phosphate in the blood is quite low and as a result, it contributes little to the buffering capacity of extracellular fluid. The phosphate buffering system is, however, active in intracellular buffering and is by far the most important buffer system in the urine. In order to secrete H^+ in the urine, it must be buffered to maintain the ionic gradients. Phosphates allow large amounts of H^+ to be excreted from the body in urine without causing large alterations to urinary pH. The pH of urine may range from 4.5-8.0 depending on the acid-base status of the extracellular fluid. At the urinary pH of 5.8, there is a 10:1 ratio of acid phosphates (H_2PO_4) to phosphate salts (HPO_4). For each phosphate ion excreted as acid phosphate, one sodium (Na^+) ion is released and reabsorbed from the kidney back into the body and one H^+ ion is excreted in urine (Muthayya, 2002). This exchange also enables the kidneys to reabsorb bicarbonate (HCO_3^-) back into the body with Na^+ as the two bind to form sodium bicarbonate ($NaHCO_3$). As will be discussed in detail later, the kidneys play a key role in the regulation of extracellular fluid [H^+] by altering the rate of excretion of acids or bases depending on the acid-base status.

2.5.1.2. Protein Buffering System

Plasma and cell proteins function as buffers through a similar mechanism to the bicarbonate buffering system below. Due to the permeability of the cell membrane to H^+, HCO_3^- and carbon dioxide (CO_2), there is a relationship between intracellular and extracellular pH, with intracellular pH, which ranges from 6.0-7.4, changing in proportion to the pH in the extracellular fluid. As a result, the intracellular proteins may also contribute to extracellular buffering. However, with the exception of the rapid equilibrium that occurs in red blood cells, the protein buffering system may require several hours to achieve a balance with the extracellular fluid due to the slow movement of H^+ and HCO_3^- through the cell membranes (Guyton and Hall, 1996).

Haemoglobin (Hb) is a globular protein found in the red blood cells which circulate throughout the bloodstream. The cytoplasm of the red blood cells also contain large amounts of the enzyme carbonic anhydrase, which catalyses the reaction converting CO_2 and H_2O to H_2CO_3 to $H^+ + HCO_3^-$ and vice versa. CO_2 is capable of rapidly diffusing across the red blood cell membrane. As a result, red blood cells have a significant influence on the extracellular pH through their absorption of circulating CO_2 from the plasma and conversion of this CO_2 to H_2CO_3. As the H_2CO_3 dissociates into $H^+ + HCO_3^-$, the chloride shift occurs, whereby the intracellular HCO_3^- ions diffuse into the plasma in exchange for extracellular chloride ions. This mass movement exchange does not require ATP to take place. The H^+ are buffered by the Hb molecules. Of the circulating CO_2 in the bloodstream, approximately 7% remains dissolved in plasma whereas the remaining 93%, approximately, diffuses into the red blood cells. Approximately 23% of the CO_2 diffuses into the red blood cells and binds to the exposed amino group (NH_2) of the Hb molecules to form carbaminohaemoglobin in the following reversible reaction (Martini, 2006):

$$CO_2 + HbNH_2 \leftrightarrow HbNHCOOH$$

The remaining 70% of circulating CO_2 is converted to H_2CO_3, resulting in the exchange of HCO_3^- for chloride ions and removal of H^+ by Hb, as mentioned above.

Hb is a potent buffer of H^+ and is responsible for the majority of the capacity of the total protein buffering system. The Hb buffer system is the only intracellular buffer system that can immediately influence the pH of the extracellular fluid. This system helps to prevent significant changes in pH when fluctuations in plasma PCO_2 (partial pressure of arterial dissolved carbon dioxide) are experienced (Martini, 2006). Residues of the amino acid, histidine, with an imidazole sidechain that are contained in Hb allow the protein to buffer effectively through the following reaction:

$$H^+ + Hb \leftrightarrow HHb$$

Deoxygenated Hb is a more effective buffer than oxygenated Hb as it possesses a greater affinity to accept protons. The superior capacity for deoxyhaemoglobin to form carbamino compounds plays a key role in the Haldane effect. The Haldane effect enhances the removal of CO_2 from oxygen-consuming tissues and promotes the dissociation of CO_2 from Hb in the presence of oxygen (Jensen, 2004).

Other proteins in the body have amino and carboxyl groups, both of which may accept or donate H^+, thus contributing to the overall protein buffering system. Proteins are one of the most abundant buffers in the body given their high concentrations particularly within the cells (Guyton and Hall, 1996). Hb and other circulating proteins are highly effective in that they may rapidly minimise the overall pH change by distributing the effects of changes in H^+ throughout the body. These proteins are then capable of transporting H^+ to the lungs to be eliminated in the form of CO_2 and H_2O. However, cellular buffering proteins may be confined to a fixed location within the cell and as result function only as slow buffers, removing H^+ gradually over a period of hours and at increased risk of becoming saturated with acid (Hulikova et al., 2012).

2.5.1.3. Bicarbonate Buffering System

The bicarbonate buffering system is the main buffering system in the extracellular fluid and is responsible for approximately 80% of extracellular buffering. The bicarbonate buffer is considered to be effective due to its high concentration in plasma (~24mmol/L) and also because of the tight regulation of two of its components, CO_2 and HCO_3^-, by the lungs and kidneys respectively (Theis and Barron, 1992).

The bicarbonate buffering system converts hydrochloric acid (a strong acid) to the much weaker carbonic acid (H_2CO_3) by combining with sodium bicarbonate ($NaHCO_3$):

$$HCl + NaHCO_3 \rightarrow NaCl + H_2CO_3 \leftrightarrow H^+ + HCO_3^-$$

Bicarbonate ions (HCO_3^-) interact with H^+ to form carbonic acid (H_2CO_3), which may then be dissociated into CO_2 and water (H_2O). This reaction is catalysed by the enzyme carbonic anhydrase and is reversible:

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$

With acidosis caused by an increase in $[H^+]$, the above reaction is driven to the left and ventilation is stimulated to eliminate the excess CO_2 produced. Conversely, the bicarbonate buffering system may react to a decrease in $[H^+]$ by inhibiting the ventilatory drive in order to retain CO_2 (Greenhaff et al., 1987), thus shifting the reaction to the right. This CO_2 then combines with H_2O to form carbonic acid, which dissociates to H^+ + HCO_3^-, thus increasing acidity and normalising pH (McArdle et al., 2010).

The enzyme catalysing this reaction, carbonic anhydrase, is particularly abundant in the alveolar walls of the lungs where CO_2 is released. This enzyme is also present in the cytoplasm of the red blood cells and also the epithelial cells of the renal tubules, where CO_2 and H_2O combined to form H_2CO_3 (Guyton and Hall, 1996).

The components of the bicarbonate buffering system and therefore, the pH of extracellular fluid, are quantifiable using the Henderson-Hasselbalch equation (Harrison, 1995):

$$pH = pKa + \log ([HCO_3^-] / [H_2CO_3])$$

pKa is the negative logarithm of Ka which is the acid dissociation constant i.e. the ease with which an acid relinquishes its H^+ (Staempfli and Constable, 2003). pKa has a value of 6.099 at a temperature of 37C and a plasma pH of 7.4. This value may be altered depending on the temperature and $[H^+]$ but is generally accepted as 6.1:

$$pH = 6.1 + \log ([HCO_3^-] / [0.03 \times PCO_2])$$

(0.03 is substituted in as the proportionality constant between dissolved CO_2 and PCO_2)

$pH = 6.1 + \log [24mmol/L / (0.03 \times 40mmHg)]$

$pH = 6.1 + \log 20/1$

The maintenance of a normal plasma pH within the body, therefore, relies on the perpetuation of a ratio of 20:1 between $[HCO_3^-]$ and $[CO_2]$ in plasma (Thies and Barron, 1992). From the Henderson-Hasselbalch equation, it is evident that an increased $[HCO_3^-]$ results in a rise in pH, shifting the acid-base balance towards alkalosis (Harrison, 1995). In contrast, an increase in PCO_2 in extracellular fluid causes a decrease in pH, shifting the acid-base balance towards acidosis. $[HCO_3^-]$ and PCO_2 are regulated by the kidneys and lungs respectively (Guyton and Hall, 1996).

2.5.1.4. Isohydric Principle

The isohydric principle states that all buffers in a common solution are in equilibrium with each other in terms of $[H^+]$. Therefore, a change in the $[H^+]$ of extracellular fluid affects all of the buffer systems due to the fact that H^+ are involved in the reactions of each of the systems. The buffer systems in fact buffer each other through the movement of H^+ between them (Guyton and Hall, 1996).

In terms of blood gas analysis, the isohydric principle infers that the concentrations of any one acid-base pair may provide a measurement of the overall acid-base balance within the body. Given that the components of the bicarbonate buffering system (HCO_3^- and PCO_2) are relatively convenient and accessible to quantify, these are the parameters typically utilised to assess overall acid-base balance. Blood gas analysis provides a direct measurement of pH and PCO_2, from which $[HCO_3^-]$ is subsequently calculated using the Henderson-Hasselbalch equation mentioned above.

2.5.1.5. Maintenance of Acid-Base Balance

While the buffering systems provide an effective method of tying up excess H^+, this only serves as a temporary solution to an acid-base imbalance. The body contains a limited

reserve of buffer molecules. These molecules will continue to bind to H^+ until they become saturated, at which point further maintenance of pH may only occur if H^+ can be eliminated from the extracellular fluid or if the buffer molecules are replaced. As a result the maintenance of acid-base balance involves the combination of the buffer systems with respiratory mechanisms and renal mechanisms. These mechanisms are capable of contributing to pH control by secreting or absorbing H^+, manipulating the excretion of acids and bases, and generating replacement buffers (Martini, 2006).

2.5.2 Respiratory Mechanisms

The respiratory system is considered the second line of defence, after the buffering systems, against acid-base balance disturbances. This system reacts to changes in $[H^+]$ within a few minutes to eliminate CO_2 through the lungs. Respiratory compensation refers to an alteration in respiratory rate to minimise a change in extracellular pH. The respiratory system has a direct effect on the bicarbonate buffering system. An increase in the PCO_2 of extracellular fluid causes a decrease in pH, whereas a decrease in PCO_2 increases the pH.

Peripheral chemoreceptors, sensitive to the PCO_2 of circulating blood, are located in the carotid and aortic bodies. The carotid and aortic bodies are stimulated by a decrease in pH or PO_2 or indirectly by an increase in PCO_2. Information from these receptors is monitored by the glossopharyngeal and vagus nerves respectively. Central chemoreceptors are located on the ventrolateral surface of the medulla oblongata. These receptors are sensitive only to the PCO_2 and pH of the cerebrospinal fluid (CSF). Stimulation of these chemoreceptors leads to an alteration in ventilation. Under normal conditions, CO_2 levels are the major factor in the regulation of respiratory activity. The respiratory centres show little response to arterial PO_2 levels until a drop of approximately 40% from 100 to 60mmHg is observed. A major decline in arterial PO_2 to 40mmHg induces only a 50-70% increase in respiratory rate, whereas, the respiratory rate is doubled in response to just a 10% elevation in arterial PCO_2 (Martini, 2006). A rise in

PCO_2 is detected by the chemoreceptors, stimulating an increase in ventilation (rate and depth of respiration). As ventilation increases, more CO_2 is eliminated from the extracellular fluid, which returns PCO_2 levels to normal and also reduces the $[H^+]$ by mass action (Guyton and Hall, 1996). Conversely, a decline in PCO_2 levels within the blood or CSF causes decreased ventilation leading to an increase in extracellular PCO_2 (Martini, 2006).

Extracellular PCO_2 may be influenced by variations in either the respiratory ventilation or the rate of intracellular metabolic CO_2 production. CO_2 is constantly being formed within the body through intracellular metabolism. Once formed, CO_2 diffuses out from the cells into the blood where it is transported to the lungs, diffuses into the alveoli and is expired. Extracellular fluid normally contains approximately 1.2mol/L of dissolved CO_2, equating to a PCO_2 of 40mmHg. An increase or decrease in the intracellular metabolic formation of CO_2 corresponds to proportionate increase or decrease in extracellular PCO_2. In addition, an increase in the rate of ventilation allows CO_2 to be eliminated from the lungs, thus decreasing the extracellular PCO_2 (Guyton and Hall, 1996).

An increase in $[CO_2]$ is associated with corresponding increases in $[H_2CO_3]$ and $[H^+]$, resulting in a decline in extracellular pH. A two-fold increase in the normal rate of alveolar ventilation induces an increase of approximately 0.23 in extracellular pH, which would cause an elevation of pH from 7.4 to 7.63. Conversely, an alveolar ventilation rate representative of one-quarter of normal reduces the pH by 0.45, which would result in a reduction in pH from 7.4 to 6.95. Given the large variations that may occur in the rate of alveolar ventilation, the respiratory system plays a major role in pH control and in fact operates as a negative feedback regulator of $[H^+]$ - an increase in $[H^+]$ stimulates respiration and an increase in alveolar ventilation in turn decreases $[H^+]$ (Guyton and Hall, 1996).

2.5.2.1 Oxyhaemoglobin dissociation curve

Under normal conditions, haemoglobin is responsible for transporting approximately 97% of the oxygen (O_2) travelling from the lungs to the tissue. The remaining 3% is dissolved in the plasma. Each haemoglobin molecule possesses the capacity to carry four O_2 molecules at any one time. O_2 saturation refers to the ratio between the amount of O_2 bound to haemoglobin and its oxygen-carrying capacity (McArdle et al., 2010). The affinity of haemoglobin for O_2 is largely dependent on the partial pressure of O_2 (PO_2) to which the haemoglobin is exposed (Guyton and Hall, 1996). For example, in the pulmonary capillaries where PO_2 is high, O_2 binds easily to haemoglobin and may be transported to the tissues. Contrastingly, in the tissue capillaries where PO_2 is low, O_2 is released from the haemoglobin. In addition, deoxygenated haemoglobin has an increased affinity for CO_2 to bind and be transported from the working tissue to the lungs to be expired (Haldane effect).

The oxyhaemoglobin dissociation curve, which represents the affinity of haemoglobin to bind to O_2 molecules, is a sigmoidal curve illustrating the progressive increase in the percentage of haemoglobin bound with O_2 as the PO_2 increases. A normal arterial PO_2 of approximately 100mmHg equates to approximately 97% O_2 saturation. The PO_2 of normal venous blood is approximately 40mmHg, corresponding to a saturation level of approximately 75% (Guyton and Hall, 1996). The oxyhaemoglobin dissociation curve is affected by several factors such as pH, temperature, PO_2, PCO_2 and 2,3-disphosphoglycerate (2,3-DPG), which is a metabolite of anaerobic glycolysis (Dickson, 1995). During exercise, the curve may be shifted to the right (low affinity for O_2) by a decrease in pH, increase in PCO_2, increase in temperature, decrease in PO_2 and an increase in 2,3-DPG (Riggs et al., 1973). The effect of the rightward curve shift during exercise encourages O_2 to dissociation from haemoglobin at the tissue. Therefore, this effect promotes O_2 delivery to the working tissue during exercise. In contrast, the curve is shifted to the left under the opposite circumstances, whereby H^+ and CO_2 are released from haemoglobin, the affinity of haemoglobin for O_2 is increased and a decreased concentration of O_2 is required to saturate haemoglobin with oxygen (Bohr effect).

2.5.2.2 Responses of pH, partial pressures and the buffering systems to exercise:

During maximal exericse, O_2 diffusing capacity (i.e. the rate at which O_2 can diffuse from the alveoli to the blood) may be increased by up to three times from resting levels. This is reported to be due to the increased blood flow which accompanies exercise. Increased blood flow encourages increased perfusion and therefore, a greater surface area over which diffusion may occur (Guyton and Hall, 1996). Changes in arterial blood gases as a consequence of exercise are normally minimal. There may be a slight rise in arterial PO_2 due to increased ventilation. However, PO_2 may eventually decrease at higher work loads. During short duration, high intensity exercise involving dominance of anaerobic glycolysis, accumulating H^+ results in an increase in PCO_2 (McArdle et al., 2010). Respiratory compensation (as mentioned in Section 2.5.2) acts to stimulate ventilation and decrease extracellular PCO_2, thereby resisting a change in pH (Martini, 2006). During moderate exercise, blood pH may remain constant for long durations. However, as exercise intensity increases there is an increased reliance on anaerobic glycolysis. The resultant accumulation of H^+ leads to a decrease in pH (Cheetham et al., 1986).

2.5.3 Renal Mechanisms

The kidneys play a major role in the regulation of acid-base balance as they function to eliminate excess acid or base from the body via the urinary system. The kidneys are relatively slow to respond to changes in $[H^+]$ compared to the buffering systems and respiratory system. However, the renal system is an important longer-term contributor to acid-base regulation, functioning to maintain the body's buffer reserve (McArdle et al., 2010). The kidneys regulate extracellular $[H^+]$ through secretion of H^+, reabsorption of filtered HCO_3^- and generation of new HCO_3^-. Renal compensation refers to an alteration in the rates of secretion or absorption of H^+ and HCO_3^- by the kidneys in response to changes in extracellular pH. In order to maintain acid-base balance the body must generate and excrete an equal amount of H^+ (Martini, 2006). In alkalosis (decreased extracellular $[H^+]$), the excess HCO_3^- ions cannot be reabsorbed and, therefore, the

kidneys increase the excretion of HCO_3^-. The removal of HCO_3^- allows the extracellular $[H^+]$ to return toward homeostasis. In acidosis (increased extracellular $[H^+]$), instead of excreting HCO_3^- in the urine, the kidneys reabsorb all the filtered HCO_3^-, generate new HCO_3^- and excrete the excess H^+ in the urine. The filtered and newly generated HCO_3^- is then returned to the extracellular fluid to function as an available buffer of H^+, thus reducing the extracellular $[H^+]$ back towards homeostasis (Guyton and Hall, 1996).

2.5.3.1. Secretion of H^+ and Reabsorption of HCO_3^-

Secretion of H^+ and reabsorption of HCO_3^- occurs in all segments of the renal tubules with the exception of the descending and ascending thin limbs of the loop of Henle. The majority (approximately 85%) of the HCO_3^- reabsorption and H^+ secretion takes place in the proximal convoluted tubule. Another 10% of the filtered HCO_3^- is reabsorbed in the thick ascending loop of Henle, with the remainder of the absorption occurring in the distal convoluted tubule and collecting duct (Guyton and Hall, 1996).

2.5.3.2. Secretion of H^+ in the Early Tubular Segments

The mechanism of HCO_3^- reabsorption also involves secretion of H^+ from the different renal tubular segments. In the early tubular segments, H^+ ions are secreted by secondary active transport. The epithelial cells of the proximal convoluted tubule, the thick ascending loop of Henle and the distal convoluted tubule all secrete H^+ into the tubular fluid by sodium-hydrogen (Na^+/ H^+) counter-transport at the membrane of the lumen of the tubule (Guyton and Hall, 1996).

The secretion of H^+ initiates the process of HCO_3^- reabsorption. HCO_3^- ions that are filtered by the glomerulus cannot be directly reabsorbed as they cannot cross the membrane from the lumen into the tubular cells in their existing form. Instead HCO_3^- must first bind with H^+ to form H_2CO_3, which eventually dissociates into H_2O and CO_2, which can pass readily into the tubular cells from the lumen. CO_2 either diffuses from the renal interstitial fluid into the tubular cells or is produced by metabolism in the tubular

epithelial cells. This CO_2 then binds with H_2O to form H_2CO_3, which subsequently dissociates into HCO_3^- and H^+. The H^+ ions combine with a carrier protein on the cell membrane and are secreted into the lumen of the tubule in exchange for Na^+ ions that also bind to a carrier protein and move into the tubular cells. The Na^+ travels into the tubular cell from the lumen of the tubule along a concentration gradient established by the sodium potassium (Na^+/K^+) ATPase pump in the basolateral membrane (membrane between the tubular cell and the renal interstitial fluid).

This concentration gradient responsible for transporting Na^+ into the cell then provides the energy required to transfer H^+ in the opposite direction from the tubular cell to the lumen of the tubule. The bicarbonate ion, formed in the cell from the dissociation of H_2CO_3 then crosses the basolateral membrane from the cell into the renal interstitial fluid and the peritubular capillary blood. In summary, the sum of the transport of ions between the tubular lumen, tubular cells and renal interstitial fluid result in a HCO_3^- ion entering the blood in exchange for every H^+ ion secreted into the tubular lumen (Guyton and Hall, 1996).

2.5.3.3. Secretion of H^+ in the Late Distal Tubule and Collecting Duct

In the later sections of the renal tubules, including the late distal convoluted tubule and the collecting duct, H^+ is secreted by primary active transport from the epithelium of the tubular cells into the lumen. For every H^+ that is secreted, a HCO_3^- ion is reabsorbed and a chloride (Cl^-) ion is passively secreted along with the H^+. This process occurs in the intercalated cells of the late distal convoluted tubule and collecting duct. Similar to the proximal tubule H^+ secretion previously discussed, dissolved CO_2 in the cell binds with H_2O to form H_2CO_3. The H_2CO_3 then dissociates into HCO_3^- ions, which are reabsorbed into the blood and H^+ ions, which are secreted into the lumen by a specific protein known as a hydrogen-transporting ATPase. The energy necessary for the transport of H^+ across the luminal membrane from the tubular cells into the tubular lumen is derived from the breakdown of ATP to adenosine diphosphate (ADP). This active energy-utilising H^+ pump operating in the distal renal tubules, in contrast to the counter-transport mechanism

involved in the proximal tubules is the main difference between the secretion of H^+ in respective segments of the renal tubules (Guyton and Hall, 1996).

2.5.3.4. Renal Buffers

Buffers in the tubular fluid play a key role in the elimination of H^+ within a normal volume of urine. Minimal urine pH is approximately 4.5, equating to a maximum excretion of approximately just 0.03milliequivalents/L of free H^+. As a result, in order to excrete the 80milliequivalents/day of non-volatile acid produced by metabolism with H^+ remaining in its free form, 2667 litres of urine would be required to be excreted. Large amounts of H^+ (up to 500milliequivalents/day) are capable of being excreted in the urine through the combination of H^+ with buffers in the tubular fluid (Guyton and Hall, 1996). These buffers maintain a pH of significant elevation to allow continued secretion of H^+, therefore, preserving acid-base balance. The three major buffer systems involved in stabilising the tubular fluid pH include the bicarbonate buffer system, the phosphate buffer system and the ammonia buffer system (Martini, 2006).

2.5.3.5. Phosphate Buffer System

As previously discussed, although the phosphate system plays only a minor role in extracellular buffering, it is, however, an important tubular buffer. HPO_4^{2-} and $H_2PO_4^-$, which comprise the phosphate buffer system, are both found in high concentrations in the tubular fluid. This is partially due to their poor reabsorption and also the reabsorption of H_2O from the tubular fluid. In addition, the pKa of the phosphate buffer system is approximately 6.8, which is close to the normal pH of urine. As a result, the phosphate buffer system is capable of operating close to its most effective pH range within the tubular fluid. For the most part, excess H^+ in the tubular fluid is buffered by HCO_3^-. However, once all of the HCO_3^- has either been reabsorbed or saturated with H^+, the remaining excess H^+ may bind to other tubular buffers, including HPO_4^{2-}. HPO_4^{2-} and H^+ amalgamate with Na^+ in the tubular fluid to form NaH_2PO_4, which can then be excreted as a salt. The phosphate buffer system allows a HCO_3^- ion to be spared from excretion. As a

result, the HCO_3^- ion generated in the tubular cell may be transported into the peritubular blood as a new additional HCO_3^- and not merely as a replacement for the HCO_3^- excreted in the buffering process. However, given that only approximately 30-40milliequivalents/day of phosphates are available for buffering H^+, the ammonia buffer system regulates the majority of the excess H^+ buffering (Guyton and Hall, 1996).

2.5.3.6. Ammonia Buffer System

The ammonia buffer system is composed of ammonia (NH_3) and ammonium (NH_4^+). Within the proximal convoluted tubule, thick ascending loop of Henle and distal convoluted tubule, this system is underpinned by the metabolism of glutamine. The breakdown of glutamine produces two NH_4^+ and two HCO_3^- ions. The NH_4^+ is actively secreted, in exchange for a Na^+, by means of a counter-transport Na^+/ NH_4^+ pump. The Na^+ is then reabsorbed along with the HCO_3^- across the basolateral membrane and into the interstitial fluid and then the peritubular blood. Similar to the phosphate buffer system, the HCO_3^- generated through this process is considered new HCO_3^-. The ammonia buffer system excretes H^+ through an alternative mechanism in the collecting duct. In this segment of the renal tubules, NH_3 diffuses easily into the tubular lumen where it combines with H^+, to form NH_4^+. The tubular cells of the collecting duct are highly permeable to NH_3 but conversely much less permeable to NH_4^+. As a result, the NH_4^+, which is composed of the diffused NH_3 along with the excess H^+, is confined to the lumen and excreted in the urine. As with the ammonia buffering in the more proximal tubules, the collecting ducts also facilitate the generation of new HCO_3^-. Each NH_4^+ excreted, allows a new HCO_3^- to be produced and transported to the blood. Under normal conditions, the ammonia buffer system is responsible for approximately 50% of the acid excreted and 50% of the new HCO_3^- produced (Guyton and Hall, 1996).

2.6. Ergogenic Aids

Ergogenic aids have been used for centuries in a superstitious, ritualistic fashion prior to physical exertion with athletes crediting their past performances to particular dietary habits. Accounts from ancient times depict athletes and soldiers consuming specific animal parts in an attempt to assimilate the physical attributes such as speed and strength associated with that animal (Applegate and Grivetti, 1997). In the early 20th Century a greater understanding of the biochemistry and physiology of muscular work, fuel use during exercise and the specific roles of carbohydrate, fat and protein, gave a degree of rationality to the use of ergogenic aids, such as alkaline salts, caffeine, carbohydrate and protein (Brown et al., 2006; McArdle et al., 2010). However, the scientific justification of the use of nutritional supplements is generally in response to athlete experimentation with various substances, volume, form and timing of administration with the aim of performance enhancement. Prohibited forms of performance enhancement substances including anabolic steroids and blood doping have also been developed for athletes, however, ethically and in the interests of health and sportsmanship, these practices should not be condoned.

Along with genetic factors and training, many athletes view ergogenic aids as essential vehicles to enhance performance (Applegate, 1999). Nutritional supplements are a multi-billion euro industry worldwide (Burke et al., 2004), form an integral part of the sports nutrition strategies of many athletes, from recreational to elite. One such supplement is sodium bicarbonate. Much of this athlete support of various supplements is based on manufacturers' assurances of positive outcomes such as weight loss, muscle gain, performance enhancement and not on definitive scientific evidence. The absence of regulation in the supplement industry facilitates product advertisements with unsupported claims about health and performance benefits. Many of these products have been found to show poor compliance with regard to labelling laws, presence of contaminants and undeclared ingredients (Burke, 2003).

2.7. Sodium Bicarbonate and Fatigue

Sodium bicarbonate (SB) is a naturally occurring buffer in the body which has resulted in reported performance enhancements when used as an ergogenic aid prior to high intensity exercise lasting between 1 and 7 minutes (McNaughton, 1992). The buffer systems of the body act to attenuate the accumulation of H^+ and therefore aid in the regulation of pH. The production and removal rates of H^+ , which both contribute to H^+ accumulation, are influenced by the muscle's oxidative capacity, muscle fibre type, transport proteins and the work-to-rest ratio involved during repeated exercise bouts (Yoshida et al., 1993). SB supplementation has been investigated as a potential aid to increase the body's buffering capacity (Hollidge-Horvat et al., 2000).

Research into the area of SB supplementation originated from findings from animal studies, demonstrating an increased resistance to fatigue in isolated muscle preparations of dogs and frogs and a change in the rate of lactate efflux from the muscle (Hirche et al., 1975; Mainwood and Worsley-Brown, 1975). By inducing a state of pre-exercise alkalosis within the body, the decrease in pH associated with exercise may be delayed. Therefore, a greater capacity for contraction of working muscular tissue may be facilitated, by means of enhanced muscle glycolytic ATP production (Sutton et al., 1981; Bishop et al., 2004). As a result, the ability to limit the changes in pH during high intensity exercise may delay fatigue and potentially enhance performance (McNaughton, 1992).

Research into the acid base balance and its effect on the capacity for work has been investigated since the 1930s (Dennig et al., 1931). In this and similar studies at the time, the investigators identified that the administration of acid salts caused runners to be more acidic and less able to efficiently utilise oxygen. According to Spriet et al., (1986), acidosis decreases the muscle's ability to generate isometric tension and diminishes aerobic and anaerobic metabolism. This led to the conclusion that induced alkalosis may potentially have the opposite effect.

2.8. Sodium Bicarbonate and Sports Performance

In terms of single bout, high intensity, short duration exercise lasting less than 120 seconds, conflicting results have been reported regarding SB supplementation. McNaughton (1992) observed significant performance enhancements with acute SB ingestion prior to a 60 second maximal cycle ergometer test. McNaughton (1992) found that SB supplementation with 0.3g/kg body mass (BM) resulted in significantly higher peak power (1295 +/- 72.8 W) and work completed (41.9 kJ/min) when compared to a placebo (PLA) trial. Similarly, in a study by Goldfinch et al. (1988), it was found that ingestion of SB resulted in significantly faster 400 m running times (1.52 seconds) when compared to the control. This evidence suggests that SB ingestion facilitates removal of excess hydrogen ions from the working muscle, decreasing intracellular pH and delaying the onset of fatigue.

Although the majority of the research involving SB supplementation demonstrates perturbations in the acid-base balance inducing a state of alkalosis, this condition does not always translate into an enhanced performance. Horswill et al., (1988) found no significant improvement in the total work performed in a 2 minute cycling sprint with SB supplementation. No improvements were recorded, either for work performed or power output, following 90 seconds of maximal cycling exercise by untrained males (Marx et al., 2002). The lack of significant findings in certain studies may also be as a result of factors such as sample size, subject characteristics, SB doses and insufficient durations and/or intensities of exercise.

Similar to the findings relating to single bout performances lasting less than 2 minutes, research examining repeated bout protocols is also inconclusive. According to the findings of a study by Lavender and Bird (1989), SB supplementation resulted in a significant improvement in performance of 10 x 10 second cycle sprints with 50 seconds recovery between each sprint. In contrast, Gaitanos et al., (1991) found no improvement in the performance of 10 x 6 second running sprints with 30 seconds recovery between each sprint.

To date the majority of studies investigating the effects of SB supplementation on exercise performance have involved short repeated bouts of high intensity exercise lasting between 60 and 360s. In contrast, numerous studies have investigated the effect of SB supplementation on prolonged endurance exercise. In a study investigating the effect of 0.2g/kg BM of SB on time to exhaustion at a running velocity corresponding to a blood lactate concentration of 4 mmol/L, George and MacLaren (1988) found a 17% longer time to exhaustion with SB supplementation when compared to the placebo trial. Similarly, McNaughton et al., (1999) reported that, following ingestion of 0.3g/kg BM of SB, 10 well-trained cyclists improved their total work by 13 % in a 1 hour maximal cycle ergometer test In contrast to these findings, Stephens et al., (2002) found no difference between SB and control trials in performance of 30 minutes of continuous cycling at 77% VO_2max followed by a performance ride (time to complete 469 +/-21 kJ work).

Analysis of SB supplementation in relation to its effect on prolonged intermittent exercise is also represented in the research, with only minimal emphasis on team field sports. One such study involving a team field sport was undertaken by Bishop and Claudis (2005), in which subjects ingested 0.2g/kg BM of SB at 90 and 20 minutes prior to completion of 2 x 36 minute "halves" of intermittent field hockey-specific activity. Each half consisted of 2 minute blocks involving a 4 second sprint, 100 seconds at 35% VO_2 max, 20 seconds passive rest with an additional two repeated sprint bouts of 5 x 2 seconds separated by 35 seconds at 35 % VO_2 max. No significant differences in the total work performed over the 72 minutes were observed between the SB and PLA trials. However, an increase in work performed in 7 of 18 second half sprints was reported.

In general, studies that have found an increase in exercise performance following SB supplementation have been those that have utilised a continuous or intermittent exercise protocol that induced a large disturbance in the acid-base balance (McNaughton et al., 2008). During continuous dynamic exercises, performance enhancements tended to be observed in studies whereby the test protocol exhausted the subjects in 1-10 minutes (Requena et al., 2005). However, during investigation of the effects of alkaline agents on

shorter duration (30-40 seconds) high intensity exercises, conflicting results have been reported (Ibanez et al., 1995 and Linderman et al., 1992).

The exact reasons for these conflicting results are unclear but may in part be due to inadequate exploitation of the maximum buffering capacity through insufficient duration or intensity of exercise, thereby limiting the ergogenic benefits (McNaughton, 1992). In addition, there appears to be a highly individual response to SB ingestion, which may be partially accounted for by the gastrointestinal side effects associated with SB ingestion. Another explanation for the lack of performance enhancement with SB supplementation found in some previous studies may be the use of untrained subjects. Elite sprint trained athletes tend to produce a greater force capacity leading to a greater systemic acidosis when compared with untrained or endurance athletes (Johansen et al., 2003). Therefore given the lower intramuscular pH and the high amount of hydrogen ions produced by sprinters in competition, it is possible that the effects of SB on trained elite sprinters may be more significant than those reported for untrained individuals (Zabala et al., 2008). Any potential ergogenic effects that may be produced by pre-exercise metabolic acidosis are dependent upon the physiological demands of the activity being sufficient enough to result in a performance-inhibiting level of metabolic acidosis (McNaughton et al., 2008).

2.9. Sodium Bicarbonate Supplementation Strategies

2.9.1. Timing of Ingestion

There is a large degree of variability across studies in the time elapsed from ingestion to the beginning of exercise performance. Although the effects of SB supplementation on short-term, high intensity exercise performance have been well-reported, very few studies have investigated the optimal loading time prior to exercise. Absorption times in different studies vary from 30 minutes (Edge et al., 2006) to 180 minutes (Sutton et al., 1981). The majority of the studies involving similar SB ingestion protocols with the first or second SB ingestion time at 60 minutes prior to exercise show either decreases or no change in

performance times or power output across a range of exercise types including exercise to exhaustion and repeated sprint tests (Siegler et al., 2008; Robergs et al., 2005; Matsuura et al., 2007; Mero et al., 2004; Aschenbach, et al., 2000). Price et al. (2003) provides an exception to this evidence by discovering an increased mean relative power output during maximal sprint efforts following ingestion of 0.3g/kg BM of SB at 60 minutes prior to exercise. From reviewing the literature, it would appear that SB ingestion commencing 60 minutes or less prior to exercise may inhibit performance due to gastrointestinal side effects arising from such a brief ingestion period.

Various other studies have investigated SB loading at both 120 minutes (Artioli et al., 2007) and 90 minutes prior to exercise (Bishop et al., 2004) with more positive results reported in terms of increased power output and work performed. Analysis of the optimum absorption time by Potteiger et al., (1996) found that, for an intake of 0.3g/kg BM of SB, 120 minutes was required to reach peak pH and an average of 100-120 minutes would allow maximum blood bicarbonate concentration to be attained. However, Siegler et al. (2012) observed that pre-exercise ingestion time did not influence the performance of 10 x 10 second treadmill sprints with 50 second active recovery in between each sprint. Subjects ingested 0.3g/kg BM of SB at 60, 120 or 180 minutes prior to exercise. No significant differences between the ingestion times were detected in either pre-exercise blood buffering capacity or performance across the ten sprints.

2.9.2. Dosage

A study by McNaughton (1992) investigated various doses of SB and their potential ergogenic efficacy on anaerobic performance times. Five doses of SB (0.1, 0.2, 0.3, 0.4 and 0.5g/kg BM), along with a placebo and control, were compared in terms of their effect on total work and peak power in an anaerobic 60s cycle ergometer test. In this study, although all doses with the exception of 0.1g/kg BM of SB resulted in more work completed than the control, the most work was completed in the 0.3g/kg BM of SB trial with this dosage also exhibiting the highest level of peak power. Analysis of blood pH revealed that a state of alkalosis was achieved after ingestion of all but the 0.1g/kg BM

dose. However, in a study by McKenzie et al. (1988), no significant differences in performance were found between a sodium bicarbonate dosage of 0.15g/kg BM and 0.3g/kg BM, suggesting that maximum buffering capacity had been achieved at the lower dose and an increased level of alkalosis had no additional benefits.

Based on the current literature it is generally accepted that the optimal SB dosage to potentially induce an improvement in performance and also to avoid gastrointestinal issues is 0.3g/kg BM (McNaughton, 1992). A SB intake of 0.3g/g BM has generally been shown to produce an increase of 4-5mmol/L of bicarbonate concentration and 0.03-0.04 pH units in venous plasma 2-3 hours after ingestion (Siegler et al., 2012).

2.9.3. Acute versus Chronic Loading

Few studies have examined the effects of chronic SB loading in the days and weeks prior to performance, with only three papers currently published outlining the effects of acute versus chronic SB supplementation. Douroudos et al., (2006) investigated a five day SB loading period with both 0.3 and 0.5g/kgBM/day of SB with no ingestion on the day of the 30 second Wingate trial. Significant improvements in relative mean power were found for both dosages when compared to the placebo group, with the higher intake of 0.5g.kgBM/day also shown to be more effective than the moderate intake of 0.3g/kgBM/day. A study by McNaughton (1999) also observed that 0.5g/kgBM/day of SB elicited significantly greater work completed and greater peak power in a 60 second cycle ergometer sprint when compared to a control and pre-ingestion group. However, no PLA group was involved in this trial. Similarly, a 7-day loading phase of alkalizing tablets produced significantly greater upper body power measures in Nordic skiers when compared to a placebo group (Heil et al., 2012).

In 2001, McNaughton and Thompson compared the effects of acute and chronic SB ingestion. The authors found that chronic ingestion of 0.5g/kg BM of SB over a period of six days significantly improved work output for at least two days after cessation of ingestion when compared with acute ingestion of 0.5g/kg BM 90 minutes prior to

performance of a 90 second maximal cycling test. A study by Edge and colleagues (2006) investigated the use of SB supplementation over an eight week training period in which subjects supplemented with SB or a placebo prior to each cycle ergometer based interval training session. Subjects performed six to twelve 2-minute cycle intervals at 140-170% of their lactate threshold three days per week over the course of the eight week period. The results of this study reported that both groups demonstrated improvements in muscle buffering capacity following the eight week period, suggesting that training intensity may be the key component for improvements in muscle buffering capacity. However, the group ingesting SB prior to each training session was found to display larger improvements in lactate threshold and endurance performance. The authors infer that this may be as a result of reduced metabolic acidosis during training and, therefore, the development of a greater muscle oxidative capacity.

In contrast to the aforementioned studies regarding chronic SB supplementation, Carr et al., (2012) found no improvements in 2000m rowing performance with either acute or chronic SB supplementation. Similarly, a study by Joyce et al., (2011) involving elite male swimmers observed no improvements in 200m swim performance with either acute or chronic SB supplementation with respect to the placebo trial. However, Lindh et al., (2008) used an identical acute loading protocol to detect a significant improvement in 200m swim performance in highly trained males.

Chronic ingestion of SB in the days prior to competition may facilitate improved performance while alleviating the potential side affects associated with SB ingestion. The use of chronic SB supplementation may also be warranted as part of the training routine to augment the training response for endurance performance (Edge et al., 2006). Therefore it may be beneficial to examine the potential effects of short or long-term SB loading to optimise performance and offset any possible gastro-intestinal issues during performance. However the health risks associated with this form of loading require further investigation.

2.10. Exercise and the recovery process

Recovery is a term referring to the adaptations to physiological stress that occur following overload such as training or competition. When recovery is utilized effectively, a positive response to exercise is initiated leading to subsequent adaptation to the stressors involved (Calder, 1990). Given the volume of competitive games in a rugby union season and the need to perform at a consistently high level, recovery plays a key role in determining subsequent athletic performance. Athletes aim to push themselves harder in training in order to perform to their potential in competition. However, by increasing intensity, volume and duration of training without allowing for adequate recovery, the risk of excessive fatigue and impaired performance is increased (Hooper and Makinnon, 1995). Insufficient recovery will invariably result in maladaptation and may lead to detrimental conditions for the athlete, including overreaching, overtraining or burnout (Calder, 1990).

Physiological adaptations to exercise come about by progressively overloading the body systems, mainly the cardiovascular, respiratory, neuromuscular and the endocrine systems. Adaptation to training is accelerated when fatigue associated with the overload is minimized and the body is returned to homeostasis as soon as possible. Fatigue can be muscular, neural, psychological and metabolic depending on the intensity, duration and volume of the training stimulus (Kellman, 2002). This accelerated adaptation to training is illustrated in Figure 2.1. When the appropriate recovery for the training stimulus is undertaken, both the response to the exercise stress and recovery are accelerated. Therefore the body is returned to homeostasis more rapidly and the body can perform to maximal capacity in subsequent exercise sessions.

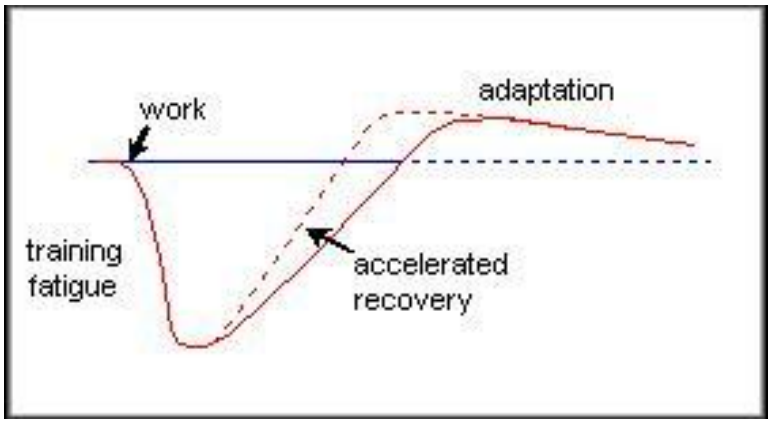


Figure 2.1: Accelerated recovery

Stress occurs when demands exceed the body's capacity to deal with those demands. Physiological stress is necessary to stimulate the body's processes to progress and become more efficient. Selye (1936) suggested that under normal conditions, the body can utilise its homeostatic mechanisms to dissipate stressors. However, excessive amounts of stress without appropriate recovery may lead to symptoms associated with overreaching, overtraining and burnout (Murphree, 2004).

Selye (1936) conceptualised the physiological processes that occur when the body is exposed to stress with the General Adaptation Syndrome (GAS). A stressor, which can range from food deprivation to excessive muscular exercise, if of sufficient intensity and duration, can produce various physiological changes as the body attempts to adapt. Selye developed the GAS following a study on rats and their responses to noxious agents such as excessive muscular exercise. Selye observed symptoms independent of the type of stressor applied and rather in response to the damage incurred. The rats developed a triad of symptoms resulting from exposure to the stressors including enlargement of the adrenal cortex, atrophy of the thymus, spleen and lymph nodes and deep bleeding ulcers in the lining of the stomach and duodenum.

The symptoms contributed to an adaptation syndrome, which was observed to progress in three main stages. The initial stage, known as the Alarm Stage, occurs when the body has first been exposed to the stress, thereby activating the autonomic nervous system and

preparing the body for a “fight or flight” response. The hypothalamus stimulates the pituitary gland to release adrenocorticotropic hormone (ACTH) into the blood stream. This signal prompts the adrenal cortex above the kidneys to produce corticoid hormones. These corticoids are then distributed throughout the body and used to defend against a stressing agent at various stages. This internal stress-processing mechanism is known as the hypothalamus-pituitary-adrenal system. Provided the organism survives this first stage, a resistance is built in the second stage of the adaptation syndrome - the stage of resistance. However, if the stressor is applied for a continued period of time, the body eventually enters the third stage - the stage of exhaustion (Sacco, 2005). The syndrome seems to represent a generalized attempt by the organism to adapt itself and so is termed the “General Adaptation Syndrome” (Selye, 1946).

Cannon (1932) proposed that organisms are open systems which can be influenced by their environment. These organisms require some autonomic adjustments to regulate their internal environment to maintain a stable, constant condition or equilibrium. Homeostasis refers to the dynamic equilibrium adjustments controlled by interrelated self-regulatory mechanisms. These mechanisms may include negative feedback, which attempts to reverse any change in the internal environmental, such as core temperature, to maintain homeostasis (Sukkar, et al, 1994).

It has been documented that performance enhancement comes about by intentionally repeating stimuli to induce recovery-adaptation while avoiding overreaching-overtraining (Stone and Stone, 2003). Overtraining can be described as a chronic state of insufficient recovery. Overtraining occurs when the volume and intensity of an athlete's training exceeds their recovery capacity. Overtraining can result in persistent muscle soreness, fatigue, elevated resting heart rate, depression, loss of motivation and appetite, increased incidence of infection or injury and/or weight loss. Short term overtraining is described as overreaching and is usually exhibited after several days of intense training and results in muscular fatigue. According to Shephard (2005), possible mechanisms for overtraining may include muscles being subjected to micro-trauma faster than the body can heal itself, depletion of amino acids faster than they can be replenished, elevated levels of cortisol

(the "stress" hormone) for long periods and dominance throughout the body of catabolism over anabolism.

Smith (2000) associates overtraining syndrome (OTS) with excessive training without appropriate recovery, which yields a decrement in performance. In a study concerning the role of cytokines in OTS, Smith proposes that high volume or intensity training, with insufficient recovery, will result in musculoskeletal trauma. Circulating monocytes are then activated by injury-related cytokines, which are signalling compounds that communicate between cells, to produce proinflammatories (IL-1beta, IL-6, TNF-alpha) resulting in systemic inflammation. Elevated circulating cytokines communicate with the central nervous system (CNS) and a set of physiological changes ensues. The liver adjusts to up-regulate gluconeogenesis, synthesize acute phase proteins and increase catabolism. Immune function is also impaired in the body's response to stress without adequate recovery. A review by Dantzer and Kelley (2007) reported that the release of proinflammatory cytokines following injury, act in the brain to induce symptoms consistent with overtraining syndrome including loss of appetite, fatigue, sleepiness, aching joints and withdrawal from social activities. Overtraining syndrome is considered as the third stage (stage of exhaustion) of Selye's general adaptation syndrome and therefore, the objective is recovery rather than adaptation.

The evidence presented thus far emphasises the importance of the recovery process in physically demanding team field sports, such as rugby union. Recovery time periods between matches may vary from days, in 15-a-side rugby, to minutes in rugby sevens. However, active recovery periods within games must also be exploited in order to optimise performance. Limited research currently exists regarding the potential of SB ingestion to impact on the recovery process. This will be discussed in more detail in Section 2.12.

2.11. Lactate Removal During Recovery

Lactic acid, which dissociates into lactate and H^+, is a compound converted from pyruvic acid, a product of anaerobic glycolysis (the production of energy from glycogen when limited oxygen is present). Lactic acid formation occurs at all times, even at rest but it is produced in such low levels that it is readily removed from the body (Martini, 2006). However, as exercise intensifies, more H^+ are present (as a result of glycolysis) than can be oxidized. To maintain energy metabolism, the H^+ must be accepted by a chemical other than oxygen, such as pyruvic acid. Pyruvic acid temporarily accepts a pair of hydrogens and this reduction of pyruvic acid forms lactic acid (McArdle et al., 2010). Lactate is a derivative of lactic acid in salt form, and is a term often used interchangeably with lactic acid. Lactic acid, however, can release a H^+ and the remaining compound, which is negatively charged, joins with a positively charged ion, such as N^+ or K^+, to form a salt known as lactate (Kravitz, 2005).

With high intensity, anaerobic exercise, there is a greater flow of excess hydrogen ions to pyruvic acid, less oxygen is available, and therefore, lactic acid increases rapidly. Lactic acid production proceeds to increase linearly with respect to exercise and eventually, a point is reached where clearance of lactic acid is outweighed by the rate of lactic acid production. As a result an overall increase in lactic acid and free H^+ is observed. This rise in H^+ exceeds the body's buffering capability resulting in decreased pH levels and therefore, greater acidity in the circulating blood. The accumulation of H^+ stimulates pain receptors giving rise to the burning sensation experienced by athletes at maximal capacity. During recovery, when more oxygen is present, the excess H^+ used to form lactic acid can be oxidized and the pyruvic acid molecule is reformed. Lactate dehydrogenase is the enzyme which facilitates this reaction (McArdle et al. 2010).
According to Baldari et al., (2004), lactate removal was significantly more efficient with 30 minutes of active recovery than with 30 minutes passive recovery. However, it would appear that, while moderate intensity active recovery results in an increased rate of lactate removal, high intensity active recovery results in less efficient lactate removal. In a study by Belcastro and Bonen (1975), after a 6 minute cycle ergometer test at 89% VO_{2max} it

was found that lactate removal rates were faster at self-regulated recoveries than during recovery at rest and exercise at 61.8% and 80.8% VO_{2max}. Lactate removal was not significantly different for self-regulated recovery and recovery at 29.7 and 45.3% $VO_{2max.}$ Additionally, in a study by Kuotedakis and Sharp (1985) concerning lactate removal during recovery after strenuous exercise, it was observed that 40% of the maximal rowing speed was appropriate for lactate removal.

For short bursts of intense activity, the body relies mainly on anaerobic rather than aerobic metabolism. This allows the body to take advantage of the resources that are located within the muscle (e.g. adenosine triphosphate (ATP), creatine phosphate and muscle glycogen) and also avoids the time lag associated with the delivery of oxygen (Bennet and Licht, 1972). The use of the anaerobic system for longer durations of exercise appears to be limited by the accumulation by-products such as H^+. Lactic acid accumulation has often been implicated in muscle fatigue (Fletcher and Hopkins, 1907). According to Putnam (1979), fatigue produces a marked increase in lactic acid production. However, the mechanism by which lactic acid could be responsible for fatigue is unknown and highly disputed.

The accumulation of H^+ results in a decreased pH below the normal 7.4 and leads to metabolic acidosis. According to Martin et al. (1998), metabolic acidosis has been shown to have a major role in inducing muscular fatigue during short, high intensity exercise. Several potential mechanisms have been proposed to explain the link between metabolic acidosis and the predisposition to muscular fatigue. These mechanisms may include decreases in pH associated with lactic acid accumulation, causing inhibition of the enzymes lactate dehydrogenase and phosphofructokinase, thereby decelerating glycolysis; Ca^{2+} can be prevented from binding to troponin; and H^+ can stimulate pain receptors (Martin et al., 1998). Despite the reputation of lactate as a limiting factor in exercise, more recent research however (Pederson et al., 2004; (Brooks, 2007, Brooks, 2010, Gladden, 2008), has purported that lactate actually has protective effects during muscle fatigue providing improved conditions for muscular contractions.

2.12. Sodium Bicarbonate and Recovery

Few studies have examined the effect of SB supplementation specifically on recovery from high intensity exercise and subsequent high intensity performance. Bishop et al. (2004) observed performance enhancement in repeated bouts of 5 x 6 second sprint cycling, separated by a 24 second recovery period, following induced alkalosis through SB supplementation. Similarly, exogenous ingestion of SB prior to 10 x 10 seconds of maximal cycling with 50 seconds recovery between each bout was found to elicit significantly greater peak power and total work (Lavender and Bird, 1989). Verbitsky et al. (1997) also reported positive results with SB supplementation in terms of recovery. The increased capacity of the quadriceps femoris muscle to generate torque with SB supplementation following high intensity cycle ergometer performance demonstrated a reduction in muscle fatigue and enhanced recovery. Siegler et al. (2010) found that a combination of SB supplementation and active recovery improved performance in repeated 30s bouts of maximal exercise separated by 3 minutes when compared with a combination of PLA and passive recovery; PLA and active recovery; and SB and passive recovery. This study appears to be unique in that the researchers examined the effects of the combination of SB supplementation with different recovery modalities (passive and active recovery) on recovery from exercise and subsequent performance.

In contrast to these findings, Aschenbach et al., (2000) found no improvement with SB supplementation in performance of eight maximal 15 second upper body sprints with a 20 second recovery period between sprints. Zabana et al. (2011) reported that, although SB ingestion had resulted in significant alterations in acid-base balance, no improvements were found in the performance of three consecutive Wingate tests separated by 15 minutes recovery. Similarly, SB supplementation prior to high-intensity intermittent running was shown to induce metabolic alkalosis but did not impact on performance or subsequent performance (Price and Simons, 2010).

This finding of induced metabolic alkalosis is indicative of the majority of the research regarding SB supplementation. However, as is demonstrated by the aforementioned

studies, this does not always translate into an improved performance, recovery or subsequent performance. Robergs et al., (2005) provides a rationale for the potential benefit of pre-exercise SB supplementation concerning recovery and subsequent performance. This study examined the kinetics of acid-base recovery over a 60 minute recovery period in response to pre-exercise alkalosis and acidosis in high intensity cycling performance. It was noted that recovery of pH to baseline levels may take over 45 minutes. In addition, recovery of blood lactate concentrations may take longer than 60 minutes. Although performance was not influenced by SB supplementation, the elevated HCO_3^- observed both prior to exercise and in the recovery period, demonstrated that induced alkalosis attenuated metabolic acidosis and enhanced pH recovery when compared with acidotic and PLA conditions (Robergs et al., 2005). Further research is necessary to determine the effect of different rates of pH and lactate recovery on subsequent exercise and the role that SB ingestion may play in this process through a pre-exercise elevation of pH.

2.13. Beta-Alanine

Beta-alanine is a non-proteinogenic amino acid, which is also a rate-limiting pre-cursor to carnosine. Carnosine is an important intramuscular buffer, constituting 10-20% of the total buffering capacity in type I and type II fibres. The resultant enhanced buffering capacity associated with increased muscle carnosine has been linked to significant improvements in high intensity exercise performance capacity (Sale et al., 2010). In addition, according to Suzuki et al. (2004), an eight week high intensity sprint training programme has been shown to almost double the carnosine content in vastus lateralis. Suzuki et al. (2002) also found a significant relationship between carnosine concentration in human skeletal muscle and high intensity exercise performance in the form of a 30 second Wingate test. In further support of the this relationship between carnosine concentration and high intensity exercise performance, a trend towards higher carnosine concentrations has been reported in athletes such as sprinters (Parkhouse et al., 1985) and bodybuilders (Tallon et al., 2005).

Ingestion of beta-alanine has been shown to increase muscle carnosine, decrease fatigue and increase total muscular work done (Derave et al., 2007; Artioli et al., 2010). With the exception of carnosine, the concentrations of the other physiochemical buffers are constrained by their involvement in the other reactions. After a 10 week strategy, Hill et al., (2007) reported a muscle carnosine increase of 58% and 80% after 4 and 10 weeks of beta-alanine supplementation respectively. This resulted in an increase of 13% in total work completed. Harris et al., (2006) have also demonstrated that carnosine in skeletal muscle may be increased by up to 60% with 2-4 weeks ingestion of beta-alanine.

Administration of large doses of beta-alanine, however, has demonstrated taurine depletion (Dawson et al., 2002 and Tallon et al., 2005). Beta-alanine ingested in a solution or in gelatine capsules can cause paraesthesia in doses above 10mg/kg body weight (Harris et al., 2006). Harris et al. (2006) investigated various different doses of beta-alanine supplementation and found that 10mg/kg body mass,, which corresponded to a mean beta-alanine level of 800mg, was the maximum tolerated single dose capable of minimising side affects but still eliciting a response in serum-beta-alanine concentration. Return to baseline values occurred three hours after capsule ingestion. As a result, it is possible to increase the daily dose without experiencing side affects provided a minimum period of 3 hours is maintained between single doses.

Recent research has demonstrated that 6.4 g/day of beta-alanine can significantly increase skeletal muscle concentrations (Kendrick et al., 2008). Sale et al. (2012) reported that beta-alanine supplementation resulted in an increase of approximately 9 seconds endurance time in an isometric contraction at 45% of maximum voluntary contraction force until fatigue. The intensity of 45% maximum voluntary contraction corresponds to the work level resulting in the greatest increase in lactate and pyruvate concentration in the muscle (Ahlborg et al., 1972), and therefore, potentially also the greatest decline in intramuscular pH.

A more recent study by Bellinger et al., (2012) examined the effects of a combination of beta-alanine and SB on 4 minute cycling time trial performance. In contrast to the above

findings, the trained cyclists exhibited no significant enhancements in performance with the combination of beta-alanine and SB. No significant improvements in performance were detected with beta-alanine supplementation alone. However, acute SB supplementation alone displayed significantly improved four-minute cycling time trial performance. In a similar study, Sale et al., (2011) also combined beta-alanine with SB prior to performance of high intensity cycling. A dosage of 6.4g/kg/day beta-alanine was shown to improve performance in a cycle to exhaustion at 110% power maximum. However, despite a 4% increase in total time to exhaustion with the added supplementation of SB, no significant enhancements in performance were observed. Although the effects of beta-alanine on performance appears to be generally positive, further research is necessary to examine different exercise intensities and durations, recovery and subsequent exercise performance.

2.14 Summary

Based on a review of the existing literature regarding the ergogenic potential of SB, several observations can be made. Firstly, the response to ingestion of SB can have a high degree of individual variability. This variability may be accredited to the individual's physical conditioning and tolerance of the particular buffer. McNaughton et al. (2008) suggest that approximately 10% of individuals do not tolerate buffering substances well. Another common trend that is observed in studies utilising buffers is that following ingestion, subjects display higher levels of pH, bicarbonate (HCO_3^-) and lactate in exercising blood than subjects that have ingested a placebo. However, this does not necessarily translate into an enhanced performance.

Future research may investigate the efficacy of SB in the following areas: short and long term chronic loading of SB; comparison and possible effects of the combination of SB with additional supplements such as beta-alanine, sodium citrate and caffeine; varying doses and ingestion times relevant to different types, intensities and durations of exercise;

improvement of performances of team field sport athletes; and the potential of SB to act as a recovery modality.

Several studies have examined the effect of SB on recovery of the blood to normal levels following exercise or performance (Pruscino et al., 2008). However, there is limited definitive scientific research investigating the effect of SB on recovery from exercise and its effect on subsequent performance. In addition, the findings of many studies relating to exercise performance are conflicting and contradictory (Peart et al., 2012; Carr et al., 2011). This may be attributed to the high degree of individual variability associated with SB ingestion (Siegler et al., 2010). This research aims to investigate the efficacy of SB ingestion to induce alkalosis and its potential effects on high-intensity intermittent exercise, recovery from exercise and subsequent performance. This aim will be achieved through the implementation of three related studies. These studies will be outlined in detail in the following three chapters.

Chapter 3

Study One: The Effects of Acute versus Chronic Sodium Bicarbonate Supplementation on High-Intensity Intermittent Performance

3.1. Introduction

The aim of this study was to examine the effects of both acute and chronic SB supplementation versus PLA on high-intensity intermittent sprint performance in sub-elite male rugby players. The hypothesis tested stated that acute and chronic SB supplementation would induce sufficient elevations in $StdHCO_3^-$, pH and BE-Ecf to delay fatigue and result in enhanced performance of high intensity intermittent sprints on a cycle ergometer when compared to a PLA trial. Given the limited research currently available regarding both chronic SB supplementation and also the effect of SB supplementation of repeated bouts of high intensity exercise, the following chapter will discuss the methodology and results associated with the controlled laboratory-based test protocol employed.

3.2. Methods

3.2.1. Subjects

Ten healthy active males, aged between 18-35 years were recruited through correspondence with rugby clubs throughout Leinster to participate in the study. Subjects consisted of trained club rugby players currently playing at a Junior 2 level or above and with no less than 5 years playing experience. Subjects were instructed to maintain their normal dietary intake, avoid strenuous exercise and abstain from alcohol in the 48 hours immediately prior to all trials. Prior to participation in the study, each subject provided written informed consent (*Informed Consent Form, Appendix A1*) and completed medical screening forms (*General Health Questionnaire, Appendix B*). Subjects were excluded from the study if they exhibited any contra-indicators to high intensity exercise or if they used supplements or drugs that may have interfered with the results. The study was approved by the Dublin City University's (DCU) Research Ethics Committee.

3.2.2. Anthropometric Measurements

Baseline anthropometric measurements were recorded prior to commencement of any physiological testing. Body mass (kg) was assessed barefoot and in minimal clothing using a portable digital scales and stadiometer (Seca, Germany). Data was reported to the nearest 0.1 kg. Standing height (cm) was measured to the nearest 0.1 cm using the same device. Body mass index (kg/m^2) was calculated by dividing each individual's body mass (kg) by the square of their height (cm).

3.2.3. Overview of Experimental Design

The study took place in the Human Performance Laboratories (HPL) in Dublin City University (DCU). 10 competitive rugby players were recruited to take part in this study. Each subject was required to report for a habituation trial. Following this, each subject completed four trials on separate days, with no less than seven days between PLA and SB trials to allow a sufficient wash-out period following supplementation. The four supplemented trials were carried out using a double-blind, randomised cross-over design:

1. Acute placebo trial
2. Chronic placebo trial
3. Acute sodium bicarbonate trial
4. Chronic sodium bicarbonate trial

The habituation trial enabled subjects to become familiar with the performance test (*see Section 3.2.5 below*) and control for any potential learning effects within the supplemented trials. Within this habituation, subjects were given a verbal explanation of the test prior to three sub-maximal practice efforts. Subjects were then instructed to complete the full repeat-sprint performance test at maximum effort. Subjects were deemed to be habituated to the protocol as no further increments in performance were observed in the latter sprints of the performance test. During the habituation visit, subjects were provided with precise, verbal and written instructions on the appropriate

procedure for supplementation over the 5 day period as well as being given the opportunity to ask questions.

Subjects were also provided with five daily individualised measures of either sodium bicarbonate (SB) or placebo (PLA) based on body mass. Each daily dose of either 0.3g/kg SB or 0.02g/kg PLA (maltodextrin) was contained in identical gelatine capsules (matched for capsule number), which ensured the subjects remained blinded and, therefore, unbiased. Subjects were advised to remain hydrated and to adhere to their normal diet throughout the 5 days of chronic supplementation. In addition, participants were instructed to replicate the same diet and hydration pattern for the 48 hour period before each trial. This included a 3 hour fasting period prior to each performance trial. Alcohol, caffeine and strenuous exercise were not permitted in the 48 hours prior to the performance test.

3.2.4. Supplementation Protocol

Subjects were required to fast for 3 hours prior to each performance test for both acute and chronic protocols.

i) Acute Supplementation

Acute supplementation consisted of ingesting either SB (0.3g/kg BM) or PLA split into three equal doses taken at 90, 60 and 30 minutes prior to the performance test. Each dose of 0.1g/kg BM of SB (0.3g/kg BM in total) or PLA was consumed with 200mls water taken with each of the three doses every 30 minutes prior to the performance test.

ii) Chronic Supplementation

Chronic supplementation involved a five day loading period with SB or PLA. Following acute supplementation on Day 1, each subsequent day of the chronic loading period involved supplementation with 0.3g/kg BM split into three doses taken

at 9am, 12pm and 3pm each day. On Day 6, subjects ingested no supplement and carried out the performance test (*see Figure 3.1*). Previous studies have shown that the 5 day loading period followed by no supplementation on the day of the performance test may induce pre-exercise alkalosis sufficiently while also allowing time for any gastrointestinal discomfort that may affect performance to be alleviated (McNaughton et al., 1999).

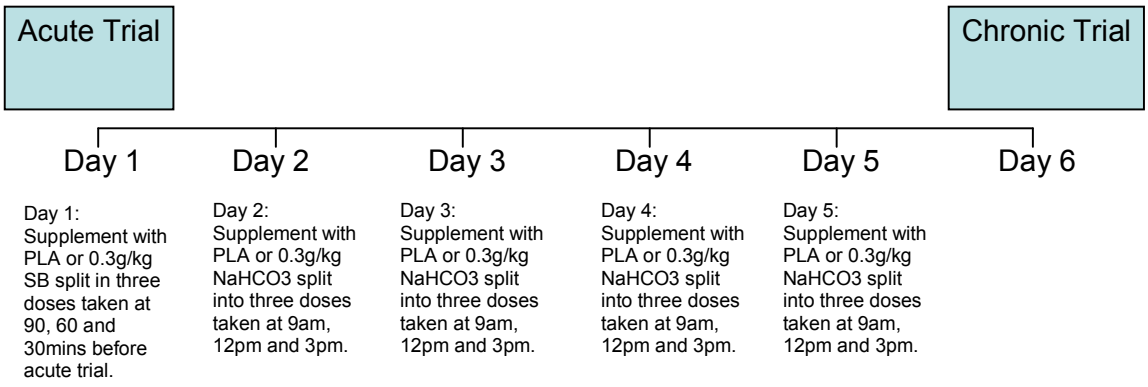


Figure 3.1: Schematic of acute and chronic supplementation protocols

3.2.5. Performance Test

The performance test selected for this study was a high-intensity, intermittent, controlled laboratory-based sprint test on a 'Monark 824E' cycle ergometer (Monark Exercise, Varberg, Sweden). The cycle ergometer (*see Illustration 3.1*) recorded various forms of power output and was automatically calibrated by the patented self-regulating braking system. Absolute peak power output (PPO) and mean power output (MPO) were recorded for all sprints. PPO and MPO were also expressed relative to body mass (relative PPO and relative MPO). Total work was calculated as a product of the sum of the MPO for each sprint and the duration of the sprint (10s). Fatigue index was also calculated for each sprint as the PPO minus the minimum power output, expressed as a percentage of the PPO (Price et al., 2003).

Illustration 3.1: Monark 824E Cycle Ergometer

Prior to each performance test, subjects performed a standardised pre-test warm-up, cycling for 5 minutes producing a power output of 120W against a resistance of 2 kg for each subject. Following the warm-up, subjects were required to perform 6 x 10 second maximal sprints (Wingate tests) at a resistance set at 7.5% BM for each subject. Each sprint was followed by a 50 second active recovery period in which subjects cycled against the resistance of the flywheel only at a cadence of 50 revolutions per minute (rpm) for the initial 45 seconds of the 50 second recovery period. A 5 second countdown was then provided before each sprint in which the subjects were required to maintain a cadence of 90 rpm. Following the 5 second countdown, the cradle loaded with 7.5% BM for each individual subject was dropped and the 10 second sprint commenced (*see Table 3.1)*.

Table 3.1: Wingate Protocol

Stage	Duration (s)	Resistance and Cadence
Countdown to sprint	5	Flywheel resistance only, cadence of 90rpm.
Sprint	10	7.5% BM resistance, maximal cadence.
Recovery period	45	Flywheel resistance only, cadence of 50rpm.

To avoid any pacing, subjects were instructed to exert themselves maximally during the test prior to each trial and reminded between sprints. Strong verbal encouragement was provided to each subject during all sprints. Optimal seat height, determined by a knee angle of between 25 and 35 degrees at the bottom of the pedal stroke, was adjusted for each subject during the habituation trial. The same seat height was used in all subsequent trials. Subjects were instructed to remain in the seated position throughout the entire test. Adjustable toe clips were used to tightly secure the subjects' feet.

3.2.6. Blood Lactate Analysis

Earlobe blood samples for blood lactate analysis were taken from the ear prior to ingestion (baseline), immediately before the warm up after all supplementation (pre-test) and in the 50-second recovery period immediately after the first, third and sixth sprints of the intermittent sprint test. Blood lactate measurements were obtained by drawing 1ml of blood from the earlobe. To ensure an accurate reading was recorded, the sample site (earlobe) was cleaned with an alcohol wipe prior to blood being drawn. The skin was then dried to avoid haemolysis and the earlobe was pricked with a lancing device. Gentle pressure was applied to the earlobe to obtain a blood sample. This first sample of blood was wiped away with gauze to make certain that perspiration did not contaminate the blood sample for lactate analysis. Pressure was applied to the earlobe once more to obtain a second sample of blood. At this point, a lactate test strip was inserted (without the

reaction area being touched) into the blood lactate analyser (Lactate Pro™, Quesnel, Canada). The blood from the earlobe was then touched with the tip of the lactate test strip and the blood lactate analyser produced a lactate reading within 60 seconds (see Illustration 3.2). The blood lactate analyser was calibrated with the calibration strip prior to each testing session and all lancing devices and lactate strips were disposed of safely upon completion of the lactate analysis.

Illustration 3.2: Lactate Pro Blood Lactate Analyser

Each lactate test strip contains two reagents in the reaction layer – lactate oxidase (1.92 units) and potassium ferricyanide (0.096 mg). When the blood touched to the tip of the lactate test strip reaches the reaction layer of the strip, lactate in the blood sample reacts with lactate oxidase (LOD). Simultaneously, potassium ferricyanide (oxidised form) located in the reaction layer, produces potassium ferrocyanide (reduced form). Potassium ferrocyanide is produced relative to the concentration of lactate in the blood sample. The build up of potassium ferrocyanide is then oxidised back to potassium ferricyanide and with this reaction an electrical current is produced and converted into the lactate concentration of the blood sample and displayed on the lactate analyser screen. The blood lactate analyser (Lactate Pro™, Quesnel, Canada) performs with a measuring range of 0.8 - 23.3 mmol/L. In terms of precision, the coefficients of variation for blood samples

containing normal and abnormal levels of lactate are 3.2% (± 0.07 mmol/L) and 2.6% (± 0.3 mmol/L), respectively, according to the Lactate Pro^{TM} manufacturer specifications.

3.2.7. Blood Gas Analysis

Blood gas analysis was used to measure arterialised capillary blood pH, standard bicarbonate ($StdHCO_3^-$), base excess (BE-Ecf), partial pressure of carbon dioxide in blood (PCO_2) and partial pressure of oxygen in blood (PO_2). Blood gas analysis, using a capillary earlobe sample, was carried out at identical time points to blood lactate analysis – baseline (prior to ingestion), pre-test, and immediately after Sprint 1, 3 and 6. Prior to baseline and pre-test samples, a topical vasodilator cream was applied to the earlobe for a period of 5 minutes in order to arterialise the capillary sample. Prior to puncturing the skin, this vasodilator cream was removed with gauze and the sample site was cleaned with alcohol to avoid contamination of the sample. At all other blood sampling time points, given the necessity to obtain an immediate sample, manual massage of the earlobe was used to ensure maximum vasodilation.

Using a similar sampling technique as outlined for blood lactate analysis in Section 3.2.6, the earlobe was cleaned with alcohol, dried and then pierced with a sterile, disposable lancet. The first drop of blood was removed and then 100µL capillary tubes were used to collect 90 µL capillary blood samples for blood gas analysis. The capillary tubes were held horizontally to the earlobe and collected the blood sample by capillary action. The blood samples were analysed immediately to avoid aeration of the sample using the 'ABL77 Blood Gas Analyser' (Radiometer Medical, Denmark). Upon collection of the capillary sample, a capillary sample adapter was attached to the inlet probe on the blood gas analyser (see Illustration 3.3). The capillary tube containing the blood sample was then inserted into the sample adapter and the capillary blood sample was aspirated into the blood gas analyser for analysis. The blood gas analyser had an integrated calibration system, which was activated before and after every use. The portable blood gas analyser, which had been validated against a number of clinical laboratory analysers (Prichard et

al., 2006; Stevenson et al., 2004), was also routinely assessed by external quality controls.

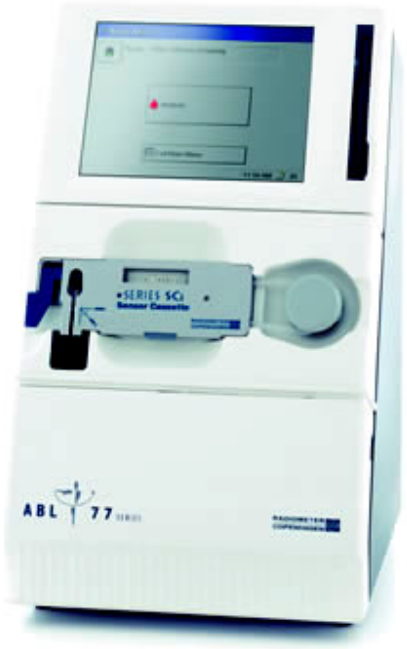


Illustration 3.3: ABL77 Blood Gas Analyser

3.2.8. Heart Rate

Heart rate was measured throughout each test and recorded at similar intervals to the blood samples - baseline, pre-warm up and immediately after Sprint 1, 3 and 6. Heart rate was recorded using a wireless Polar heart rate monitor (Polar Vantage NV™ Polar, Port Washington, NY).

3.2.9. Subjective Ratings

3.2.9.1 Muscle Soreness

A level on the muscle soreness scale (*Appendix C*) was selected by each subject at identical time points to the blood sampling (baseline, pre-warm up and following Sprint 1, 3 and 6). The muscle soreness scale was a simple 11 point scale ranging from 0-10, which was used to allow subjects to rate their muscle soreness levels at different points before and after each aspect of the testing. The muscle soreness scale is adapted from the differential descriptor scale (DDS) from Gracely et al., (1978), which involved a soreness rating based on both sensory and affective descriptors.

3.2.9.2 Rating of Gastro-Intestinal (GI) Discomfort

Subjects rated GI discomfort on a scale of 0-5 at baseline, pre-warm up and immediately following Sprint 1, 3 and 6.

3.2.10. Statistical Analysis

SPSS for Windows v.16.0 (SPSS, USA) was used to conduct the statistical analysis. Normality of data distribution was tested using the Shapiro Wilks test. As the same subjects were used for each of the four treatments, an analysis of variance (ANOVA) with repeated measures was carried out. ANOVA with repeated measures was used to determine any statistical significance between supplementation groups at each time point. Mauchly's Test of Sphericity was used to validate the ANOVA. If Mauchly's test was significant, the Greenhouse-Geisser correction was utilised. An ANOVA establishes the levels of significance between the groups at different intervals. A probability of 0.05 was used to ascertain significance. After obtaining a significant ANOVA result, post hoc Bonferroni testing was used to determine which means were different from each other and the location of any significant findings.

3.3 Results

3.3.1. Subject Characteristics

The descriptive and anthropometric data for the ten subjects are presented in Table 3.2.

Table 3.2: Descriptive and Anthropometric Data

Variable (n=10)	**Mean (± SD)**
Body mass (kg)	86.1 ± 9.7
Height (cm)	183.8 ± 5.9
Body mass index (kg/m^2)	25.5 ± 2.6
Age (yrs)	28 ± 5

3.3.2. Standard Bicarbonate ($StdHCO_3^-$)

As illustrated in Figure 3.2, $StdHCO_3^-$ levels $StdHCO_3^-$ were significantly greater with acute SB supplementation for all time points post-ingestion when compared to all other supplementation protocols (p=0.0005). The acute SB supplementation protocol elicited a mean peak concentration of 30.8 ± 1.1 mmol/L following the ingestion protocol compared to 24.5 ± 1.2 mmol/L for the acute PLA supplementation protocol. Chronic SB supplementation also showed significantly greater $StdHCO_3^-$ following Sprint 1 when compared to the acute PLA supplementation protocol (p=0.016).

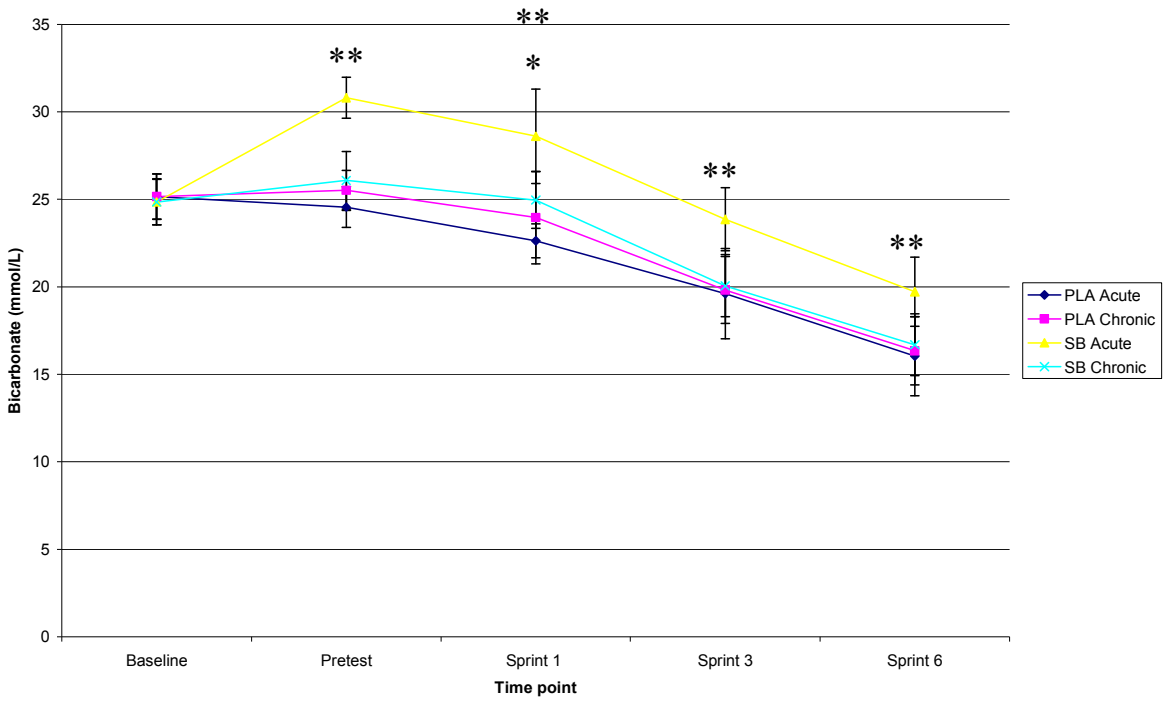


Figure 3.2: Standard bicarbonate ($StdHCO_3^-$) levels across time points for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, repeated measures ANOVA with Bonferroni post hoc test)

3.3.3. Blood pH

Following ingestion, blood pH was found to be significantly higher at all time points for acute SB supplementation when compared to all other supplementation protocols ($p<0.05$). Peak mean pH was recorded immediately prior to the performance test, with acute SB ingestion registering a significantly elevated pH of 7.45 ± 0.02 in comparison to chronic SB (7.39 ± 0.02; $p=0.0005$), acute PLA (7.39 ± 0.03; $p=0.001$) and chronic PLA (7.40 ± 0.02; $p=0.0005$) supplementation protocols. Chronic SB supplementation was not found to exhibit a significantly higher pH at any time point (*see Figure 3.3*) when evaluated against the other three supplementation protocols ($p>0.450$).

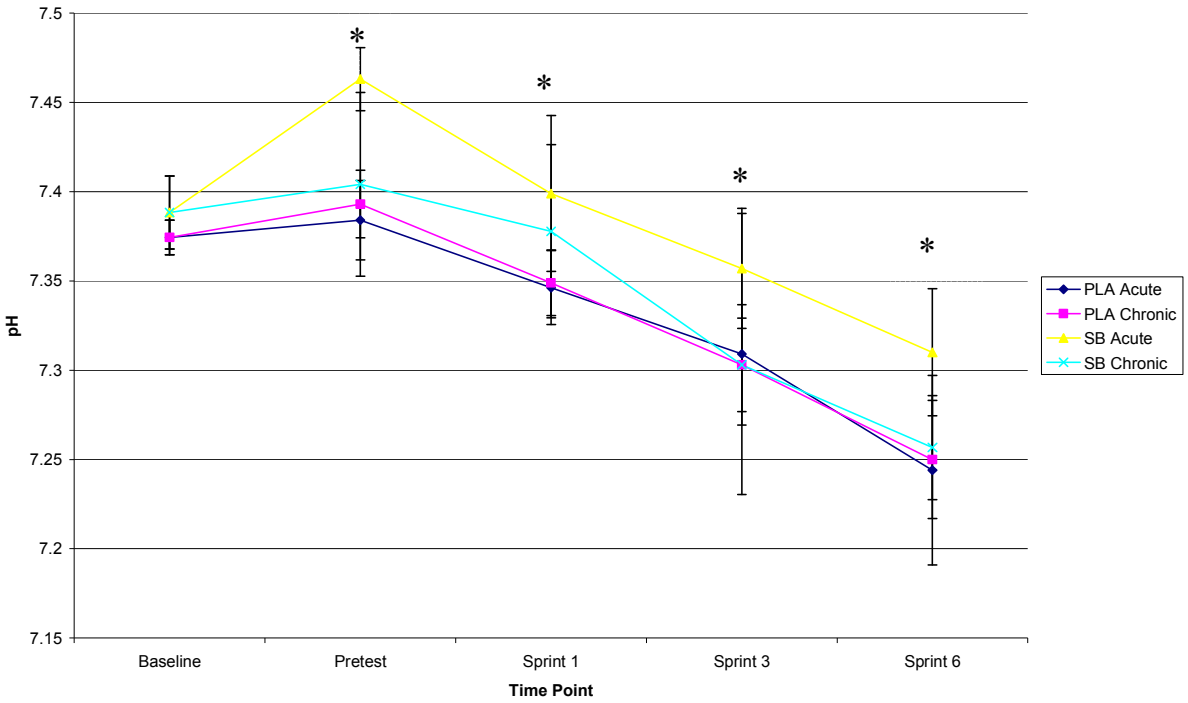


Figure 3.3: pH levels across the time points for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, repeated measures ANOVA with Bonferroni post hoc test)

3.3.4. Base Excess in Extracellular Fluid (BE-Ecf)

As is evident in Figure 3.4, no significant differences were detected between the four supplementation protocols for baseline levels of BE-Ecf (p=1.000). Following supplementation and immediately prior to the repeat sprint cycle test, acute SB supplementation demonstrated a significantly higher BE-Ecf (7.93 ± 1.54 mmol/L) when compared with chronic SB (2.16 ± 1.53 mmol/L; p=0.0005), acute PLA (0.50 ± 1.56 mmol/L; p=0.0005) and chronic PLA (1.66 ± 1.61 mmol/L; p=0.0005) supplementation protocols. Acute SB supplementation also elicited a significantly higher BE-Ecf response following Sprint 1 (4.23 ± 1.96 mmol/L) relative to acute PLA ingestion (-2.23 ± 0.77 mmol/L; p=0.008) and also chronic PLA ingestion (-1.50 ± 2.13 mmol/L; p=0.021).

Although the relationship was not found to be significant, chronic SB supplementation did exhibit a trend towards inducing a higher BE-Ecf when compared with acute PLA supplementation (p=0.083). Following Sprint 3 (the halfway point of the repeat sprint cycle test), and also Sprint 6 (the final sprint), acute SB supplementation displayed significantly higher BE-Ecf values than all other supplementation protocols (p<0.001).

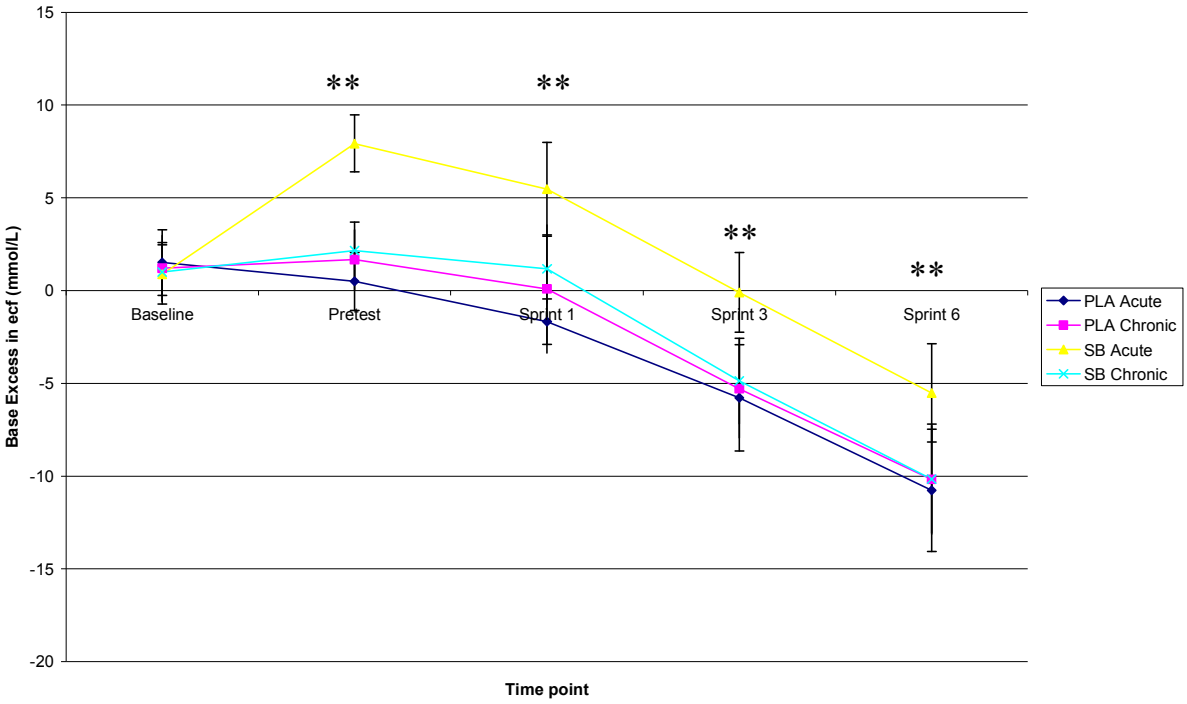


Figure 3.4: BE-Ecf across time points for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, repeated measures ANOVA with Bonferroni post hoc test)

3.3.5. Blood Lactate Analysis

Baseline lactate levels were identified as lying within the normal ranges prior to supplementation for all subjects (0.8-1.8 mmol/L) with no significant differences between the groups (p = 1.000). Following acute SB supplementation, blood lactate concentrations

(*see Figure 3.5*) were found to be significantly greater than the other supplementation protocols after the final sprint only. Following the sixth and final sprint, acute SB ingestion resulted in a mean blood lactate concentration of 13.71 ± 1.92 mmol/L, which was significantly greater than acute PLA (11.76 ± 1.53 mmol/L; p=0.008), chronic PLA (11.56 ± 2.1 mmol/L; p=0.029) and chronic SB ingestion (11.76 ± 2.07 mmol/L; p=0.016) supplementation protocols.

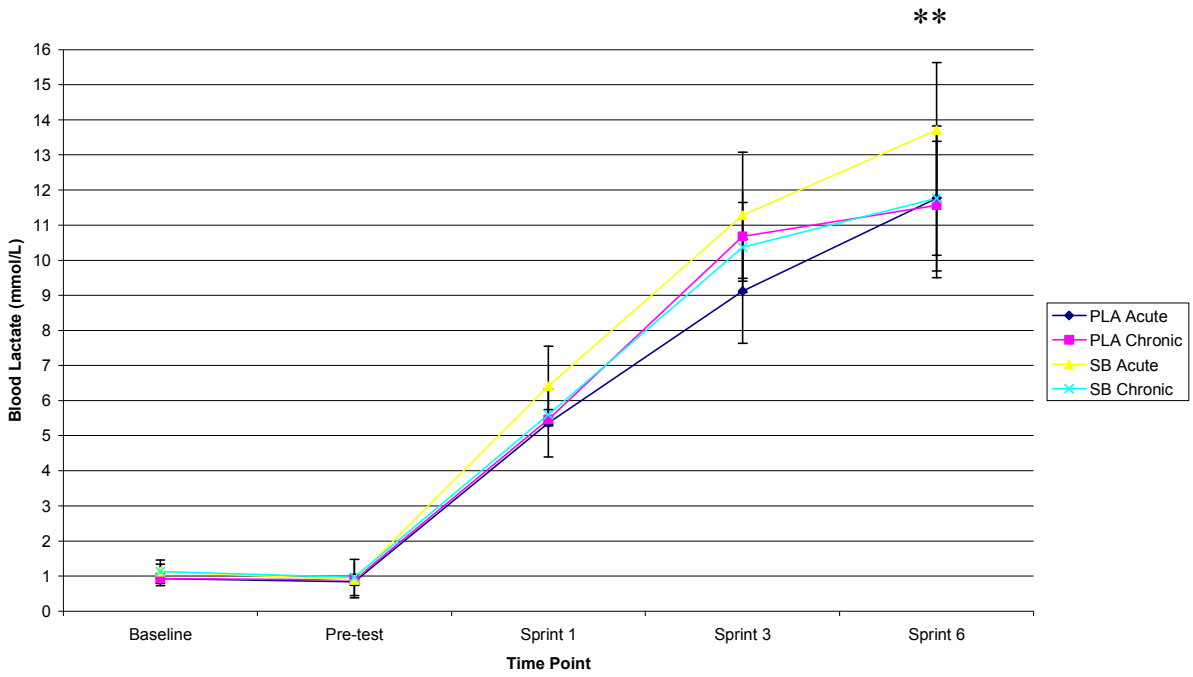


Figure 3.5: Comparison of lactate concentrations for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, repeated measures ANOVA with Bonferroni post hoc test)

3.3.6. Partial Pressure of Carbon Dioxide (PCO_2)

As illustrated in Figure 3.6, significant differences between supplementation protocols were found for mean PCO_2 at two separate time points. Following Sprint 3, acute SB ingestion produced a significantly higher PCO_2 when compared to acute PLA (6.04 ±

0.53 vs. 5.56 ± 0.58 kPa respectively; p=0.044). In addition at this time point (Sprint 3), although not reaching statistical significance, a trend towards significance was identified between acute SB and chronic PLA ingestion (p=0.087). Following Sprint 6, however, the difference between acute SB and chronic PLA ingestion was found to be significant with acute SB supplementation exhibiting an elevated PCO_2 (5.44 ± 0.54 vs. 5.01 ± 0.71 kPa; p=0.014).

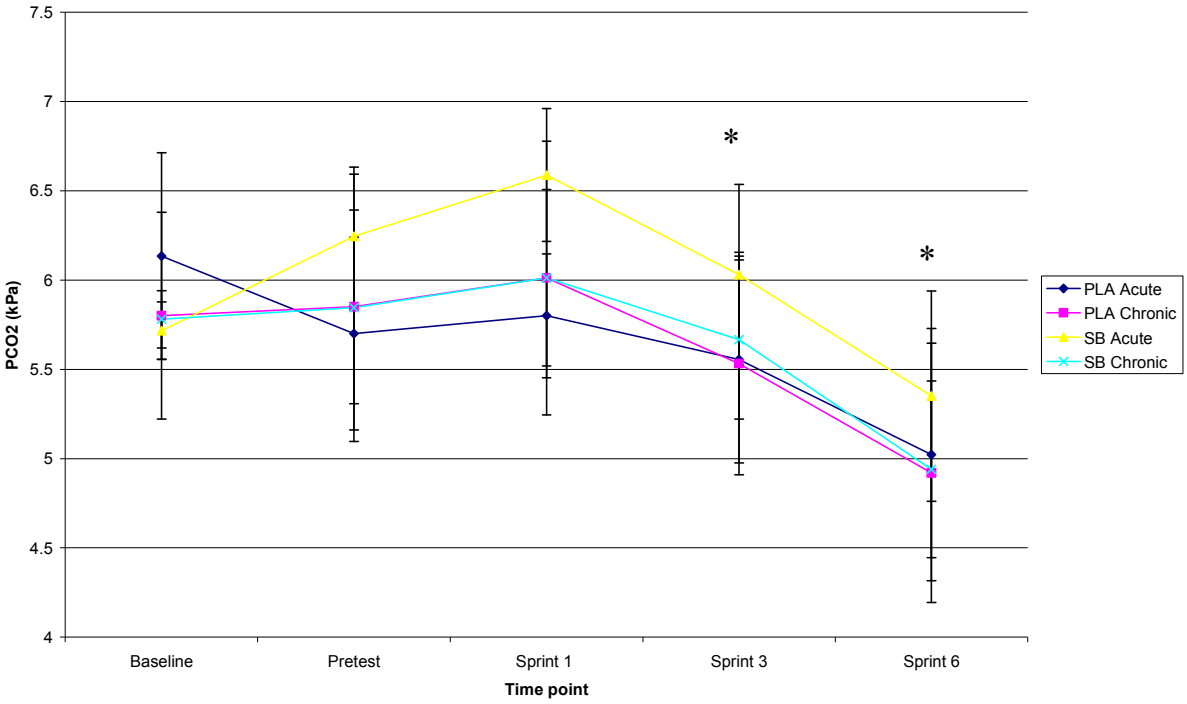


Figure 3.6: PCO_2 (kPa) across time points for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, repeated measures ANOVA with Bonferroni post hoc test)

3.3.7. Partial Pressure of Oxygen (PO_2)

No significant differences between supplementation protocols were observed at any time point for PO_2 (*see Figure 3.7*). Baseline PO_2 values were identified as 11.03 ± 0.69,

11.22 ± 0.91, 11.63 ± 0.70 and 11.80 ± 1.08 kPa for acute SB, chronic SB, acute PLA and chronic PLA supplementation, respectively ($p>0.114$). Post-test PO_2 values were recorded as 12.25 ± 0.80, 12.55 ± 1.31, 13.14 ± 0.87 and 12.70 ± 6.1 kPa for acute SB, chronic SB, acute PLA and chronic PLA supplementation, respectively ($p>0.100$).

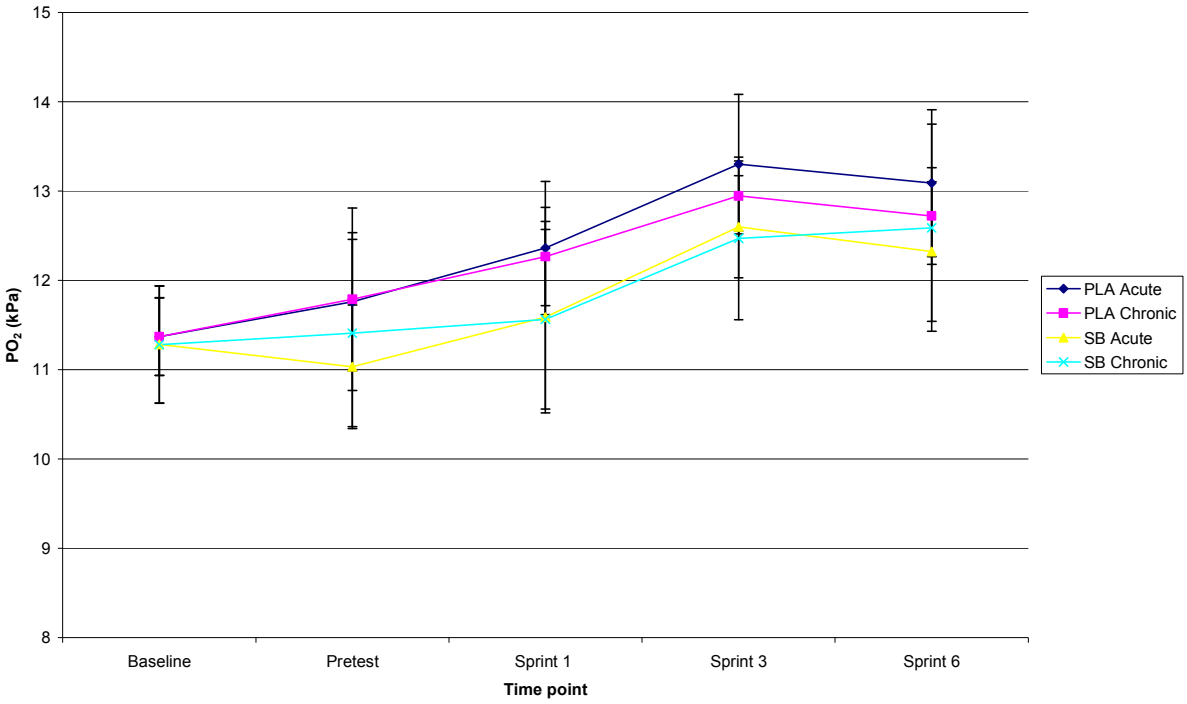


Figure 3.7: PO_2 (kPa) across time points for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, repeated measures ANOVA with Bonferroni post hoc test)

3.3.8. Overview of Performance Outcome

Despite the significantly elevated pH, $StdHCO_3^-$, blood lactate, BE-Ecf and PCO_2 levels observed at certain time points with SB supplementation, no significant differences in performance were demonstrated between subjects across the four conditions.

3.3.9. Peak Power Output (PPO)

Following analysis of absolute mean peak power output (PPO) for all subjects across all six sprints, no significant differences (*see Figure 3.8*) were found among the four supplementation protocols (p=1.000). The highest mean PPO within each supplementation protocol was recorded during Sprint 1, resulting in a PPO of 1028.3 ± 161.4 W, 1027.5 ± 219.4 W, 1032.0 ± 201.8 W and 1016.9 ± 208.3 W for acute SB, chronic SB, acute PLA and chronic PLA respectively (p=1.000). A similar relationship was identified for relative PPO, which also exhibited no significant differences between supplementation protocols at any time point (p=1.000). Absolute and relative PPO across each of the six sprint trials for all supplementation conditions was also analysed. No significant differences were found between the supplementation protocols for any of the sprints from Sprint 1 through to Sprint 6 (p>0.05).

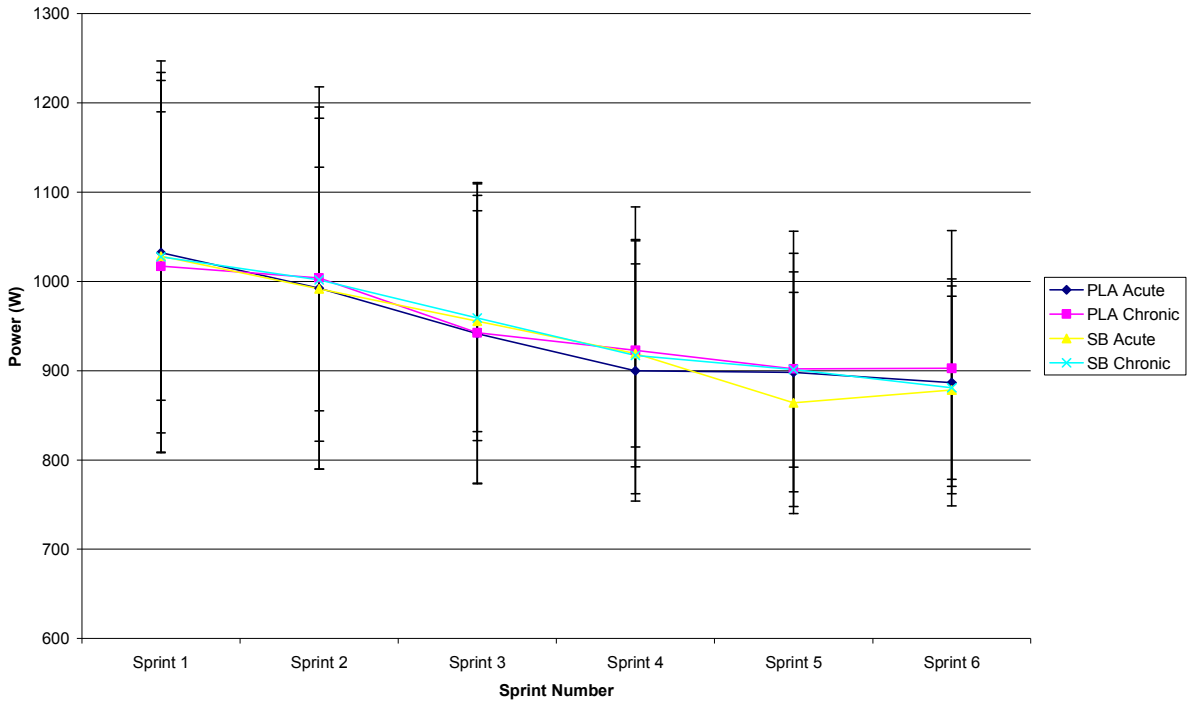


Figure 3.8: Absolute peak power output across all six sprints for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, repeated measures ANOVA with Bonferroni post hoc test)

3.3.10. Mean Power Output (MPO)

Mean power output (MPO) was analysed for each sprint using each of the experimental supplementation protocols with no significant differences observed between protocols in any of the sprints (*see Figure 3.9*). The greatest MPO for each supplementation protocol was observed during Sprint 1, at which point MPO values of 886.8 ± 135.3 W, 894.4 ± 159.2 W, 891.4 ± 160.4 W and 882.5 ± 162.5 W were recorded for acute SB, chronic SB, acute PLA and chronic PLA respectively (p=1.000). Similarly, relative MPO exhibited no significant differences between supplementation protocols (p>0.05). Mean power outputs for each individual sprint from Sprint 1 through to Sprint 6 were also investigated. Similar to PPO, no significant differences were detected between the supplementation protocols (p>0.05).

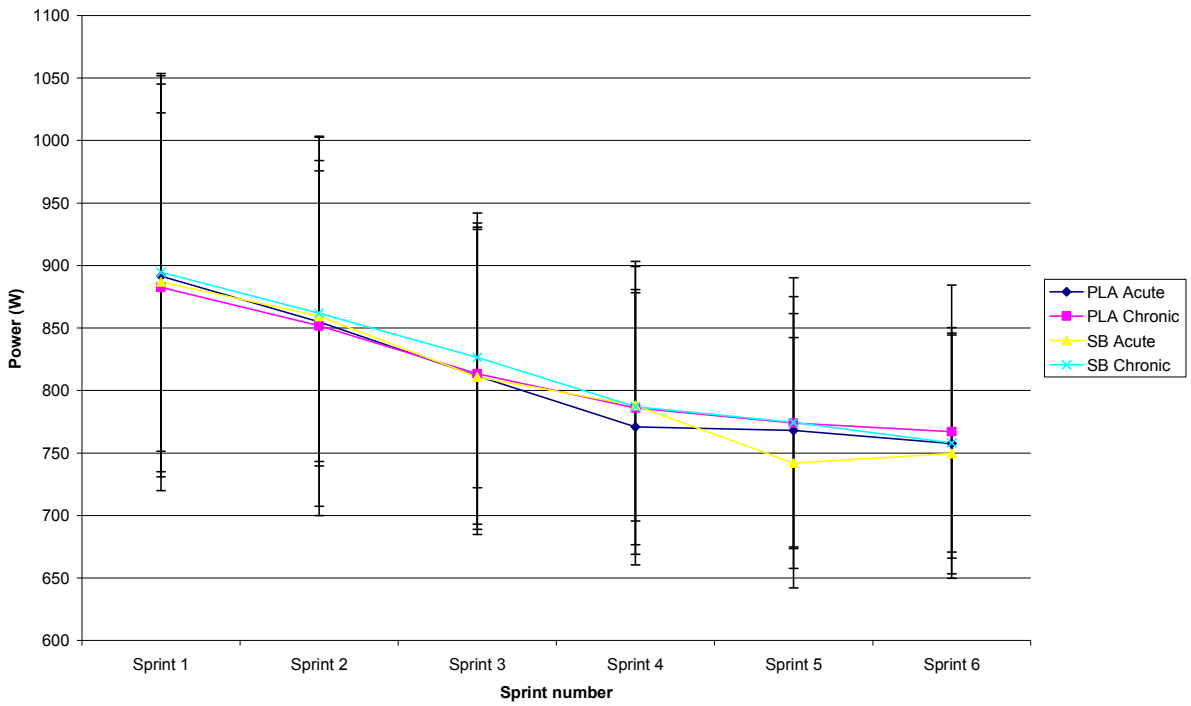


Figure 3.9: Absolute mean power output across all six sprints for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, repeated measures ANOVA with Bonferroni post hoc test)

3.3.11. Total Work

No significant differences in total work performed over the six sprints were identified between supplementation protocols (p=1.000). Total work was calculated as 47.2 ± 6.4 kJ, 48.2 ± 6.7 kJ, 47.6 ± 7.5 and 47.7 ± 8.7 kJ for acute SB, chronic SB, acute PLA and chronic PLA ingestion protocols, respectively.

3.3.12. Fatigue Index

No significant differences between supplementation protocols were found for fatigue index over the six sprints (p=1.000). Fatigue index was calculated as 27.2 ± 6.5 %, 27.9 ± 6.5 %, 27.4 ± 6.7 % and 27.5 ± 5.4% for acute SB, chronic SB, acute PLA and chronic PLA, respectively.

3.3.13. Individual Subject Performance Data Analysis

3.3.13.1. Individual PPO

Individual PPO with each supplementation protocol were also analysed across the six sprints in each trial. Figure 3.10 represents the absolute PPO (W) for each subject for each supplementation trial. In terms of individual peak power, 20% of the subject group (n=2) experienced an improvement in performance with either acute or chronic SB supplementation; 70% of subjects (n=7) exhibited no change in PPO across the four trials; with the remaining 10% of the cohort (n=1) demonstrating a negative performance effect with acute SB supplementation. Three individual case studies displaying differences between the trials are presented below.

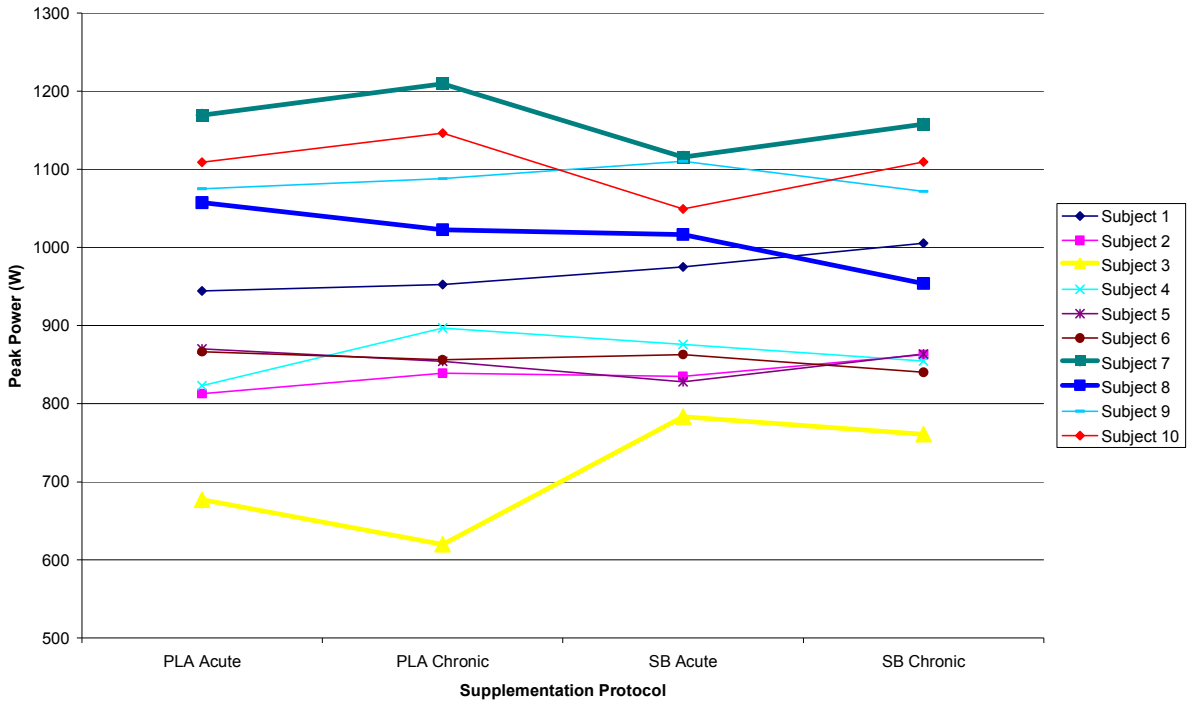


Figure 3.10: Absolute peak power output (W) for each subject using each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, repeated measures ANOVA with Bonferroni post hoc test)

Across the mean of the six sprints, Subject 8 in Figure 3.10 was found to exhibit greater absolute PPO in the acute SB supplementation trial (1016.5 ± 25.3W) than in the chronic SB supplementation trial (953.9 ± 21.2W). The superior absolute PPO associated with the acute SB supplementation trial also transferred to a significantly greater relative PPO (11.05 ± 0.27 W/kg vs. 10.26 ± 0.23 W/kg, respectively) for this subject when compared with chronic ingestion of SB. Although absolute and relative PPO were lower with chronic SB supplementation, Subject 8 also experienced a lower power drop in this chronic SB trial when compared to both the acute PLA and chronic PLA trials (141.7 ± 34.8 W vs. 283.8 ± 37.1 W and 233.8 ± 27.4 W, respectively).

Subject 3, illustrated in Figure 3.10, demonstrated differences between trials for both absolute and relative PPO. Acute supplementation with SB generated a greater absolute and relative PPO (783.5 ± 22.5 W and 11.52 ± 0.33 W/kg) when compared with both the acute PLA (677 ± 32.1 W and 9.96 ± 0.47 W/kg) and chronic PLA (620 ± 21.5 W and 9.12 ± 0.32 W/kg) trials. Subject 3 also displayed greater absolute and relative PPO with chronic SB supplementation (760.9 ± 30.7 W and 11.19 ± 0.45 W/kg) in relation to the chronic PLA trial only (620 ± 21.5 W and 9.12 ± 0.32 W/kg) over the six sprints. A difference in power drop for this subject was also observed, with the acute and chronic PLA trials exhibiting lower power drop values (140.8 ± 29.7 W and 154.2 ± 33.7 W, respectively) than the acute and chronic SB supplementation protocols (230.1 ± 21.5 W and 236.8 ± 32.7 W, respectively).

In contrast to Subject 8 and Subject 3, Subject 7 generated a lower mean PPO with acute SB ingestion when compared to acute PLA supplementation (1115.5 ± 156.9 W vs. 1169.3 ± 136.4 W, respectively).

3.3.13.2. Individual MPO

In terms of individual MPO (*see Figure 3.11*), 10% of subjects (n=1) experienced a performance enhancement with both acute and chronic SB supplementation when compared with the PLA trials; 50% of subjects (n=5) showed no change in performance following supplementation; and 40% of the subject group (n=4) exhibited a negative effect on performance following either acute or chronic SB ingestion. The five subjects displaying a change in performance with SB supplementation are presented as case studies below.

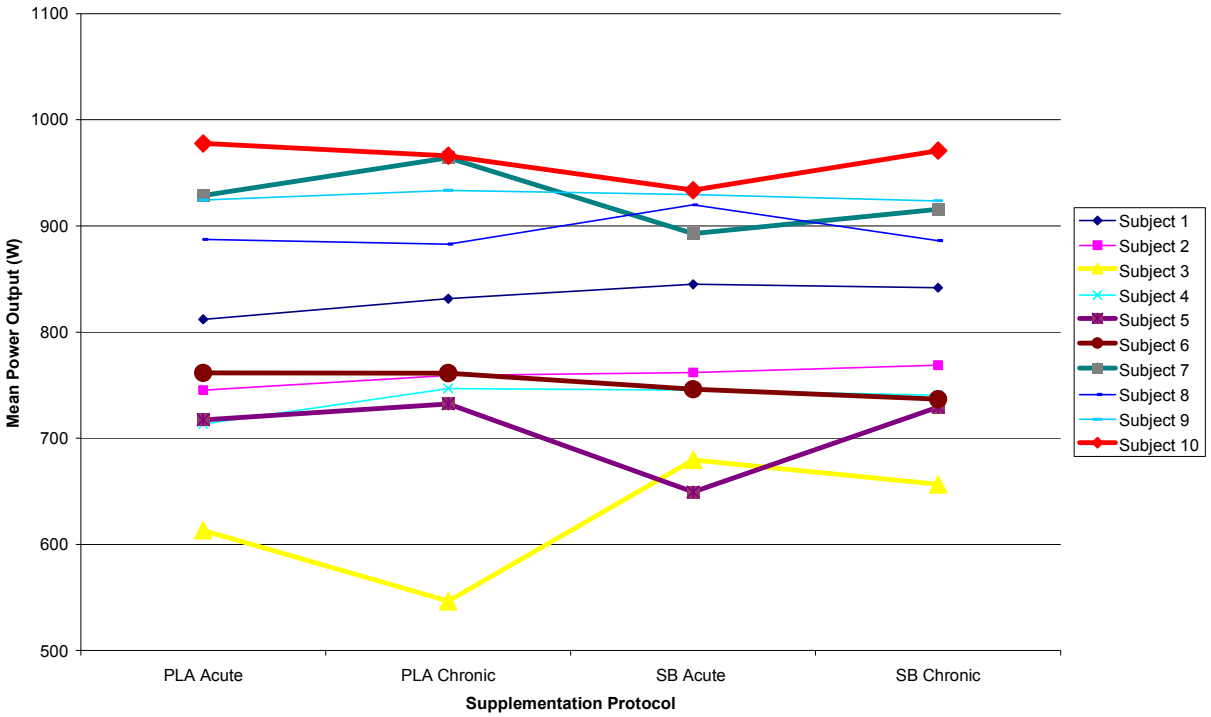


Figure 3.11: Absolute mean power output for each subject using each supplementation protocol (n=10, values denote mean ± SD)

Subject 3 demonstrated differences between all four trials, with acute SB supplementation eliciting the highest MPO (679.6 ± 10.7 W). MPO with acute SB supplementation was found to be greater than that reported for chronic SB supplementation (679.6 ± 10.7 W vs. 656.5 ± 16.8 W), which, in turn, was greater than the MPO for acute PLA supplementation (656.5 ± 16.8 W vs. 613.4 ± 19 W). MPO following acute PLA supplementation was greater than MPO performance with chronic PLA supplementation (613.4 ± 19 W vs. 546.4 ± 8.2 W).

In contrast to Subject 3, Subject 10 displayed a lower MPO for acute SB supplementation when compared with acute PLA ingestion (933.8 ± 72.3 W vs. 977.7 ± 88 W). Similarly, Subject 5 exhibited a MPO with acute SB ingestion that was lower than chronic SB, acute PLA and chronic PLA ingestion (649.4 ± 102.3 W vs. 729.4 ± 105.3 W, 732.5 ± 64.5 W and 717.4 ± 77.2 W, respectively). Subject 6 displayed a lower MPO with chronic SB

supplementation when compared with the acute and chronic PLA supplementation protocols (736.8 ± 25.9 W vs. 761.3 ± 26.4 W and 761.5 ± 20.3 W, respectively). In addition, Subject 7 demonstrated a lower MPO with acute SB supplementation when compared with chronic SB supplementation (892.7 ± 129.4 W vs. 915.6 ± 132 W, respectively).

3.3.14. Gastro-Intestinal (GI) Ratings

The greatest incidence of GI side effects was recorded with the acute SB supplementation protocol. However, this was not found to be statistically different when compared to all other supplementation protocols. To assess the potential effect of GI discomfort experienced by subjects on performance during the trials, a linear regression analysis was conducted. The relationship between GI side effects and repeat-sprint cycle performance was low and non-significant ($r=0.537$, $r^2=0.289$, $p=0.109$).

3.3.15. Heart Rate

No significant differences in heart rate responses were identified between the supplementation protocols at any time point ($p>0.05$).

3.3.16. Ratings of Muscle Soreness

No significant differences in perceived muscle soreness ratings were detected between the four supplementation protocols at any time point throughout the test (*see Figure 3.12*). The highest muscle soreness ratings were reported immediately following Sprint 6 (post-test). At this point acute SB, chronic SB, acute PLA and chronic PLA resulted in muscle soreness ratings of 4.6 ± 2.8, 4.4 ± 2.6, 4.4 ± 3.0 and 3.6 ± 2.5 on a scale of 0-10, respectively ($p>0.05$).

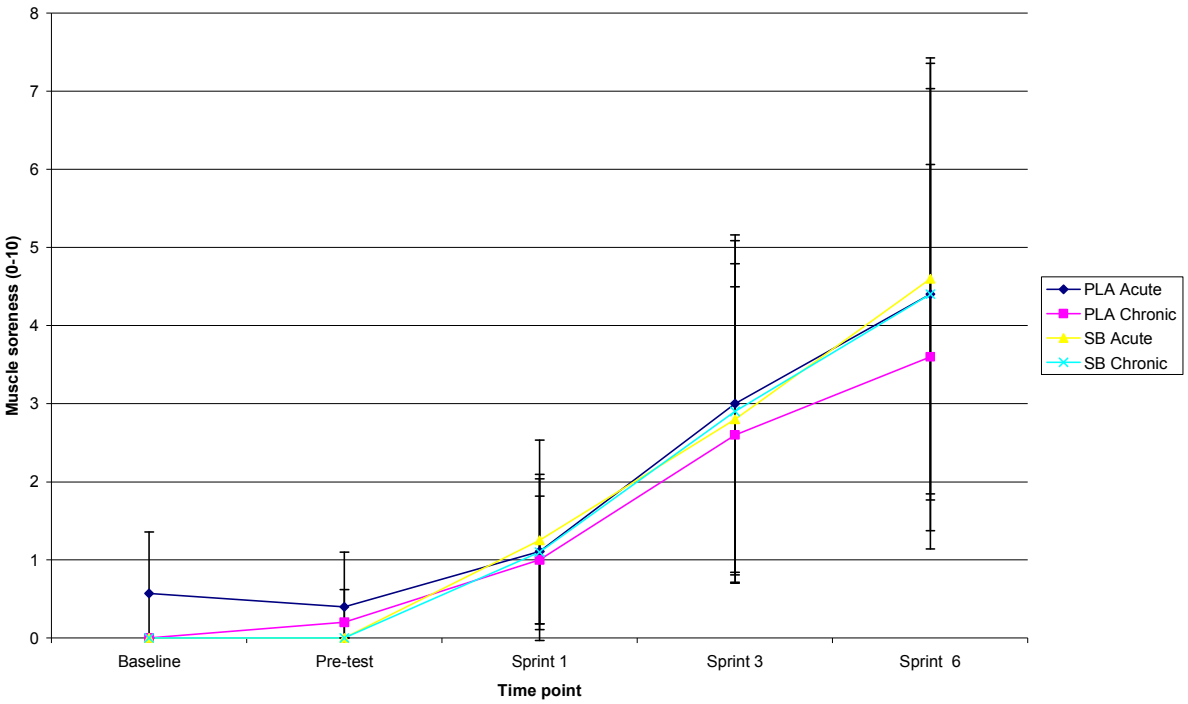


Figure 3.12: Mean muscle soreness ratings using each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, repeated measures ANOVA with Bonferroni post hoc test)

3.3.17. Summary

Acute SB supplementation resulted in significantly elevated blood pH, $StdHCO_3^-$ and BE-Ecf at all time points following ingestion when compared to the other supplementation protocols ($p<0.05$). Chronic SB supplementation also showed significantly greater $StdHCO_3^-$ following Sprint 1 only, when compared to the acute PLA supplementation protocol ($p=0.016$). In addition, chronic SB supplementation exhibited a non-significant trend towards inducing a higher BE-Ecf when compared with acute PLA supplementation ($p=0.083$).

Acute SB supplementation also demonstrated significantly greater post-exercise blood lactate concentrations following Sprint 6, when compared with all other supplementation protocols ($p<0.029$). Significantly elevated PCO_2 values were also observed with acute SB supplementation following Sprint 3 when compared to acute PLA ($p=0.044$) and following Sprint 6 when compared to chronic PLA supplementation ($p=0.014$). No significant differences in PO_2 were detected between the four supplementation protocols at any time point throughout the test.

Despite the changes in acid-base balance elicited by SB supplementation, no significant differences in group performance (absolute and relative PPO, absolute and relative MPO, total work, fatigue index) were observed between the supplementation protocols. Also, no significant differences in heart rate, muscle soreness or GI ratings were reported between the four trials. Analysis of the relationship between GI ratings and performance adjudged the effect of GI discomfort on performance to be minimal and non-significant ($r=0.537$, $r^2=0.289$, $p=0.109$). In addition, as outlined in a number of individual case studies in Section 3.3.13, it is evident that the performance response to SB supplementation has a high degree of inter-individual variability. The results of the current study, along with proposed rationales and possible implications, will be discussed in further detail in the following section (Section 3.4).

3.4 Discussion

3.4.1. Introduction

The purpose of this study was to evaluate the effects of acute and chronic SB supplementation on high-intensity intermittent sprint performance in sub-elite male rugby union players. The major finding of the present study indicated that neither acute nor chronic SB supplementation had a significant ergogenic effect on high intensity intermittent performance. Due to the inconclusive and contradictory findings of the current literature regarding the effect of SB supplementation on performance, this finding may be both supported and opposed. Similar to the current study, Joyce et al., (2012) observed no improvement in mean 200m swim time (mm:ss.00) with acute or chronic loading of SB (acute SB 1:59.57 ± 0:06.21, chronic SB 1:58.53 ± 0:05.64 and PLA 1:59.02 ± 0:05.82 mm:ss.00; $p>0.05$). Conversely, McNaughton et al. (1999) reported a significant ergogenic benefit, in terms of total work completed, with chronic SB supplementation during 60 seconds of high intensity cycling (chronic SB 24.1 ± 0.9 vs. control 21.1 ± 0.9 MJ; $p<0.05$). The ambiguity surrounding chronic SB ingestion may be explained by the small number of studies conducted to date. A thorough review of the current literature revealed that only eight studies have examined the effects of chronic SB supplementation on exercise performance. Of these eight papers, six relate to anaerobic single bout efforts (Carr et al., 2012; Driller et al., 2012; Douroudos et al., 2006; Joyce et al., 2012; McNaughton and Thompson, 2001; McNaughton et al., 1999) with the remaining two relating to endurance performance indicators (Edge et al., 2006; Heil et al., 2012). As a result, the current study is the first to consider the use of chronic SB supplementation as an ergogenic aid in the performance of repeated bouts of high intensity exercise.

3.4.2. Efficacy of SB Ingestion in Inducing Metabolic Alkalosis

The efficacy of the acute SB supplementation protocol in inducing metabolic alkalosis was demonstrated through the significant elevation of standard blood bicarbonate concentration $StdHCO_3^-$ and blood pH. As outlined in the results section, acute SB supplementation maintained a significantly greater $StdHCO_3^-$ and blood pH at all time points when compared to all other supplementation protocols. Acute SB supplementation corresponded to a significant increase of 5.9mmol/L in mean $StdHCO_3^-$ (from 24.9 ± 1.3mmol/L at baseline to 30.8 ± 1.1mmol/L pre-test) and a significant increase of 0.06 in pH (from 7.39 ± 0.02 at baseline to 7.45 ± 0.019 pre-test) following ingestion and immediately prior to the performance test ($p<0.01$). These differences are of a similar magnitude to the 5.4mmol/L increase in $StdHCO_3^-$ recorded in Bishop et al. (2004). However, in contrast to the present study, the increase in $StdHCO_3^-$ observed in Bishop et al. (2004) corresponded to significant improvements in total work and peak power output. A meta-analysis by Matson and Tran (1998), also involving a dose of 0.3g/kg, found a mean overall increase of 5.3mmol/L consistent with the findings of the current study. Furthermore, the increase in $StdHCO_3^-$ of 5.9mmol/L in the current study was slightly higher than the overall difference between SB and PLA of 3.9 ± 0.9mmol/L reported following a meta-analysis of thirty-eight studies (Carr et al. 2011). Carr et al. (2011) also reported a mean overall increase in pH of 0.069 (± 0.018) with acute SB supplementation when compared with PLA. This meta-analysis found a moderate overall enhancement of performance by 1.7% (± 2.0%) for protocols involving acute SB ingestion of 0.3g/kg BW prior to a single bout 1-minute sprint. Therefore, the pre-exercise alkalosis induced by SB ingestion in this study was representative of the magnitude of alkalosis that has resulted in performance enhancements in previous studies. However, in contrast to these studies, no improvements in performance were observed in the current study.

In terms of chronic SB ingestion, although mean $StdHCO_3^-$ was significantly greater than acute PLA at one time point (following Sprint 1), pre-exercise alkalosis was not found to be significantly elevated nor significantly different from the PLA supplementation protocols. In a recent study by Driller et al. (2012) involving chronic SB

supplementation, similar results for acid base balance variables for chronic SB supplementation were presented including a non-significant difference in blood $StdHCO_3^-$ and blood pH post-ingestion, along with no difference in peak blood lactate concentration when compared to placebo. However, significant enhancements in performance were found with both acute and chronic SB supplementation in the 4-minute cycling test. Driller et al., (2012) purported that the enhanced mean power output observed with chronic SB supplementation in spite of a lack of pre-exercise alkalosis may suggest an alternative ergogenic mechanism. However, the state of induced alkalosis appears to be a key predictor of the magnitude of effect on performance. In the most recent meta-analysis of studies relating to sodium bicarbonate use for athletic performance, Peart et al., (2012) identified a significant overall relationship between performance enhancement and the degree of induced alkalosis ($r=0.45$, $p=0.02$). As was demonstrated by McNaughton et al., (1999) and Douroudos et al., (2006), a higher chronic dose of 0.5g/kg body mass, in contrast to the 0.3g/kg body mass, each day may be necessary to significantly increase blood $StdHCO_3^-$ and blood pH.

3.4.3. Effects of Metabolic Alkalosis on Performance

As shown in Section 3.3 of this thesis, although acute SB supplementation was shown to induce significant metabolic alkalosis, as assessed by significantly elevated standard blood bicarbonate concentration blood $StdHCO_3^-$, pH and BE-Ecf, this did not impact on the performance outcome. This finding is corroborated by Cameron et al. (2010) who concluded that acute SB supplementation induced an increase in pre-exercise $StdHCO_3^-$ (30.33 ± 1.25 vs. 24.03 ± 0.34 mmol/L; $p<0.001$), extracellular pH (7.47 ± 0.01 vs. 7.39 ± 0.01; $p<0.001$) and also diminished the exercise-induced acidosis (7.25 ± 0.02 vs. 7.19 ± 0.02) recorded immediately following the performance test. However, no significant effect on high intensity intermittent field sport-specific activity was observed. Similarly, Gaitanos et al. (1991) found that despite a significantly greater muscle buffering capacity associated with acute SB supplementation, power output was unaffected in performance of 10 x 6 second maximal treadmill sprints with 30 second recovery periods. These

findings are also consistent with those of Kozak-Collins et al. (1994) who reported no performance enhancements with acute SB ingestion despite significant pre-exercise alkalosis in repeated 1-minute cycling bouts to exhaustion with 1 minute recovery between each bout.

Conversely, Bishop et al. (2004) found that significant elevations in resting blood $StdHCO_3^-$ (30.0 ± 3.0 vs. 23.6 ± 1.1 mmol/L) and pH (7.50 ± 0.04 vs. 7.42 ± 0.02) recorded with acute SB ingestion coincided with significant increases in total work (acute SB 16.5 ± 3.1 vs. control 15.7 ± 3.0 kJ; $p<0.05$) and power output in sprints 3, 4 and 5 of a 5 x 6 second maximal repeat sprint test with 24 seconds recovery on a cycle ergometer ($p<0.05$). Similarly, results from research by Costill et al. (1984) and Siegler et al. (2010) also adjudged acute SB supplementation to elicit a positive influence on repeated bouts of high intensity performance on a cycle ergometer. However, in both of these studies the duration of sprint length and rest period was not indicative of the physiological demands of many team field sports. The study by Costill et al. (1984) examined the effects of SB ingestion on 5 x 1 minute cycling bouts at 100% VO_{2max} with 30 seconds recovery between each bout (work to rest ratio of 2:1). The fifth bout was performed to exhaustion and time to exhaustion was measured as the performance variable. This study is renowned for reporting a significant 42% increase in time to exhaustion for SB versus PLA during the fifth cycling bout (160.8 ±19.1 vs. 113 ±12.4; $p<0.01$). Siegler et al. (2010) employed a work to rest ratio of 1:6, which is in accordance with the typical mean work to rest ratios of 1:5.7 (back) and 1:5.8 (forward) reported for 15-a-side rugby union by Cunniffe et al. (2009). However, the performance test utilised by Siegler et al. (2010) involved 3 x 30 second Wingate tests on a cycle ergometer each separated by 3 minute rest periods, which represented a more extended sprint and rest duration than that experienced within the demands of rugby union.

A study by Nicholas (1997) made reference to rugby union as an intermittent sport in which players were required to perform a large number of high intensity bouts of between 5 to 15 seconds duration. For the purposes of the current study, in an attempt to comply with the previously reported work to rest ratios and typical match-play sprint durations

(Cunniffe et al. 2009; Nicholas, 1997), a cycle ergometer test consisting of 6 x 10 second maximal bouts with 50 seconds recovery between each bout was undertaken. This protocol was designed to replicate the high-intensity intermittent efforts indicative of rugby union in the controlled setting of the laboratory. Extracellular pH, $StdHCO_3^-$, BE-Ecf and blood lactate concentrations were recorded and monitored throughout testing in order to provide potential justification for any performance variable differences observed between the experimental supplementation protocols.

Given that no performance variable differences were observed between treatments, the physiological demands of the performance test cannot be excluded as a possible explanation for the conflicting results. The exercise mode (cycling) and sprint duration combined with recovery duration involved in the test protocol undertaken in the current study may not have induced significant intramuscular pH changes. As previously mentioned, Bishop et al. (2004) reported an ergogenic benefit with acute SB supplementation in 5 x 6 second cycling bouts performance every 30 seconds. Performance enhancements were also found by Siegler et al. (2012) during 3 x 30 second Wingates with 3 minutes recovery between efforts. Potentially the use of a shorter recovery period between high intensity bouts or, alternatively, longer duration sprints may induce the intramuscular pH changes necessary for SB supplementation to elicit a more positive ergogenic response. Further research is necessary in this area including the possible sampling of muscle biopsy to analyse the intramuscular environment. However, the results of this study would suggest that SB supplementation does not have the capacity to improve performance indicative of the physiological demands of rugby union.

According to Roberts et al. (2008), fatigue during rugby competition may be anticipated through the quantity of high intensity activities performed. Roberts et al. (2008) also noted that repeated bouts of high intensity sprinting are most likely to influence match-play and determine the match outcome. Therefore, the ability to delay the onset of fatigue contributes considerably to the maintenance of high intensity sprint performance throughout a match and, ultimately, the outcome of a rugby union match. Previous studies have reported enhanced performance of single bout high-intensity short duration

cycling following chronic SB ingestion (McNaughton et al., 1999; McNaughton and Thompson, 2001; Douroudos et al., 2006). The major difference between this study and previous studies demonstrating an ergogenic effect of chronic SB ingestion is the repeated bout nature of the performance test. As mentioned, to knowledge this is the first study to examine the effects of chronic SB supplementation on repeated bouts of high intensity short duration exercise.

McNaughton et al. (1999) examined the effects of 0.5g/kg body mass per day of SB (0.2g/kg higher than the current study) for a period of five days on a single bout maximal 60 second sprint on a cycle ergometer. In a similar study by McNaughton and Thompson (2001), a 6 day loading period was employed involving chronic SB ingestion of 0.5g/kg body mass per day. From the resultant blood pH, $StdHCO_3^-$ and BE-Ecf, McNaughton and colleagues (1999) purported that this loading protocol allowed the extra bicarbonate to be stored within the body, thus enhancing the buffer reserve. Both studies reported a significant performance enhancement in single bout high intensity short duration cycling performance. The latter study (McNaughton and Thompson, 2001) also attributed chronic SB loading with a sustained performance enhancement in a second trial performed 24 hours after the initial trial without any further ingestion of SB. However, no acid-base variables were reported pertaining to this second trial. Therefore, inferences regarding sustained alkalosis cannot be supported. In both of the aforementioned studies, the acid-base variables increased significantly above baseline levels in the initial 24 hours after ingestion. The initial elevation in pH, $StdHCO_3^-$ and BE-Ecf recorded was maintained for the remaining four (McNaughton et al., 1999) or five days (McNaughton and Thompson, 2001), respectively, of the loading period but did not exhibit any further increases despite continued daily supplementation. This would suggest that a chronic loading period of less than 5 days may have been sufficient to obtain the same response.

Joyce et al. (2012) also observed a significant degree of pre-exercise alkalosis but no significant alteration in 200m swimming performance with chronic SB loading when compared to PLA. Joyce et al. (2012) established that a chronic SB loading dose of 0.3g/kg body mass per day for 4 days was sufficient to stimulate similar significant

increases in pH and $StdHCO_3^-$ as those previously induced by a higher daily dosage (McNaughton et al., 1999; McNaughton and Thompson, 2001). However, similar to the current study, no performance enhancement was observed.

In the present study, a SB dose of 0.3g/kg body mass per day was administered for five consecutive days prior to performance of 6 x 10 second Wingate's on a cycle ergometer with 50 seconds recovery between each sprint. However, as is shown in the results, acid-base variables, measured immediately before the performance test, had returned to pre-ingestion values for chronic SB supplementation. This finding suggests that the body may have compensated for the daily SB ingestion-induced perturbations in acid-base balance through manipulation of the acid-base status of the urine (Guthrie, 1983) or PCO_2 (Greenhaff et al., 1987). Within the current study, blood gas analysis was not carried out during the 5 day loading phase of the chronic supplementation protocol. However, significantly elevated PCO_2 values were observed with acute SB supplementation following Sprint 3 when compared to acute PLA ($p=0.044$) and following Sprint 6 when compared to chronic PLA supplementation ($p=0.014$). Future research may benefit from daily analysis of blood and urine samples throughout the chronic loading period.

3.4.4. Effects of Metabolic Alkalosis on Post-Exercise Lactate Concentration

Acute SB ingestion has been associated with significantly greater post-exercise blood lactate concentrations when compared with a placebo (Bishop et al., 2004; Lindh et al., 2008; Gao et al., 1988). The additional ingested HCO_3^- circulating in the extracellular fluid promotes a greater efflux of lactate/H^+, through the lactate/ H^+ co-transporter (Juel, 1998), as demonstrated by the widely reported significant elevation of post-exercise blood lactate following SB supplementation (Siegler et al., 2008). In this regard, the current study is consistent with the majority of the literature in terms of post-exercise blood lactate concentration. Following the final bout of the high intensity performance test, acute SB ingestion resulted in a mean blood lactate of 13.71mmol/L ± 1.92, which was significantly greater than acute PLA, chronic PLA and chronic SB ingestion, which

resulted in blood lactate values of 11.76 ± 1.53, 11.56 ± 2.1 and 11.76 ± 2.07mmol/L respectively. Elevated post-exercise blood lactate concentrations associated with acute SB ingestion tend to accompany enhancements in performance (Requena et al., 2005; Bishop et al., 2004; Sutton et al., 1981). Edge et al. (2006) purported that a greater lactate accumulation may occur due to an enhanced buffering capacity as a result of SB ingestion. The enhanced buffering of the excess H^+ allows anaerobic glycolysis to continue, thereby providing energy for a prolonged period and attenuating fatigue (Edge et al., 2006). However, despite significantly elevated post-exercise blood lactate concentration with acute SB ingestion, no performance enhancements were observed.

3.4.5. Gastro-Intestinal Discomfort

In the present study, no statistically significant differences were observed for ratings of gastro-intestinal discomfort between acute SB, chronic SB or PLA ingestion protocols. This contradicts previous findings (Requena et al., 2005), which describe increased GI disturbances with acute SB ingestion. This may be attributed to the staggered loading protocol utilised in the current study, involving ingestion of 0.1g/lg BM at 90, 60 and 30 minutes prior to the performance test. A study by Cameron et al. (2010) used a similar ingestion protocol of 0.3 g/kg BM taken in one dose 90 minutes prior to the performance test. This research found that the severity of GI discomfort was significantly greater with acute SB supplementation when compared to the PLA trial. In the current study, an identical dose of 0.3g/kg BM was distributed into three doses and ingested at 90, 60 and 30 minutes prior to performance in an attempt to maximise blood $StdHCO_3^-$ while minimising GI discomfort. The objective of this protocol was achieved, as is evident from the significant elevation in pre-exercise $StdHCO_3^-$ and the lack of a significant difference in GI ratings between the four supplementation protocols. However, no improvements in performance were observed in spite of the similar GI ratings reported for each supplementation protocol.

3.4.6. Summary

Consistent with the findings within the literature, analysis of both acute and chronic SB ingestion within the current study appears to be inconclusive. In a review by Peart et al. (2012) forty research articles were analysed involving 395 participants (348 males, 47 females), fifteen of the forty studies (38%) found an ergogenic benefit. Although metabolic alkalosis was induced with acute SB ingestion within the present study, identified by increases in blood $StdHCO_3^-$, pH and BE-Ecf, no ergogenic benefit was observed. Overall effect size has been shown to be higher in untrained individuals as opposed to trained individuals (Peart et al., 2012). Trained individuals appear to have a higher muscle buffering capacity and, therefore, SB loading may have a diminished impact on highly trained athletes (Robinson and Verity, 1987). Given that the subjects involved in the current research were trained athletes, this may provide a partial explanation for the insignificant findings in relation to performance.

More positive ergogenic results have also been found in single bout as opposed to repeated bout performance, possibly due to the inability of the buffering system to recover sufficiently in the rest periods associated with repeated bout performance (Peart et al., 2012). This may indicate that the high intensity intermittent exercise protocol involved in the current study may not be enhanced by SB ingestion. Furthermore, in a review by Matson and Tran (1993), thirty-five investigations formed the basis for a meta-analysis. Within this review, it was identified that the nineteen studies showing positive ergogenic effects following ingestion in comparison to the sixteen other studies showing no effect. Price and Simons (2010) cited individual variability as an explanation for a lack of significant group performance enhancements in high intensity intermittent treadmill running despite significant shifts in the acid-base balance with SB ingestion. This may also apply to the current study given the large degree of individual performance variation in response to SB ingestion. Nonetheless, the current study shows no evidence to support the use of either of these specific acute or chronic SB supplementation protocols to enhance performance within this particular high intensity, intermittent cycling test. Although acute SB supplementation did induce significant elevations in

blood parameters as predicted, the overall hypothesis stating that acute and chronic SB supplementation would enhance performance was disproved.

Chapter 4

Study Two: The Effects of Chronic Sodium Bicarbonate Supplementation on Simulated Rugby Sevens Performance

4.1. Introduction

The aim of this study was to examine the effects of chronic five-day supplementation with SB on a specifically designed test protocol developed to replicated the specific physiological demands associated with a typical rugby sevens match. The hypothesis devised stated that chronic SB supplementation, using a specifically designed loading protocol, would stimulate metabolic alkalosis and result in enhanced performance, recovery and subsequent performance of high-intensity intermittent performance when compared to a PLA trial. Having previously analysed the effect of both acute and chronic SB supplementation on a laboratory-based test in Study 1 (Chapter 3), Study 2 aimed to focus on chronic SB supplementation with respect to a field-based sport-specific rugby sevens test protocol. The previous study (Study 1) had identified the potential for the use of chronic SB supplementation through a significantly elevated $StdHCO_3^-$ value at one time point. In addition, by focusing on five days of chronic SB supplementation alone, as opposed to the acute trial involved on Day 1 of Study 1, it was believed that this experiment may add to the limited current literature investigating chronic SB supplementation. The research protocol and results are outlined and discussed throughout this chapter (Chapter 4).

4.2. Methods

4.2.1. Subjects

10 healthy male subjects, aged between 18-35 years volunteered to participate in the study. Subjects consisted of trained club rugby players of Junior 2 level or above and were recruited from University and Leinster rugby clubs. Playing experience of each player was no less than 5 years. Subjects were excluded from the study if they were suffering from any cardiovascular, pulmonary or metabolic diseases; if they were smokers; or if they had any injury or illness that may have affected their performance, as determined by a general health questionnaire (*see Appendix B*) completed by each subject

prior to testing. All experimental procedures were approved by the Dublin City University Research Ethics Committee.

4.2.2. Screening

During the first visit to DCU, written informed consent (*see Appendix A2*) was obtained from all subjects after explanation of the experimental procedure, possible risks and proposed benefits of participating in the study. A general health questionnaire, outlining the subjects' medical eligibility to participate in the study was also completed. Baseline anthropometric measurements including height (m), body mass (kg), resting heart rate (bpm) and body mass index (kg/m^2) were also assessed.

4.2.3. Overview of Experimental Design

The study took place in the Exercise Physiology Research Hall (EPRH) in Dublin City University (DCU). The present investigation took place during the latter phase of the competitive season, with trials being performed no less than 2 days after the previous match in order to standardise pre-test conditions as much as possible. During this phase of the season, players were participating in four training sessions per week (including skill, tactical and physical sessions) with a competitive match at the weekend. In addition to a habituation session on a separate day, each subject successfully participated in two experimental trials which differed only in terms of the supplement ingested in the preceding 5 days (sodium bicarbonate - SB versus placebo - PLA). The purpose of the habituation visit was to familiarise the subjects with the exercise patterns of the specifically developed field based performance test (*see Section 4.2.5*), designed to replicate the demands of rugby union. This initial visit also functioned to control for any performance enhancements in subsequent trials as a result of potential learning effects by ensuring that performances had begun to plateau by the end of the habituation session. Within the habituation session, subjects were given a verbal and physical demonstration before 3 sub-maximal practice sets of the full performance test. Subjects then completed the full performance test at maximum effort. The two subsequent performance trials were

performed in a randomised order using a double-blind, cross-over design and there was no less than a seven-day washout period separating the performance test of one trial and supplementation for the subsequent trial.

4.2.4. Supplementation Protocol

Prior to the first main trial, participants were instructed to maintain their normal their physical activity, diet and hydration pattern for the 48 hour period before the trial and were directed to replicate this before the second trial. The two experimental trials involved supplementation with either SB or PLA. Following on from Study 1, a 5 day chronic loading supplementation protocol with SB and PLA (*see Figure 4.1 below*) was the selected loading protocol as opposed to acute ingestion 90 minutes prior to exercise. Chronic SB supplementation for five days prior to performance with no supplementation on the day of performance has been shown to potentially minimise the gastro-intestinal side effects associated with SB ingestion. Chronic SB supplementation has shown similar peak power outputs, mean power outputs and total work performed when compared to acute SB supplementation. However, decreased pre-performance gastrointestinal disturbances were exhibited with chronic SB supplementation when compared to acute SB supplementation (McNaughton and Thompson, 2001).

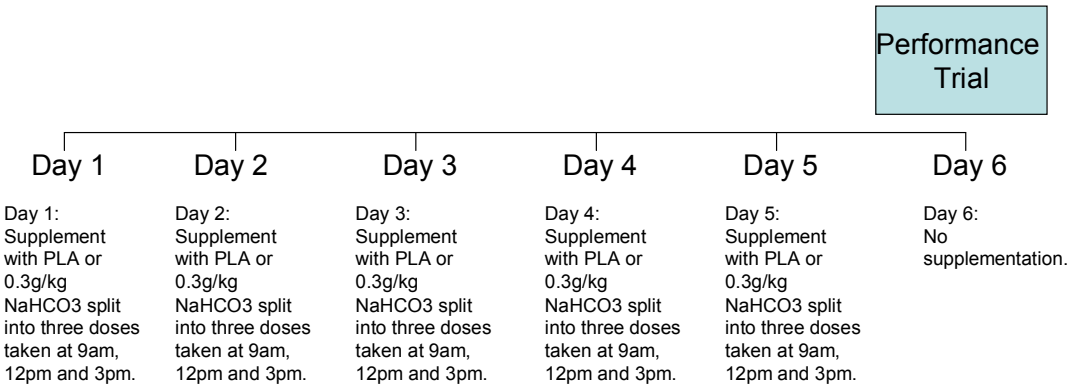


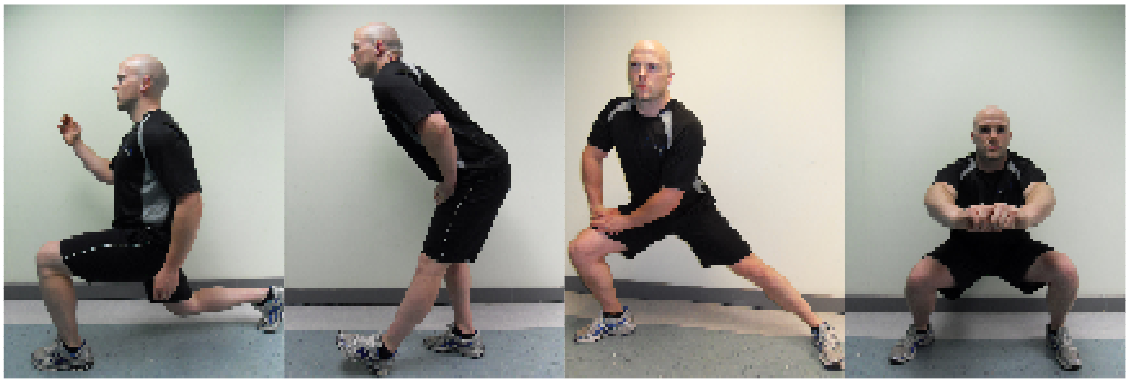
Figure 4.1: Schematic of chronic supplementation protocol

During the habituation visit, subjects were provided with precise, written instructions on the appropriate procedure for supplementation over the 5 day period. Subjects were provided with five measures of their respective daily quantities of SB or PLA. Each daily dose of either 0.3g/kg SB or 0.02g/kg PLA (maltodextrin) was contained in identical gelatine capsules (matched for capsule number), which ensured the subjects remained blinded and, therefore, unbiased. The gelatine capsules also served to attempt counteract any potential gastro-intestinal disturbances due to their purported slow-release mechanism (Carr, et al., 2011). The daily doses were divided into three parts to be ingested at 9am, 12pm and 3pm each day for five consecutive days prior to the performance test. Subjects were advised to refrain from eating 60 minutes either side of ingesting the capsules to avoid gastrointestinal disturbances. Subjects were also advised to remain fully hydrated and to adhere to their normal diet throughout the week. Alcohol, caffeine and strenuous exercise were not permitted in the 24 hours prior to the performance test.

4.2.5. Performance Test

Following the five-day supplementation protocol, subjects returned to the exercise hall on Day 6 to complete the performance test. The performance test utilised in this study was a repeatable, controllable, specific protocol designed to simulate the typical movement patterns observed during GPS movement pattern analysis of rugby union sevens (Higham et al., 2012; Suarez-Arrones et al., 2011). The test, therefore, simulated the physiological demands of rugby union, in which repeated bouts of high intensity activity are interspersed with low-intensity active recovery periods (Suarez-Arrones et al., 2011; Deutsch et al., 1998, Roberts et al., 2008).

Subjects began with a 10 minute standardised warm-up (*see Illustration 4.1*) which consisted of jogging at 3m/s, dynamic stretching (walking lunges, hamstring and groin stretches and sumo squats) along with exercises involving explosive leg movements (high knees, heel flicks and squat jumps).



a) **b)** **c)** **d)**

Illustration 4.1: Dynamic warm-up stretching including a) walking lunge; b) walking hamstring stretch; c) walking groin stretch; d) walking sumo squat.

The performance test was divided into two distinct stages with a 2 minute standardised period of combined static and active recovery separating each stage. A detailed schematic of the performance test is presented in Figure 4.2.

Stage 1

Stage 1 (see Figure 4.2 below which corresponds to the colour code following each step outlined) consisted of six repetitions of a continuous seven step circuit outlined below:

- *Step 1: 20m walk (1.4m/s), 20m cruise (4.2m/s), 20m jog (3m/s)* • • •

- *Step 2: 9m 25kg tackle bag carry shuttle x 2* • • •

    - From a standing start 0.5m behind timing gate A (*Figure 2*), subjects passed through the gate, picked up the tackle bag, carried it 9m and placed it over a line before sprinting 9m back to the start line. The subject then turned and sprinted 9m back to the tackle bag, picked up the bag and carried it back 9m placing it back where it began and sprinted back through timing gate A.

- *Step 3: Reactive agility speed (RAS) test* • • •

    - Subjects sprinted straight 15m from timing gate 1 through timing gate 2, which triggered either timing gate 3 or 4 to flash continuously. The subject continued to sprint, making a sudden change of direction to sprint through the timing gate that was flashing. Gate 3 or 4 only flashed once the subject had passed through gate 2.
    - Reactive agility speed (RAS) may be broken down into three distinct elements:
        - RAS_15m (the initial 15m sprint)
        - RAS_reaction (a visually triggered change of direction reaction element over the subsequent 7m immediately following the 15m sprint)
        - RAS_total (the sum of RAS_15m and RAS_reaction)

- *Step 4: 25s jog back to start line* • • •

    - The subject then had 25s from the end of the RAS above to jog back to the start line.

- *Step 5: 15m sprint – Linear speed (LS)* • • •

    - Upon return to the start line, the subject performed a single, maximum effort 15m sprint between timing gates 1 and 2.

- *Step 6: Jog backwards to start line (3m/s)* • • •

    - After completion of the 15 sprint, subjects jogged back to the start line at a speed of 3m/s.

- *Step 7: Change of direction speed (CODS) test* • • •

    - Similar to the RAS test, subjects sprinted from a standing behind timing gate 1 through gate 2 and changed direction to sprint through gate 3 or 4. However, in contrast to the RAS, subjects were verbally instructed as to which direction to change prior to each sprint. Subjects were encouraged to perform the change of direction as if to evade a defender thus resulting in a sharp change of direction.
    - Similar to RAS, change of direction speed (CODS) may be broken down into three distinct elements:
        - CODS_15m (the initial 15m sprint)
        - CODS_reaction (pre-ordained change of direction element over the subsequent 7m immediately following the 15m sprint)
        - CODS_total (the sum of CODS_15m and CODS_reaction)

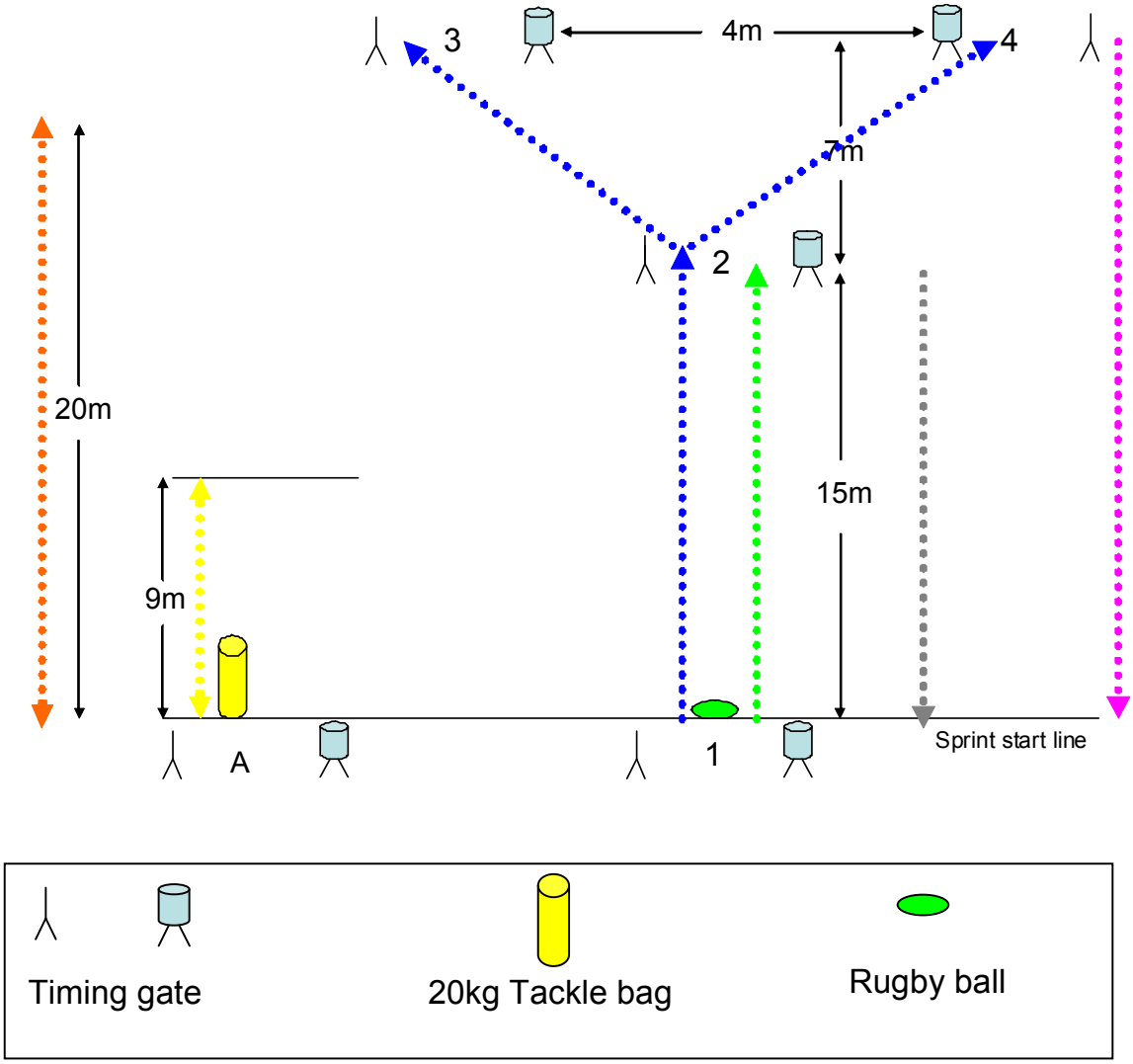


Figure 4.2: A schematic representation of the performance test area (not to scale).

2 minute recovery phase:

- Subjects recovered passively for 1 minute while readings for MS, RPE and blood samples were collected. Subjects then performed 1 minute of active recovery which involved slow walking at 1.4m/s.

Stage 2:

Stage 2 consisted of the Illinois agility test (*See Figure 4.3 below*). The Illinois test consists of an agility course within a grid 10m long and 5m wide as illustrated in Figure 3. Four cones were used to mark the starting point, finishing point and the two turning points. Another four cones were placed down the centre of the grid, an equal distance of 3.3m apart. Each shuttle requires the subject to run approximately 60 metres in total. Each subject began the test lying faced down facing forwards with arms flat on the ground. On the "Go" command the subject jumped to their feet, picked up the rugby ball which was positioned at their head from their start position on the ground and negotiated the course in the fastest possible time. Subjects were instructed prior to the test to keep the ball in two hands at all times and to work maximally and avoid pacing. Each subject completed six shuttles of this agility test with 20 seconds recovery between each shuttle. The test was timed using Photocell Intermediate Beam timing gates (BrowerXS, Vermount, USA).

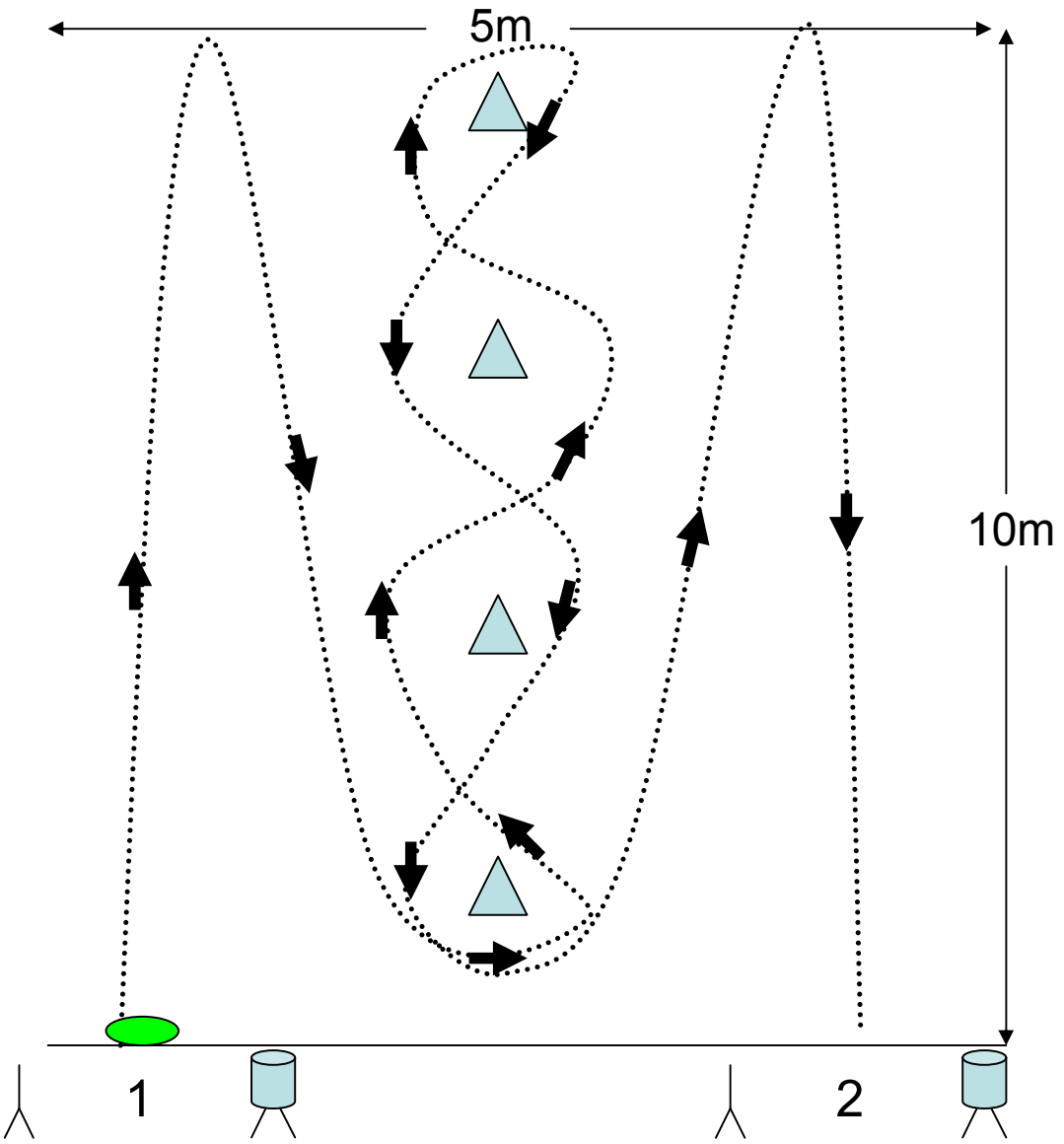


Figure 4.3: Illinois Agility Test

4.2.6. Blood Lactate Analysis

Blood lactate sampling technique was identical to the description in Section 3.2.6. Blood sampling occurred at pre-warm up, pre-test (post-warm up), pre-agility (within the 2 minute recovery period between stage 1 and 2 of the test) and post-test (following stage 2).

4.2.7. Blood Gas Analysis

Blood gas analysis, as previously described in Section 3.2.7, was carried out at identical time points to those mentioned for blood lactate (pre-warm up, pre-test, pre-agility, post-test).

4.2.8. Heart Rate

Heart rate (HR) was recorded throughout the test using recordable HR-enabled watches with heart rate monitors (Garmin, Kansas, USA). Heart rate was subsequently wirelessly downloaded to a computer using the Garmin Training Centre (Garmin, Kansas, USA).

4.2.9. Subjective Ratings

At pre-warm up, pre-test, pre-agility and post-test time points, ratings of GI discomfort, perceived exertion (RPE) and muscle soreness were obtained using visual analogue scales (0-5, 6-20 and 0-10 for GI discomfort, RPE and muscle soreness, respectively).

4.2.10. Testing Variability

Subjects completed their performance testing on the same day, at a similar time of day and under similar environmental conditions to avoid any diurnal variation. Subjects were instructed to maintain their normal diet and hydration pattern, refrain from consuming any alcohol, sports drinks or ergogenic aids and avoid strenuous exercise – defined as exercise requiring great exertion, such as a match or strenuous training session – for the 48 hours prior to each test. Subjects were familiarised with the testing procedure and protocols prior to each test. The performance test was performed in an indoor sports hall with an air temperature of 16-20ºC. All performance tests were measured using electronic

timing gates (Fusion Sport Smartspeed Timing Gates, Australia and BrowerXS, Vermount, USA).

4.2.11. Statistical Analysis

All statistical analyses were conducted using SPSS for Windows v. 16.0 (SPSS, USA). Paired sample t-tests were used to identify differences between the two conditions for all parameters. Linear regression of performance variables with mean GI symptoms for each condition was also performed in order to detect any relationship between performance and GI symptoms. A p value less than 0.05 was considered statistically significant.

4.3. Results

4.3.1. Subject Characteristics

Descriptive and anthropometric data for the 10 subjects are presented in Table 4.1.

Table 4.1: Descriptive and Anthropometric Data (n=10)

Variable	**Mean (± SD)**
Body mass (kg)	85.6 ± 17.6
Height (cm)	180.9 ± 7.5
Body mass index (kg/m^2)	25.9 ± 3.6
Age (yrs)	25 ± 3

4.3.2. Standard Bicarbonate ($StdHCO_3^-$)

No significant differences were observed between SB and PLA trials at any time point throughout the testing process (*see Figure 4.4*). Mean pre-warm up $StdHCO_3^-$ levels were 26.4±1.4 mmol/L for SB supplementation and 26.1±0.85 mmol/L for PLA supplementation, respectively (p=0.882). Post-test $StdHCO_3^-$ was recorded as 16.0±6.9mmol/L vs. 13.0±3.2mmol/L for SB and PLA supplementation, respectively (p=0.455).

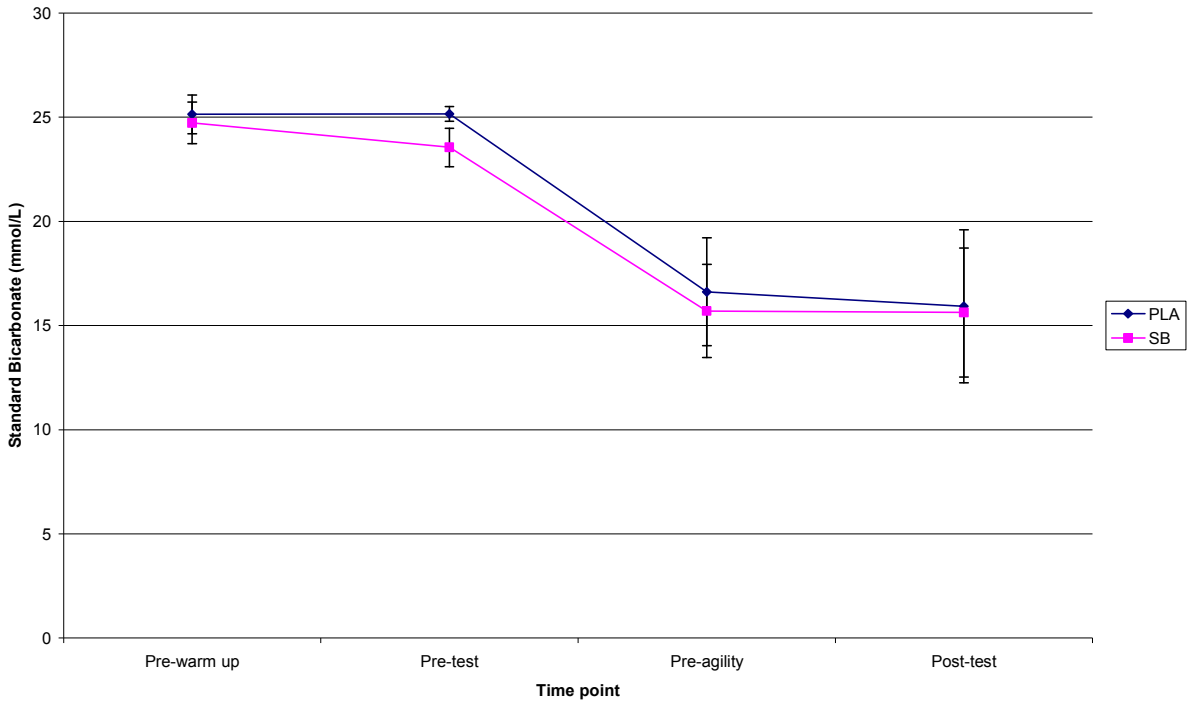


Figure 4.4: Standard bicarbonate concentrations across time points for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

4.3.3. Blood pH

Baseline blood pH was measured prior to the chronic 5 day supplementation protocols and was identified as 7.40 ±0.01 pH units and, therefore, was within normal resting levels. No significant differences were found between SB and PLA trials at any time point ($p>0.05$) throughout the testing process (*see Figure 4.5*). Mean pre-warm up pH was recorded as 7.38 ± 0.02 for SB supplementation and 7.39 ± 0.02 for PLA supplementation, respectively ($p>0.05$). Post-test blood pH was recorded as 7.24 ± 0.10 vs. 7.26 ± 0.07 for SB and PLA supplementation, respectively ($p>0.05$).

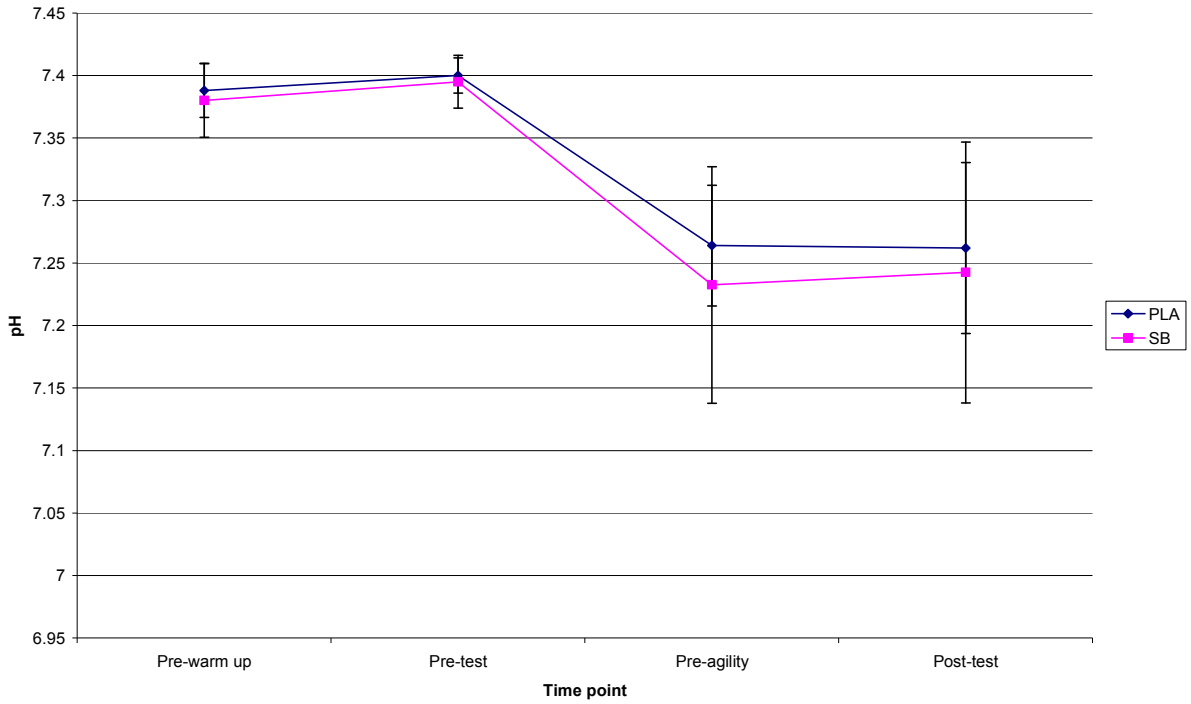


Figure 4.5: Comparison of pH levels across time points for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

4.3.4. Base Excess in Extracellular Fluid (BE-Ecf)

BE-Ecf was measured using blood gas analysis prior to the chronic loading period and then at four time points on the day of the performance trial – pre-warm up, pre-test, pre-agility test and post-test. No significant differences in BE-Ecf were detected between chronic SB and PLA supplementation trials at any of these time points (*see Figure 4.6*). BE-Ecf values of 1.5 ± 1.1 vs. 1.8 ± 0.7 mmol/L were identified at the pre-warm up stage for SB and PLA trials, respectively ($p>0.05$). Post-test BE-Ecf also exhibited no significant difference between SB and PLA supplementation protocols (-11.4 ± 4.0 vs. -11.0 ± 4.9 mmol/L, respectively ($p>0.05$).

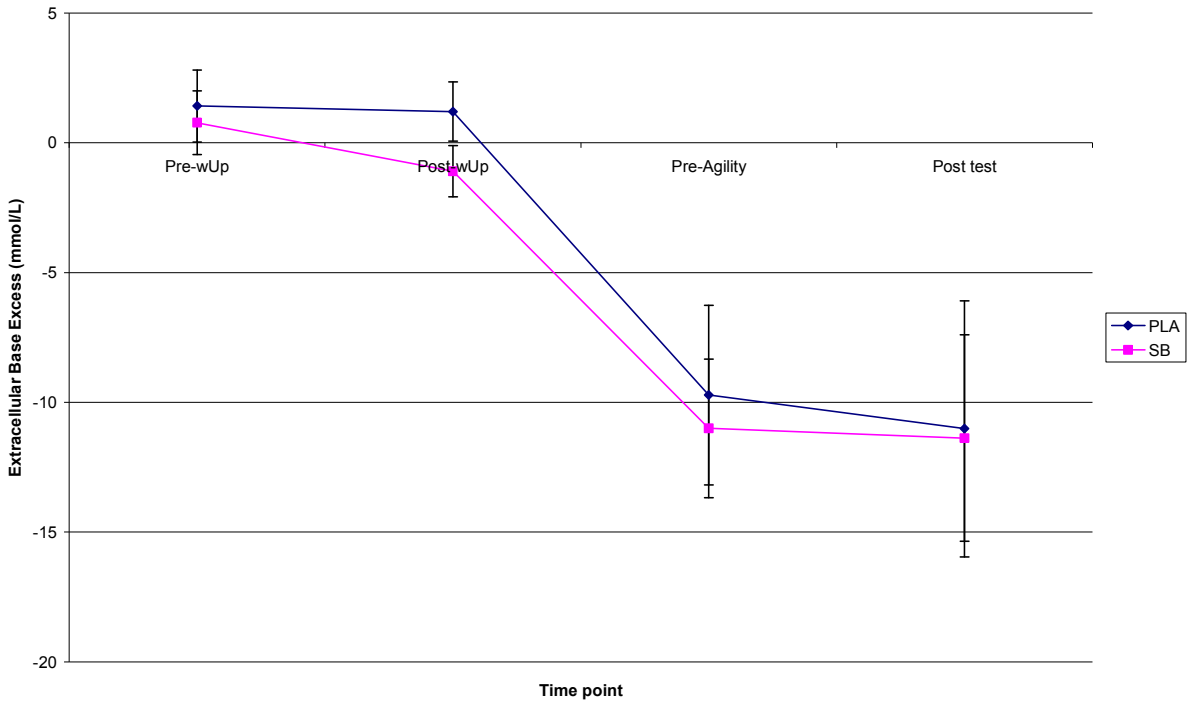


Figure 4.6: BE-Ecf across time points for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

4.3.5. Blood Lactate Analysis

Baseline lactate levels were identified as lying within the normal ranges prior to supplementation for all subjects (0.8-1.8 mmol/L) with no significant differences between the groups (p = 1.000). No significant differences in blood lactate concentration (*see Figure 4.7*) were observed between the SB and PLA chronic supplementation protocols at any of the four time points measured throughout the performance test (p>0.05 for all time points) – pre-warm up (SB 0.97 ± 0.14 vs. PLA 0.98 ± 0.18 mmol/L), pre-test (SB 1.50 ± 0.39 vs. PLA 1.18 ± 0.21 mmol/L), pre-agility test (SB 8.66 ± 2.78 vs. PLA 8.76 ± 3.25 mmol/L), post-test (SB 10.01 ± 3.51 vs. PLA 9.22 ± 3.47 mmol/L).

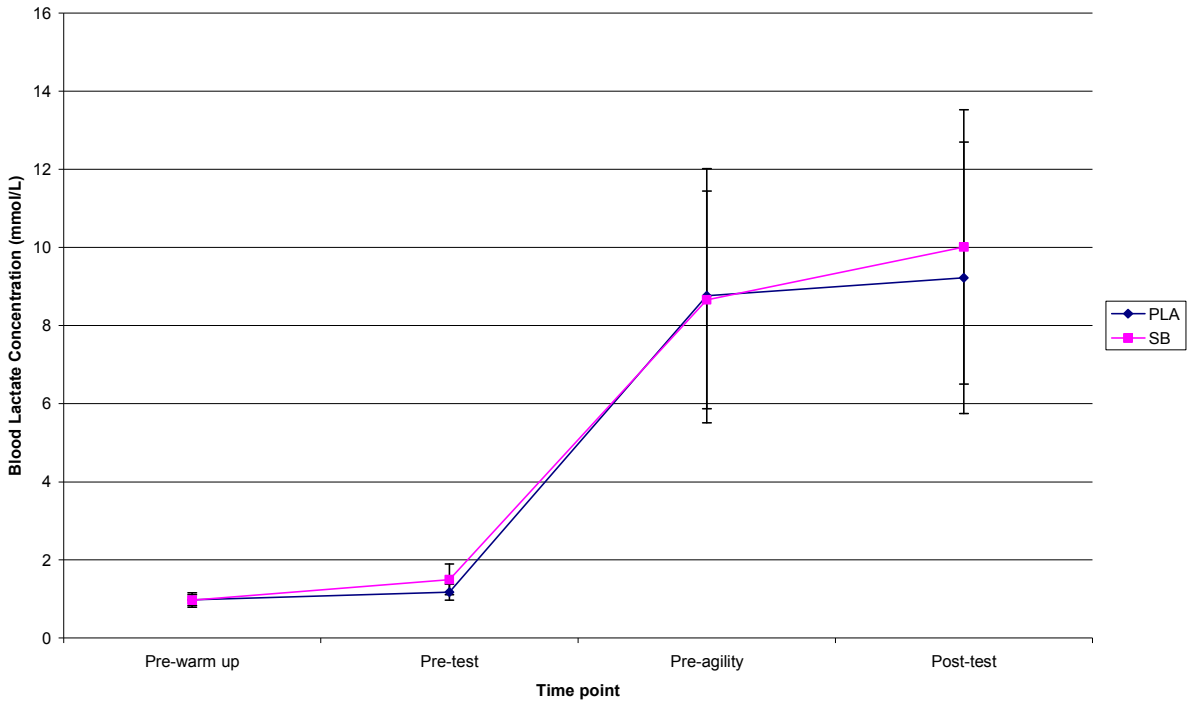


Figure 4.7: Comparison of blood lactate concentrations across time points for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

4.3.6. Partial Pressure of CO_2 (PCO_2)

No significant differences between supplementation protocols were observed at any time point for PCO_2 (*see Figure 4.8*). Pre-warm up PCO_2 values were identified as 5.57 ± 0.40 vs. 6.03 ± 0.61 kPa for chronic SB and chronic PLA supplementation, respectively (p=0.349). Post-test PCO_2 values were recorded as 4.60 ± 0.26 vs. 4.35 ± 0.82 kPa for chronic SB and chronic PLA supplementation, respectively (p=0.564).

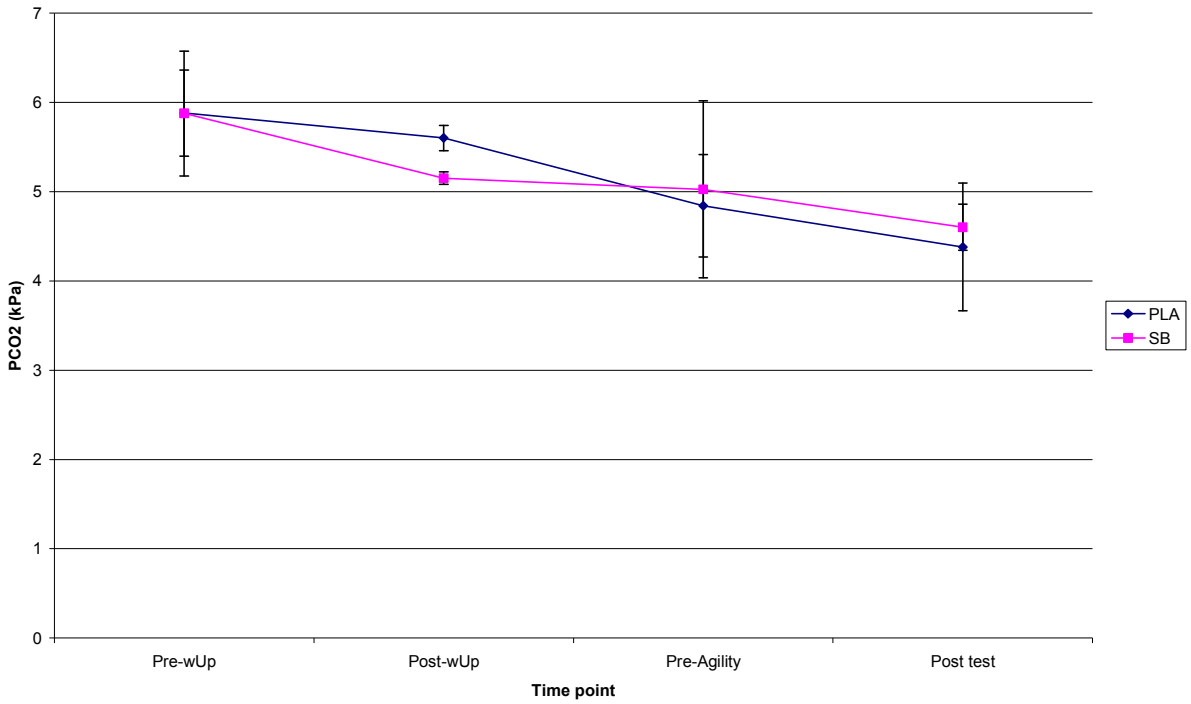


Figure 4.8: PCO_2 (kPa) across time points for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

4.3.7. Partial Pressure of O_2 (PO_2)

Similar to PCO_2, no significant differences in PO_2 were detected at any time point between SB and PLA trials (*see Figure 4.9*). Pre-warm up PO_2 values were identified as 10.00 ± 0.57 vs. 10.55 ± 1.34 kPa for SB and PLA supplementation, respectively (p=0.754). Post-test PO_2 values were recorded as 10.87 ± 1.12 vs. 11.70 ± 0.72 kPa for SB and PLA supplementation, respectively (p=0.452).

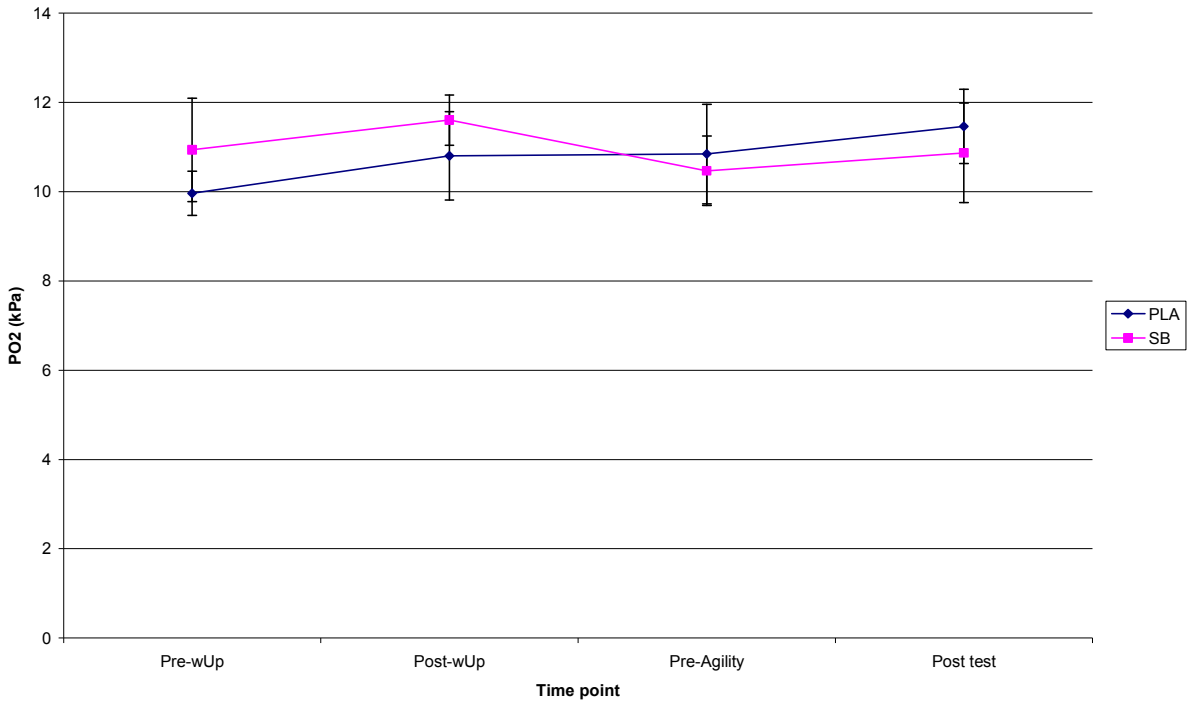


Figure 4.9: PO_2 (kPa) across time points for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

4.3.8. Rugby Specific Performance Test

No differences were detected between supplementation protocols in any of the aforementioned blood parameters analysed (see Sections 4.3.2 – 4.3.7). Similarly, no significant mean differences were observed in the performance test between the SB and PLA trials following the implementation of the chronic supplementation protocols. Five performance measures comprising the rugby sevens-specific circuit protocol were analysed with the group and individual results outlined below. These performance measures included:

1. 2 x 9m tackle bag carry shuttle
2. Reactive agility speed (RAS)
3. 15m sprint
4. Change of direction speed (CODS)
5. Illinois agility test

4.3.9. Tackle Bag Carry Shuttle

No significant group differences were observed for the 2 x 9m tackle bag carry shuttle between the SB and PLA trials (17.09 ± 1.82s vs. 17.01 ± 1.01s for SB and placebo supplementation trials, respectively; p=0.847).

4.3.10. Reactive Agility Speed (RAS)

Group means for all three elements of the RAS (15m sprint time, reaction time and total time) were not found to differ significantly between the SB and PLA supplementation trials. Total time for the RAS was 5.31 ± 0.32s for the SB trial and 5.25 ± 0.26s for the PLA trial (p=0.348).

4.3.11. 15m Sprint

As with the other performance measures, no significant group differences were observed between SB and PLA supplementation trials for 15m sprint times. Chronic SB supplementation produced a mean 15m sprint time of 3.05 ± 0.19s over the six circuits of the performance test, whereas chronic PLA supplementation resulted in a mean time of 3.10 ± 0.22s (p=0.568).

4.3.12. Change of Direction Speed (CODS)

No significant differences were found between SB and PLA trials for any element of CODS performance. The recorded means ± standard deviations were 4.69 ± 0.27s vs 4.72 ± 0.34s for SB and PLA supplementation trials respectively (p=0.847).

4.3.13. Illinois Agility Test

The Illinois agility test was performed following a two minute rest period subsequent to the initial six circuits of the rugby sevens-specific test protocol. Mean sprint times of 16.99 ± 0.92s and 17.19 ± 0.81s were recorded for SB and PLA supplementation trials respectively, which were not found to be statistically significant from each other (p=0.728).

4.3.14. Individual Performance Data

Individual subject performances comparing the two trials were also investigated. From this analysis, it was determined that, out of the ten subjects tested, two subjects (20%) demonstrated enhanced performance with SB supplementation across the six circuits of the performance test when compared with the PLA trial. Furthermore, two subjects (20%) displayed no significant differences between the two trials. One subject (10%) showed improved performance with both SB and PLA supplementation in two different tasks of the performance test. The remaining five subjects (50%) recorded enhanced performance with PLA supplementation when compared to the SB trial. These individual results are outlined in detail as case studies below. Means and standard deviations presented are based on analysis of all six circuits completed by one individual subject.

Subject 1 demonstrated lower sprint times with SB supplementation when compared with the PLA trial in several performance parameters measured. In relation to the 2 x 9m tackle bag carry shuttle, Subject 1 recorded an enhanced performance with SB when compared to PLA supplementation (15.87 ± 0.17 vs. 17.18 ± 0.33s, respectively). 15m sprint time also demonstrated a difference between the two trials for the same subject (2.78 ± 0.13 vs. 3.42 ± 0.16s for SB and PLA supplementation, respectively). With regard to the Illinois agility test, the SB protocol elicited a faster mean time across the six shuttles for Subject 1 when compared to the PLA trial (SB 16.08 ± 0.13 vs. PLA 18.9 ±

0.46s). In addition, Subject 1 recorded a lower CODS_15m sprint time with SB when compared to PLA supplementation (3.03 ± 0.14 vs. 3.29 ± 0.66s, respectively).

Subject 2 exhibited a similar response to Subject 1 with a greater performance in the SB trial for the tackle bag carry shuttle when compared to PLA (16.12 ± 0.43 vs. 17.67 ± 0.65s, respectively). CODS_total was also found to be faster with SB rather than PLA supplementation (4.62 ± 0.31 vs. 4.96 ± 0.49s, respectively).

Subject 8 appeared to have responded comparably with Subject 1 and 2 to the SB supplementation protocol, recording a lower CODS_reaction speed than in the PLA trial (1.24 ± 0.03 vs. 1.28 ± 0.06s, respectively). However, conversely, this subject also demonstrated enhanced performance across the six circuits of the Illinois agility shuttle test with the PLA as opposed to SB supplementation (SB 16.54 ± 0.34 vs. PLA 16.17 ± 0.26s).

Subjects 4, 6, 7, 9 and 10 all displayed evidence of enhanced performance with PLA supplementation as opposed to the SB protocol. In Subject 4, differences between PLA and SB ingestion were observed for tackle bag carry shuttle speed (SB 17.38 ± 1.04s vs. PLA 16.39 ± 0.39), 15m sprint time (SB 3.25 ± 0.21s vs. PLA 2.84 ± 0.16), the Illinois agility shuttle (SB 18.45 ± 0.07s vs. PLA 17.39 ± 0.33s) and CODS_reaction (SB 1.42 ± 0.09s vs. PLA 1.26 ± 0.03s). Subject 6 also recorded reduced Illinois agility shuttle times with the PLA (17.72 ± 0.32s) trial when compared to the SB trial (18.44 ± 0.12s). Faster RAS_reaction times were expressed in Subject 7 with PLA supplementation when evaluated against the SB trial (SB 1.75 ± 0.08s vs. PLA 1.58 ± 0.04s). In Subject 9, tackle bag carry shuttle (SB 19.35 ± 0.26s vs. PLA 17.84 ± 1.00s), RAS_15m (SB 3.60±0.09s vs. PLA 3.38±0.15s) and RAS_total (SB 5.52±0.18s vs. PLA 5.16±0.17s) were all adjudged to be faster with PLA supplementation in relation to the SB trial. Similarly, Subject 10 demonstrated faster CODS_total times with PLA when compared to SB supplementation (SB 4.86±0.22s vs. PLA 4.62±0.10s). Meanwhile, Subjects 3 and 5 exhibited no differences in performance between the SB and PLA supplementation protocols.

4.3.15. Gastro-Intestinal (GI) Ratings

The difference between the SB and PLA supplementation protocols was not found to be statistically significant for either the mean or the total sum of GI ratings during the entire test overall. Nor was any significant difference found between the two trials at any of the four time points measured during the test (*see Figure 4.10*) – pre-warm up, pre-test, pre-agility and post-test ($p>0.05$). In addition, the relationship between subjects' GI ratings and each element of the performance test was low and not considered to be statistically relevant ($r<0.620$; $r^2<0.385$; $p>0.05$ for all performance variables).

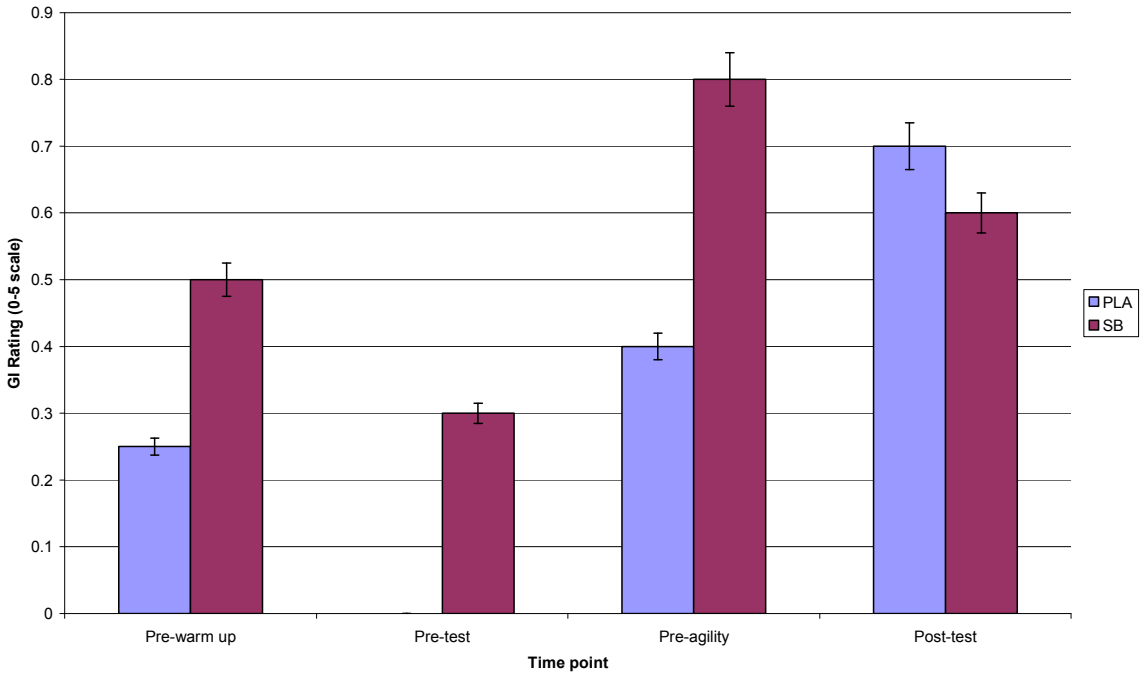


Figure 4.10: Mean GI ratings across each time point for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

4.3.16. Ratings of Muscle soreness

No significant differences in perceived muscle soreness ratings were detected between the two supplementation protocols at any time point throughout the test (*see Figure 4.11*). The greatest muscle soreness ratings were recorded post-test, at which point mean ratings of 6.1 ± 1.8 and 5.1 ± 2.9 on a scale of 0-10 were observed for SB and PLA supplementation trials, respectively (p=0.363).

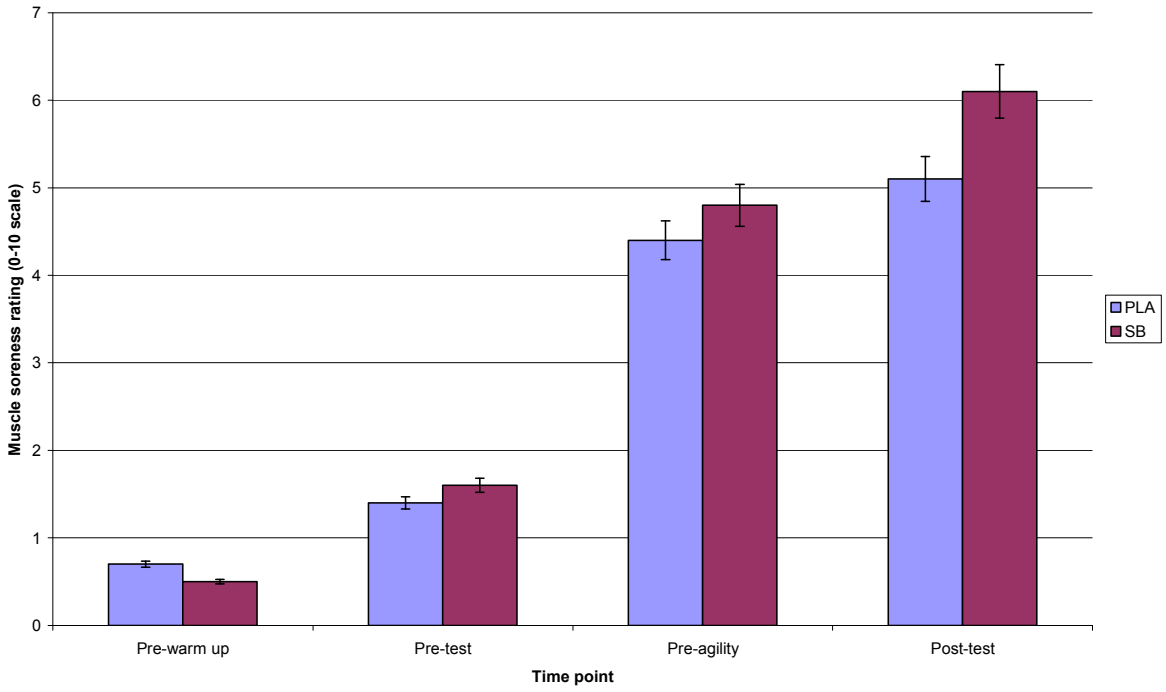


Figure 4.11: Mean muscle soreness ratings across each time point for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

4.3.17. Rating of Perceived Exertion (RPE)

Similarly, RPE exhibited no significant differences between the SB and PLA supplementation trials at any time point (pre-warm up, p=1.000; pre-test, p=0.280; pre-agility, p=0.462; and post-test, p=1.000) at the respective time points pre-test, pre-agility and post-test).

4.3.18. Heart Rate

As illustrated in Figure 4.12, no significant differences in mean (SB 137 ± 12 bpm vs. PLA 143 ± 10 bpm; p=0.204) or maximum (SB 192 ± 12 bpm vs. PLA 187 ± 8 bpm; p=0.181) recorded heart rates were observed between SB and PLA ingestion protocols over the course of the performance test.

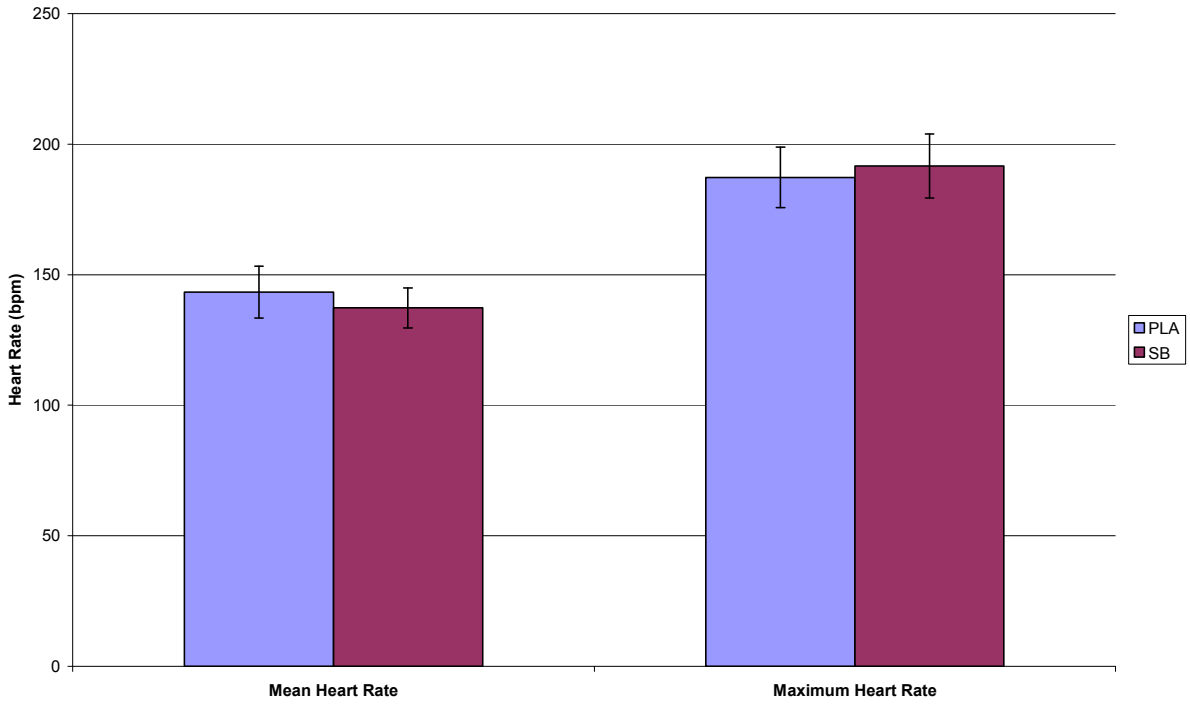


Figure 4.12: Mean and maximum heart rate observed during performance test for each supplementation protocol (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

4.3.19. Summary

To summarise these results, no significant differences in any of the measured blood parameters were observed between chronic SB and PLA supplementation trials. Blood $StdHCO_3^-$, pH, BE-Ecf, lactate, PCO_2 and PO_2 following chronic SB ingestion were found to be no different to the PLA trial at any time point ($p>0.05$). Similarly, no significant differences in performance variables were detected between SB and PLA trials ($p>0.05$). Furthermore, in relation to heart rate data, RPE, ratings of muscle soreness and GI, SB and PLA trials were not found to be significantly distinct from each other. Also, the effect of GI ratings on performance was deemed to be low ($r<0.620$; $r^2<0.385$; $p>0.05$). The large degree of individual variability associated with SB ingestion was evident through the various individual differences in performance. The majority of subjects exhibited enhanced performance with PLA supplementation as opposed to SB. The following section (Section 4.4) will discuss these findings and present the potential underlying principles.

4.4. Discussion

4.4.1. Introduction

The findings of Study 1 revealed that chronic SB ingestion was not found to exhibit a statistically significant difference in any of the performance parameters measured. However, chronic SB ingestion, within Study 1, did show a significantly elevated mean $StdHCO_3^-$ after the first sprint of the repeated bout performance test when compared to the acute PLA condition ($p=0.016$). In addition, at certain points during the Study 1 testing protocol, the difference between chronic SB ingestion and acute PLA relating to BE-Ecf was tending towards significance (immediately prior to the performance test $p=0.083$; and following Sprint 1 $p=0.169$). Furthermore, it has been widely documented that there is a high degree of individual variability associated with SB ingestion. This is believed to be due to adverse GI symptoms which may be experienced acutely following ingestion (Requena et al., 2005). Chronic SB ingestion has been purported to attenuate these symptoms (McNaughton and Thompson, 2001). In addition to this, there is an absence of scientific research concerning the effects of chronic SB ingestion on repeated bout high intensity performance. As a result, Study 2 sought to examine the effects of chronic SB supplementation on repeated bout high intensity intermittent activity.

There are several other novel aspects to this study. In addition to the unique focus on the effects of chronic SB supplementation on repeated bout high intensity performance, this study incorporated a field-based test protocol which was designed to specifically replicate the physiological demands associated with a typical rugby union sevens match (Suarez-Arrones et al., 2011). To the author's knowledge, no other study has used such a test protocol. In addition, current research relating to rugby sevens has, thus far, been limited to analysis of either the incidence of injuries or the physiological demands of the sport (Higham et al., 2012; Suarez-Arrones et al., 2011; Suarez-Arrones et al., 2011; Fuller et al., 2010; Rienzi et al., 1999). Apart from one study examining the effects of Ramadan fasting on rugby sevens performance (Trabelsi et al., 2011), no other research has examined the effect of an intervention, such as SB ingestion, on performance in rugby

sevens. Similar to the findings reported in Study 1, the current study observed no significant differences in performance or blood parameters between chronic SB and PLA ingestion. The results of the current study would suggest that chronic SB supplementation has no ergogenic benefit in the performance of high intensity intermittent exercise indicative of the movement patterns and physiological demands of team field sports, in particular rugby sevens.

4.4.2. Effects of Chronic SB Supplementation on Simulated Sports Performance

A limited number of studies have examined the effect of chronic SB supplementation on simulated sports performance (Driller et al., 2012; Carr et al., 2012; Joyce et al., 2012). However, each of these three studies involve sport-specific performance in individual single bout events including 4-minute cycling time trial performance (Driller et al., 2012), 2000m rowing performance (Carr et al., 2012) and 200m swim performance (Joyce et al., 2012). Carr et al. (2012) and Driller et al. (2012) published similar findings to the current study in relation to acid-base variables with chronic SB ingestion, demonstrating no significant increases in pH or $StdHCO_3^-$. Despite this, however, Driller et al. (2012) reported a significantly higher mean power output in a 4-minute time trial cycle ergometer test with chronic SB supplementation when compared to the PLA trial (427.5 ± 43.6 W and 418.3 ± 41.5 W, respectively; p=0.01). Evidence supporting the ergogenic effect of SB supplementation is generally accompanied by elevated $StdHCO_3^-$]and an increase in extracellular pH (McNaughton et al., 2008). Given that a significant performance enhancement was observed in the absence of pre-exercise alkalosis, Driller et al. (2012) proposed a potential alternative mechanism but failed to elaborate. However, within the aforementioned investigation (Driller et al., 2012), there was a trend towards an increased buffering capacity, as measured by blood $StdHCO_3^-$, with chronic SB ingestion when compared to the PLA trial (p=0.09). Therefore, it is, perhaps, more likely that the sample size (n=7) was insufficient to exhibit statistical significance in acid-base variables (Driller et al., 2012).

In the context of the current study, although the sample size was slightly higher (n=10) than Driller et al. (2012), this may provide some explanation for the lack of significant findings in blood or performance parameters for chronic SB ingestion in relation to the PLA trial. Previous research has also cited training status as a potential inhibiting factor to the ergogenic potential of SB ingestion due to an already enhanced buffering capacity (Peart et al., 2012). In contrast to the findings of the present study and previous research (Carr et al., 2012; Driller et al., 2012), Joyce et al. (2012) demonstrated a significant increase in plasma $StdHCO_3^-$, pH and BE-Ecf ($p<0.05$) following four days of chronic SB ingestion. However, this pre-exercise alkalosis did not translate into enhanced 200m swim performance (chronic SB 1:58.53 ± 0:05.64 vs. PLA 1:59.02 ± 0:05.82 mm:ss.00; $p>0.05$). Joyce et al. (2012) proposed that the training status of the athletes may have accounted for the inconsistent findings. As mentioned in Study 1, a meta-analysis by Peart and colleagues (2012) highlighted the potential effect of training status on the efficacy of SB supplementation. Ordinarily, throughout the current literature, studies involving trained subjects are statistically less likely to detect an ergogenic benefit with SB ingestion than those studies that have recruited untrained individuals (Peart et al., 2012; Carr et al., 2011). Within the review by Peart et al. (2012), trained subjects were classified as athletes following a training plan relevant to their respective exercise task. In contrast, untrained participants made reference to healthy, recreationally active participants. In light of the high training status subjects involved in the current study, this may provide a potential explanation to the failure of the present study to demonstrate improved performance with chronic SB ingestion. Another possible justification for these results may be that this protocol of chronic SB ingestion was perhaps not conducive to causing a sufficient pre-exercise perturbation in acid-base balance. However, this SB dosage of 0.3g/kg BM per day had demonstrated a significant level of pre-exercise alkalosis in previous research (Joyce et al., 2012).

Ingestion of SB chronically, in three or four multiple SB doses of 0.1g/kg BM throughout the day over 3-5 consecutive days prior to performance, has previously been reported to offset the gastro-intestinal side effects associated with SB ingestion (Burke et al., 2007; McNaughton and Thompson, 2001). This is corroborated by the current study, which

displayed no significant differences in GI disturbance between SB and PLA ingestion trials. RPE and muscle soreness were also no different between chronic SB ingestion and the PLA trial. Although no ergogenic benefits were recorded, chronic SB ingestion was not found to be detrimental to performance, as performance was maintained. This may represent a point of interest for athletes considering SB supplementation prior to performance. As previously mentioned in Study 1, it is widely acknowledged that there is a high degree of inter-individual variability and GI distress associated with acute SB ingestion (Requena et al., 2005; Carr et al., 2011). Therefore, chronic SB ingestion may provide a low-risk approach for athletes to experiment with this supplement.

4.4.3. Chronic Supplementation Protocol

Carr et al. (2012) employed a SB supplementation protocol of 0.5g/kg BM per day for a period of 3 days, resulting in no significant effect on induced alkalosis ($StdHCO_3^-$ values: chronic SB 28.0 ± 2.8 mmol/L vs. PLA 27.2 ± 2.5 mmol/L; $p>0.05$) or 2000m rowing performance (chronic SB 282 ± 65 W vs. PLA 277 ± 60 W; $p>0.05$) in comparison to the PLA trial. In contrast to Carr et al. (2012), McNaughton and Thompson (2001) dispensed an identical dose (0.5g/kg BM per day) but for a longer loading duration of 6 days and found significant pre-exercise alkalosis and performance enhancement in a single bout of 90 second maximal cycling. In addition, Driller et al. (2012) administered a slightly lower chronic dose of 0.4g/kg BM per day for a period of 3 days, leading to no significant difference in acid-base balance but a significant improvement in 4 minute cycling time trial performance. Furthermore, Joyce et al. (2012) administered a dose of 0.3g/kg BM per day for a period of 4 days inducing a significant level of alkalosis but no improvement in 200m swim performance. The aforementioned research highlights the ambiguity surrounding chronic SB ingestion timing and dosage protocols and their respective outcomes in terms of performance.

A chronic SB ingestion dose of 0.3g/kg BM per day, as implemented in the current study, has been shown to induce significant metabolic alkalosis, while minimising the potential

GI side effects (Joyce et al., 2012). The materialisation of GI symptoms often associated with higher doses (Peart et al., 2012) were deemed to be impractical for players in the 5 days preceding a team field sport event , such as a rugby sevens tournament. The 5 day loading period selected was adjudged to be a realistic time frame for the loading period for team field sport athletes between tournament/match performances. As a result, this SB loading protocol of 0.3g/kg BM per day for 5 days functioned to most closely represent the preparation period prior to competitive team field sport performance. In accordance with previous studies (Joyce et al., 2012; McNaughton et al., 1992; Siegler et al., 2010), the dosage adopted within the current study has been shown to induce pre-exercise alkalosis, thereby providing the potential for an ergogenic effect on performance. However, this proved not to be the case in this study as no significant differences in blood $StdHCO_3^-$, pH, BE-Ecf or lactate were observed throughout the testing process between chronic SB and PLA trials.

4.4.4. Summary

Similar to Study 1, this research further highlights the contradictory nature of the area of SB supplementation. To date, few studies have investigated optimal SB loading strategies in terms of timing and dosage and the varied ingestion protocols utilised across different studies appear to contribute to the inconsistent results. The current study reported no significant shift in acid-base balance following chronic SB ingestion consisting of 5 days loading with 0.3g/kg BM per day. According to previous research (Peart et al., 2012; Carr et al., 2011; Matson and Tran, 1993), an enhancement of the body's buffering capacity is the major mechanism by which SB ingestion may contribute to improved performance. In light of this, a performance enhancement in the absence of an alkaline environment within the blood would seem improbable. Although manifestation of pre-exercise alkalosis does not guarantee performance enhancement, a chronic SB ingestion protocol involving a higher daily dose may elicit a significant alkalotic response within the blood (McNaughton et al., 2001), thus enabling scientific examination of the proposed physiological mechanism. Several other factors such as training status, severity of GI

discomfort and specificity and nature of the performance test may also impact on the efficacy of chronic SB ingestion (Joyce et al., 2012; Peart et al., 2012). In addition, individual variability may play a key role in the outcome of studies involving SB ingestion. Although these other factors may have played a role, this study provides no evidence of any scientific basis for the use of this chronic SB supplementation protocol, involving the specific dosage and duration utilised, to enhance rugby sevens performance. Hence, the hypothesis that chronic SB supplementation would induce metabolic alkalosis and improve performance has been disproved.

Chapter 5

Study Three: The Effects of Acute Sodium Bicarbonate Supplementation on High-Intensity Intermittent Performance using a Simulated 80 minute Rugby-Specific Test Protocol in Elite Female and Sub-Elite Male Rugby Players

5.1. Introduction

The aim of this study was to examine the effects of acute SB supplementation versus a PLA on high-intensity intermittent performance using a specifically designed test protocol developed to simulate typical demands of 80-minute rugby union match. The hypothesis involved predicted that acute SB supplementation would cause a significant increase in $StdHCO_3^-$, pH and BE-Ecf and result in enhanced high intensity intermittent performance, recovery during the 10 minute half time interval and subsequent performance when compared to a PLA trial. Following Study 2, it was evident that chronic SB supplementation appeared to show no effect on either blood or performance parameters. As a result, Study 3 returned to focus on acute SB supplementation, this time in relation to a field-based, sport-specific 15-a-side rugby test protocol. The study design, rationale and results are discussed within this chapter.

5.2. Methods

5.2.1. Subjects

10 healthy male subjects and 10 healthy female subjects, aged between 18-35 years volunteered to participate in the study. Male subjects consisted of sub-elite trained club rugby players of Junior 2 level or above and were recruited from University and Leinster rugby clubs. Playing experience of each player was no less than 5 years. Female subjects consisted of elite trained rugby players currently involved at either International or Provincial level. Subjects were excluded from the study if they were suffering from any cardiovascular, pulmonary or metabolic diseases; if they were smokers; or if they had any injury or illness that may have affected their performance, as determined by a general health questionnaire (*see Appendix B*) completed by each subject prior to testing. Prior to participation in the study, each subject also provided written informed consent (*see Appendix A3*) Subjects were also excluded if they had used supplements or substances in

the previous six months that may have interfered with the results. All experimental procedures were approved by the Dublin City University Research Ethics Committee.

5.2.2. Anthropometric Measurements

Baseline anthropometric measurements were recorded prior to commencement of any physiological testing. Body mass (kg) was assessed barefoot and in minimal clothing using a portable digital scales and stadiometer (Seca, Germany). Data was reported to the nearest 0.1 kg. Standing height (cm) was measured to the nearest 0.1 cm using the same device. Body mass index (kg/m^2) was calculated by dividing each individual's body mass (kg) by the square of their height (cm).

5.2.3. Overview of Experimental Design

The study took place in the Exercise Physiology Research Hall (EPRH) in DCU. The study used a randomised, double-blind, cross-over design in which subjects performed an 80-minute simulated rugby performance test on two separate occasions. Following a habituation session, each subject completed two experimental trials ingesting an acute pre-exercise dose of either 0.3g/kg BM of either SB or a PLA (maltodextrin). As with Study 1 and 2, every effort was made to standardise the pre-test procedures to ensure that any changes in performance could be attributed to the experimental protocol alone. Subjects were tested at the same time of day for both trials. Subjects were also requested to maintain normal dietary and hydration patterns for the 48 hours prior to Trial 1 and to replicate that for Trial 2. Alcohol, caffeine and strenuous exercise were not permitted in the 48 hours prior to the performance test. Within the habituation session, subjects were given a verbal and physical demonstration before three sub-maximal practice sets of the full performance test (described in Table 5.1). Subjects then completed 40 minutes (one half of a simulated match) of the 80 minute protocol at maximum effort. Subjects were

deemed to be habituated to the protocol as they exhibited no further improvements in performance towards the latter stages of the performance test.

5.2.4. Supplementation Protocol

Subjects were required to fast for 3 hours prior to each performance test. Supplementation consisted of acutely ingesting either PLA (maltodextrin) or SB (0.3g/kg BM) subdivided into three doses of 0.1g/kg BM taken at 90, 60 and 30 minutes prior to the performance test. Therefore, subjects ingested the 0.1g SB/kg BM with 200mls water taken with each of the three doses (0.3g/kg BM in total) every 30 minutes for 90 minutes before commencement of the performance trial.

5.2.5. Performance Test

Immediately prior to testing, the subjects performed a standardised 10 minute standardised warm-up (*see Section 4.2.5 and Illustration 4.1*), which consisted of jogging at a self-selected pace, dynamic stretching, along with exercises involving explosive leg movements. The rugby match simulation test was completed in two 40 minute halves with a 10 minute half time period in between. The test consisted of 14 circuits of 11 stations (*see Table 5.1*) involving the agility, speed, power and accuracy performance-related activities designed to replicate the demands and specific movements involved in a rugby match situation. Players had 30 seconds to complete each station within the circuit. Once the task was completed at each station the player had the remainder of the 30 seconds to rest and move to the next station. The test also included the passive and active recovery periods of standing and walking that comprise a large portion of games according to GPS match analysis (Cunniffe et al., 2009; Roberts et al., 2008; Deutsch et al., 1998). The 14 circuits were divided into two 40 minute halves with 7 circuits in the first half and 7 circuits in the second half, separated by a 10 minute half time period.

There was a 2 minute rest period after the fourth circuit of each half - circuit 4 in the first half and circuit 11 in the second half.

All performance tasks were timed or quantified in an appropriate manner for that task (i.e. sprints were timed, jump height was measured, accurate passes were counted). The walking pace at stations 3, 5, 7 and 11 was set at 1.4 m/s in accordance with the pace observed for the action defined as walking in literature analysing match demands (Duthie et al., 2005, Roberts et al., 2008). The remaining stations consisted of the following activites:

- Station 1: 20m sprint
  - Subjects performed a single, maximum effort 20m sprint.
- Station 2: Reactive agility sprint (*see Step 3 in Section 4.2.5 and Figure 4.2*)
- Station 3, 5, 7 and 11: Walk
  - Subjects were instructed to walk for 30 seconds at 1.4m/s.
- Station 4: Power Phase 1 and 2
  - Phase 1: Standard vertical jump test – Subjects performed a vertical jump keeping hands on hips and legs straight in the air.
  - Phase 2: Weighted vertical jump – Subjects performed a vertical jump with a 10kg weight in each hand keeping arms and legs straight in the air.
- Station 6: Modified T-test
  - Cones were placed in a "T" shape 5m away from each other. Subjects began lying face down at the start line (bottom of the "T"). Subjects sprinted 5m to the cone straight in front of them, then side shuffled 5m to the left cone and then 10m past the middle cone to the right cone, back to the middle cone and backwards to return to the start line. Subjects also completed a "down-up" at every cone, touching their chest off the ground and jumping back up.
- Station 8: Tackle bag carry
  - Subjects were required to complete a shuttle of 4 x 5m sprint carrying a 25kg tackle bag as fast as possible.

- Station 9: Passing accuracy
    - Subjects were required to pass a ball as rapidly as possible at a 1 x 1m target, which was placed 4m from the player and 1.5m above the ground. After each pass the subjects were instructed to pick up the next ball from the ground and repeat. Subjects were permitted 15 seconds to complete five passes.
- Station 10: Agility shuttle
    - Cones were placed at 5m, 10m and 20m away from the start line. Subjects ran continuously out to each cone and back to the start line, performing as many shuttles as possible within 30 seconds.

Table 5.1: Rugby-Specific 80 minute Performance Test Protocol

Circuit Station	Task	Task Description	Performance Measure
1	20m Sprint	20m straight sprint	Time
2	Offensive Sprint	Reactive Agility Sprint (RAS)	Time
3	Walk		
4	Power: Phase 1 Phase 2	Vertical jump Weighted vertical jump	Height Height
5	Walk		
6	Defensive Sprint	Modified T-test	Time
7	Walk		
8	Tackle Sprint	4 x 5m shuttle 25kg tackle bag carry	Time
9	Passing Accuracy	Pass 5 balls at a target	Number of target hits
10	Agility shuttle	70m agility shuttle (5m, 10m and 20m distances)	Time
11	Walk		

Blood samples, muscle soreness, GI disturbance levels and RPE were taken at eight stages throughout each performance trial: pre-warm up, pre-first 40 minute half, after fourth circuit (Circuit 4) in first half, post-first 40 minute half, 5 minutes into half time, pre-second 40 minute half, after fourth circuit (Circuit 11) in second half, and post-second 40 minute half.

5.2.6. Blood Lactate Analysis

Blood lactate sampling technique was identical to the description in Section 3.2.6. Blood lactate concentrations were recorded at the aforementioned blood sampling time points (*See Section 5.2.5*)

5.2.7. Blood Gas Analysis

Blood gas analysis, as previously described in Section 3.2.7 and was also carried out at identical time intervals to the blood gas analysis and subjective ratings.

5.2.8. Heart Rate

Heart rate (HR) was measured throughout each test and was recorded using recordable HR-enabled watches with heart rate monitors (Garmin, Kansas, USA).

5.2.9. Subjective Ratings

A level on the muscle soreness, GI disturbance and RPE scales (see Appendix C) were selected by each subject at the same time points as the blood sampling. The muscle soreness scale was a simple 11 point scale ranging from 0-10, which was used to allow subjects to rate their muscle soreness levels at different points before and after each

aspect of the testing..The RPE scale is the standard Borg scale ranging from 6-20 which is used to gauge the exercise intensity perceived by the subject. The GI disturbance scale was a six point scale ranging from 0-5.

5.2.10. Statistical Analysis

All statistical analyses were conducted using SPSS for Windows v. 16.0 (SPSS, USA). Paired sample t-tests were used to identify differences between the two conditions for all parameters. Linear regression of performance variables with mean GI symptoms for each condition was also performed in order to detect any relationship between performance and GI symptoms. A *p* value less than 0.05 was considered statistically significant.

5.3. Results

5.3.1. Subject Characteristics

The descriptive and anthropometric data for the 10 sub-elite male subjects are presented in Table 5.2.

Table 5.2: Descriptive and Anthropometric Data Males (n=10)

Variable	**Mean (± SD)**
Body mass (kg)	92.6 ± 7.4
Height (cm)	182.3 ± 4.6
Body mass index (kg/m^2)	27.9 ± 2.9
Age (yrs)	23.4 ± 2.6

The descriptive and anthropometric data for the 10 elite female subjects are presented in Table 5.3.

Table 5.3: Descriptive and Anthropometric Data Females (n=10)

Variable	**Mean (± SD)**
Body mass (kg)	70.4 ± 9.6
Height (cm)	168.2 ± 6.4
Body mass index (kg/m^2)	24.9 ± 2.8
Age (yrs)	29 ± 3

5.3.2. Standard Bicarbonate ($StdHCO_3^-$)

5.3.2.1 Sub-Elite Male Data

No significant differences were detected between the two supplementation protocols for baseline levels of $StdHCO_3^-$ (p=1.000). As illustrated in Figure 5.1, $StdHCO_3^-$ were significantly greater with SB supplementation pre-warm up and post-warm up when compared with the PLA protocol. Immediately prior to the warm up, SB supplementation produced a mean $StdHCO_3^-$ of 28.94 ± 3.42 mmol/L as opposed to a mean of 24.30 ± 1.02mmol/L recorded for PLA supplementation (p=0.021). This significantly elevated $StdHCO_3^-$ as a result of SB supplementation was maintained to the post-warm up sampling point ten minutes later, where p=0.028 (27.86 ± 3.79 vs. 23.22 ± 0.87mmol/L for SB and PLA supplementation, respectively). Although the mean $StdHCO_3^-$ visually appeared to be greater for SB than PLA supplementation at every other time point, no other significant differences between the two conditions were found throughout the remainder of the performance test (p=0.063 at post-test time point).

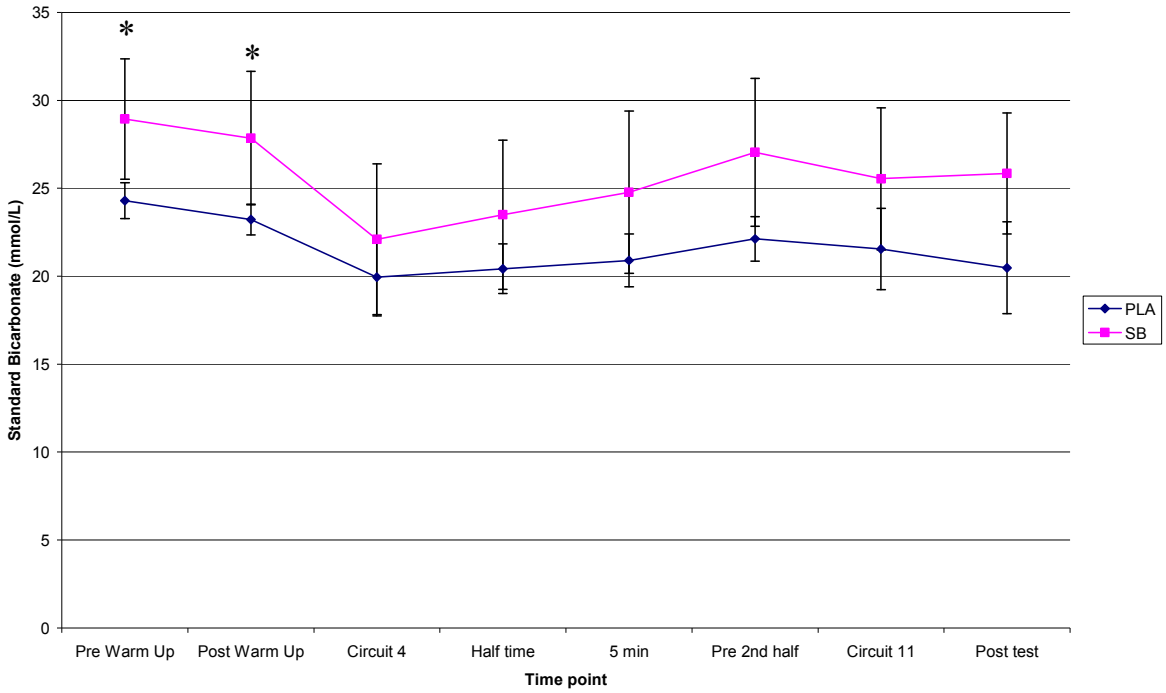


Figure 5.1: Standard bicarbonate levels across time points for each supplementation protocol (males), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.2.2 Elite Female Data

As illustrated in Figure 5.2, $StdHCO_3^-$ levels were significantly greater with acute SB supplementation for all time points post-ingestion with the exception of 5 minutes into the half-time period along with the post-test measurements. The most significant difference between the two supplementation protocols occurred at the pre-warm up stage, at which point recorded $StdHCO_3^-$ consisted of 29.0 ± 1.8mmol/L and 23.2 ± 1.5mmol/L for SB and PLA supplementation, respectively (p=0.001)

This significantly increased $StdHCO_3^-$ observed with SB supplementation when compared to PLA was maintained at the majority of sampling points including circuit 4 (SB 23.0 ± 1.9 vs. PLA 17.5 ± 1.0mmol/L; p=0.016), half-time (SB 22.5 ± 1.3 vs. PLA 17.8 ± 0.9mmol/L; p=0.041), pre-2nd half (SB 26.8 ± 1.7 vs. PLA 19.7 ± 0.5mmol/L;

p=0.011) and circuit 11 (SB 24.9 ± 2.6 vs. PLA 18.4 ± 0.5mmol/L; p=0.043). Although $StdHCO_3^-$ also appeared to remain elevated with SB supplementation at the remaining two blood sampling points (5 minutes into the half-time period and immediately post-test), the difference between the two supplementation protocols was not found to be significant (p=0.054 and p=0.053 for each time point, respectively).

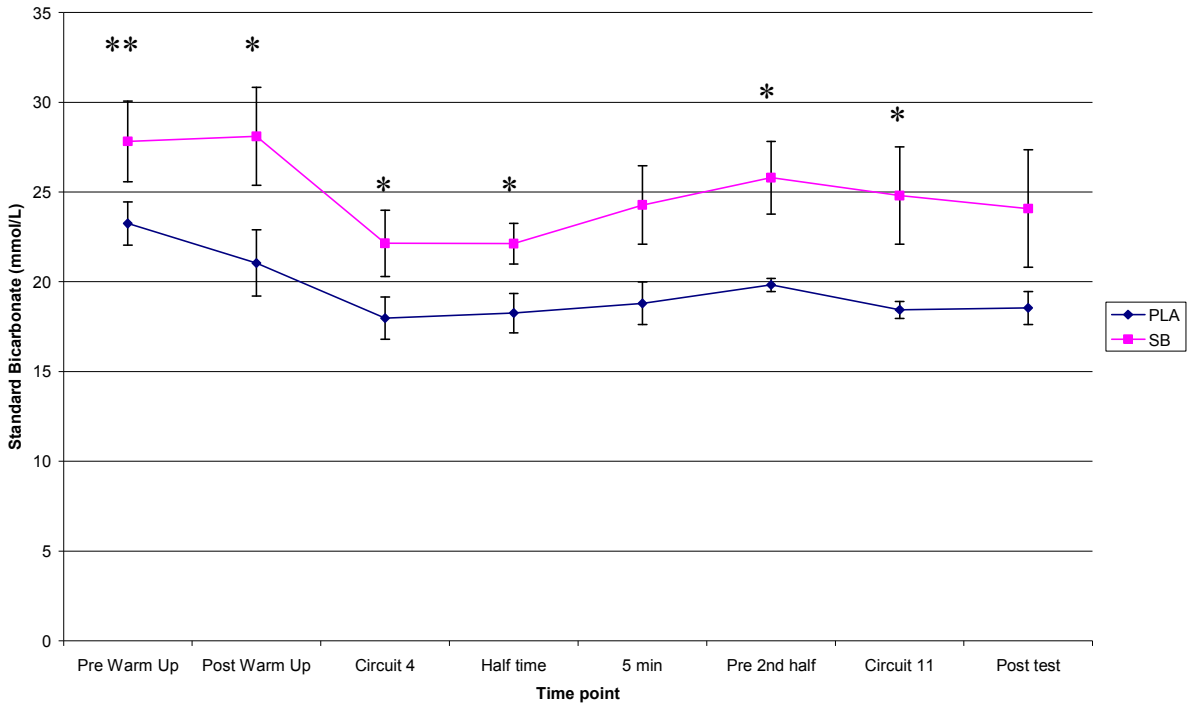


Figure 5.2: Standard bicarbonate levels across time points for each supplementation protocol (females), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.3. Blood pH

5.3.3.1 Sub-Elite Male Data

Baseline blood pH was measured prior to supplementation and was identified as 7.40 ± 0.01 pH units and therefore was within normal resting levels (p=1.000). Statistically

significant differences in pH were found between SB and PLA supplementation protocols at blood sampling time points throughout the 80 minute rugby-specific protocol with the exception of post-circuit 4 and half-time (*see Figure 5.3*). The most significant difference between the two conditions was observed immediately prior to the 2nd half (pre-2nd half time point), where p=0.007 (7.43 ± 0.03 vs. 7.35 ± 0.01 pH units for SB and PLA supplementations, respectively). Comparable significant differences between SB and PLA supplementation protocols were also identified at pre-warm up (7.42 ± 0.03 vs. 7.37 ± 0.01 pH units, respectively; p=0.014), post-warm up (7.42 ± 0.03 vs. 7.37 ± 0.01 pH units, respectively; p=0.017), 5 minutes into half-time (7.40 ± 0.05 vs. 7.33 ± 0.02 pH units, respectively; p=0.030), post-circuit 11 (7.41 ± 0.04 vs. 7.35 ± 0.01 pH units, respectively; p=0.047) and post-test (7.41 ± 0.04 vs. 7.35 ± 0.02 pH units, respectively; p=0.037).

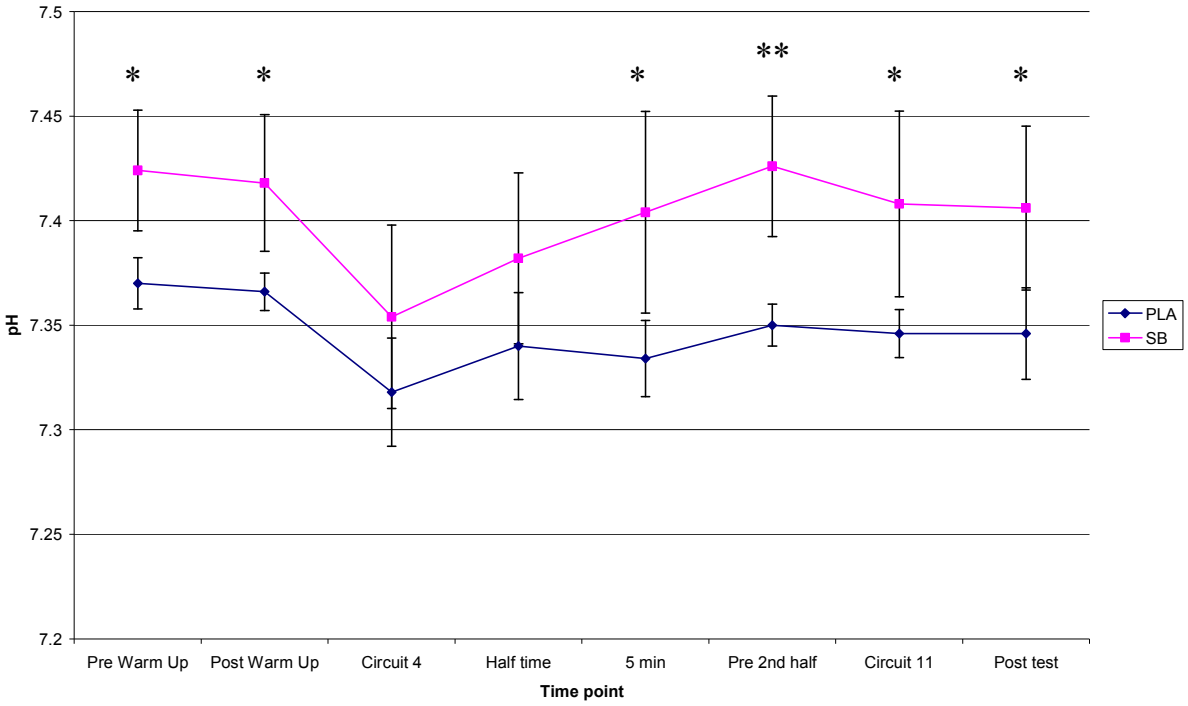


Figure 5.3: Comparison of blood pH levels across time points for each supplementation protocol (males), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.3.2 Elite Female Data

Baseline blood pH was measured prior to supplementation and was identified as 7.40 ± 0.01 pH units and therefore was within normal resting levels (p=1.000). Statistically significant differences in pH were found between SB and PLA supplementation protocols at several time points throughout the 80 minute rugby-specific protocol (*see Figure 5.4*). Immediately prior to the warm up, supplementation with SB elicited a significantly higher mean pH value (7.47 ± 0.03 vs. 7.38 ± 0.02 pH units for SB and PLA, respectively; p=0.046).

Blood pH was also found to be significantly different between the supplementation trials at Circuit 4. At this point, a mean value of 7.41 ± 0.007 pH units was reported for SB supplementation, whereas mean pH at Circuit 4 for the PLA trial was 7.31 ± 0.014 pH units (p=0.033). Following the 10 minute half-time period and immediately prior to the 2nd half of the rugby specific protocol, pH was observed to be significantly different between SB and PLA supplementation (7.46 ± 0.006 vs. 7.36 ± 0.001, respectively; p=0.001). Similarly, at Circuit 11 (7.43 ± 0.03 vs. 7.33 ± 0.01; p=0.026) and also immediately post-test (7.42 ± 0.02 vs. 7.33 ± 0.01; p=0.016) i.e. for the remainder of the sampling time points in the 2nd half, pH was found to be significantly higher with SB when compared to PLA supplementation.

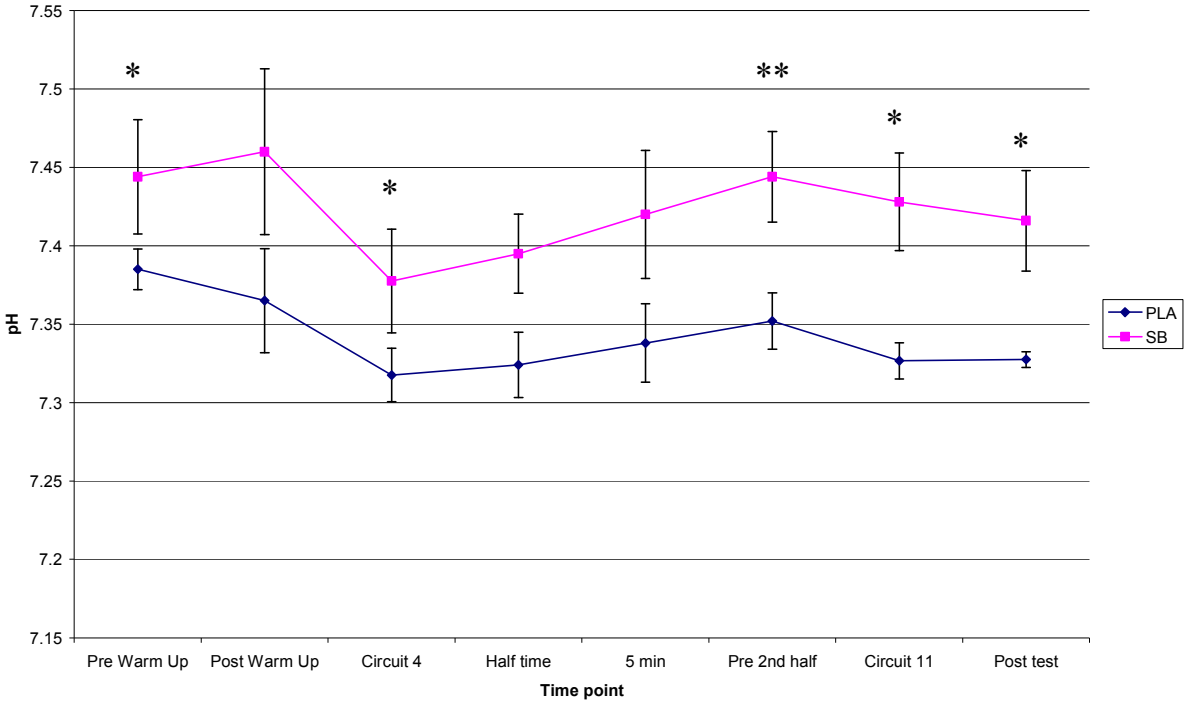


Figure 5.4: Comparison of blood pH levels across time points for each supplementation protocol (females), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.4. Base Excess in Extracellular Fluid (BE-Ecf)

5.3.4.1 Sub-Elite Male Data

No significant differences were detected between the two supplementation protocols for baseline levels of BE-Ecf (p=1.000). Following supplementation and immediately prior to the warm-up for the performance test, SB supplementation demonstrated a significantly higher BE-Ecf when compared with PLA supplementation (5.82 ± 4.12 vs. 0.48 ± 1.55mmol/L for the respective supplementation protocols; p=0.020). SB supplementation appeared to elicit higher levels of BE-Ecf for all remaining blood

sampling time points (*see Figure 5.5*). However, this was not found to be significant at any other time point (p>0.065).

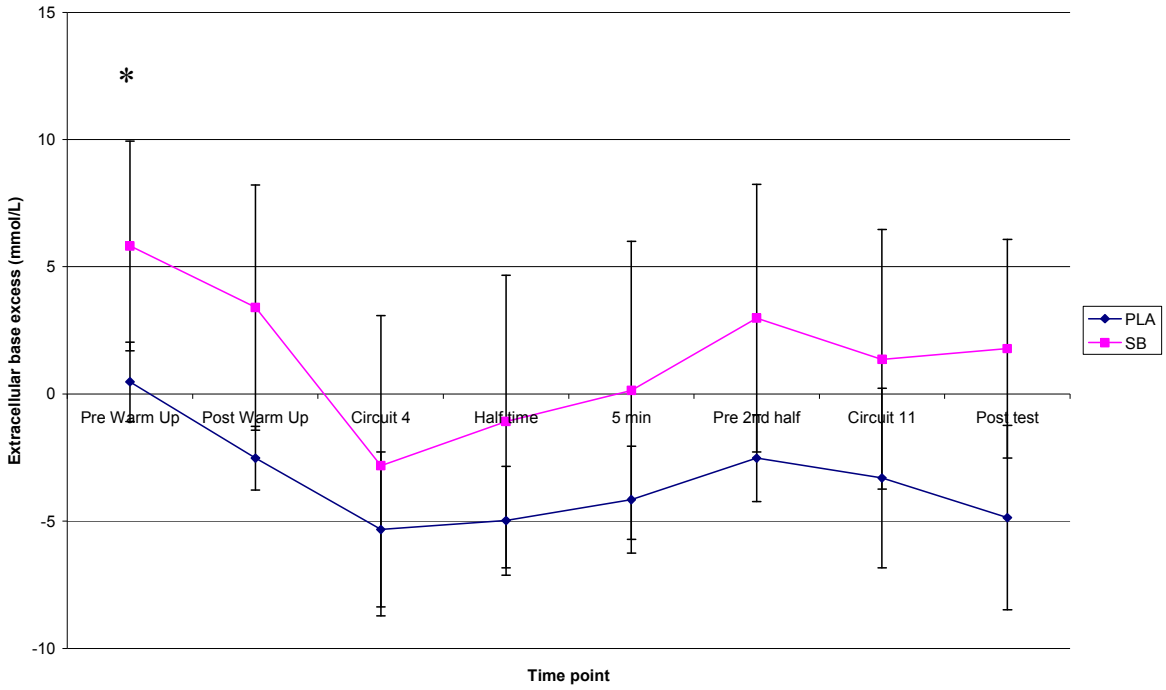


Figure 5.5: BE-Ecf across time points for each supplementation protocol (males), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.4.2 Elite Female Data

No significant differences were detected between the two supplementation protocols for baseline levels of BE-Ecf (p=1.000). Following supplementation and immediately prior to the warm-up for the rugby-specific, high intensity, intermittent test, acute SB supplementation demonstrated a significantly higher BE-Ecf when compared with acute PLA supplementation (5.23 ± 2.10 vs. -1.33 ± 2.34mmol/L for the respective supplementation protocols; p=0.002). SB supplementation also elicited a significantly higher BE-Ecf response relative to PLA ingestion at 5 minutes into half time (1.30 ± 1.56 vs. -7.15 ± 1.06mmol/L, respectively; p=0.026), pre-2nd half (3.65 ± 0.92 vs. -5.70 ±

0.01mmol/L, respectively; p=0.044) and following circuit 11 (0.13 ± 3.30 vs. -7.73 ± 1.04mmol/L, respectively; p=0.040). The relationship between the two supplementation protocols followed a similar pattern at the remaining four time points (*see Figure 5.6*). However, this relationship, while tending towards significance, did not reach a statistically significant difference between SB and PLA supplementation, with the p value ranging from 0.055 to 0.089 for those remaining blood sampling time points during the test.

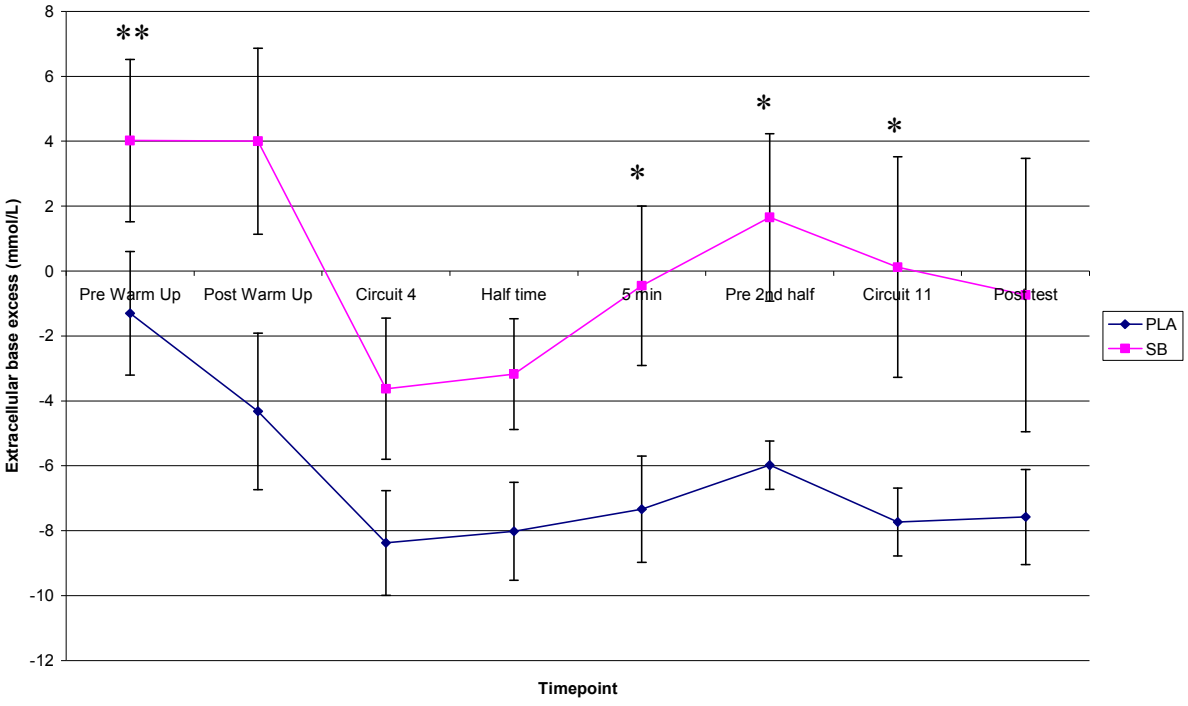


Figure 5.6: BE-Ecf across time points for each supplementation protocol (females), (n=10, values denote mean ± SD)

(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.5. Blood Lactate Analysis

Baseline lactate levels were measured prior to supplementation and were identified as lying within the normal ranges prior to supplementation for all subjects (0.8-1.8mmol/L) with no significant differences between the groups (p = 1.000). Blood sampling was carried out at eight time points throughout the performance test:

1. Pre-warm up
2. Post-warm up
3. Post-circuit 4
4. Half-time
5. 5 minutes into half-time
6. Pre-2nd half
7. Post-circuit 11
8. Post-test

5.3.5.1 Sub-Elite Male Data

Blood lactate concentrations were found to be statistically greater with SB supplementation at several time points when compared to PLA trial (*see Figure 5.7*). These significant differences between SB and PLA trials occurred at post-warm up (1.70 ± 0.67 vs. 1.13 ± 0.31 mmol/L, respectively; p=0.024), half-time (7.20 ± 2.39 vs. 5.51 ± 1.65 mmol/L, respectively; p=0.007), 5 minutes into half-time (6.47 ± 2.24 vs. 4.61 ± 1.57 mmol/L, respectively; p=0.003), post-circuit 11 (6.83 ± 3.01 vs. 4.50 ± 1.73 mmol/L, respectively; p=0.028) and post-test (6.76 ± 2.57 vs. 4.67 ± 1.67 mmol/L, respectively; p=0.006). In addition, although not reaching significance, there was a tendency towards a similar difference between the two trials at pre-warm up (p=0.098) and pre-2nd half (p=0.064).

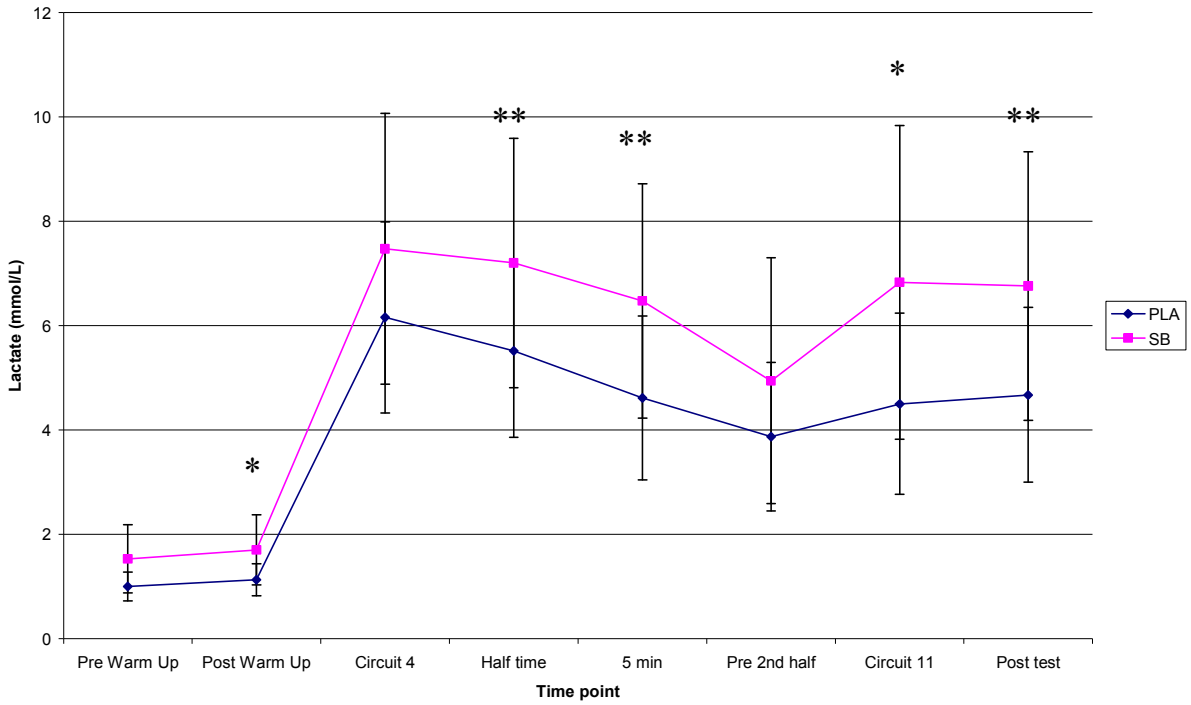


Figure 5.7: Comparison of blood lactate concentrations across time points for each supplementation protocol (males), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.5.2 Elite Female Data

Although blood lactate appeared to tending towards significance with SB supplementation following circuit 4 (p=0.078), no significant differences in blood lactate concentration were observed between the SB and PLA acute supplementation protocols at any of the eight sampling time points (*see Figure 5.8*).

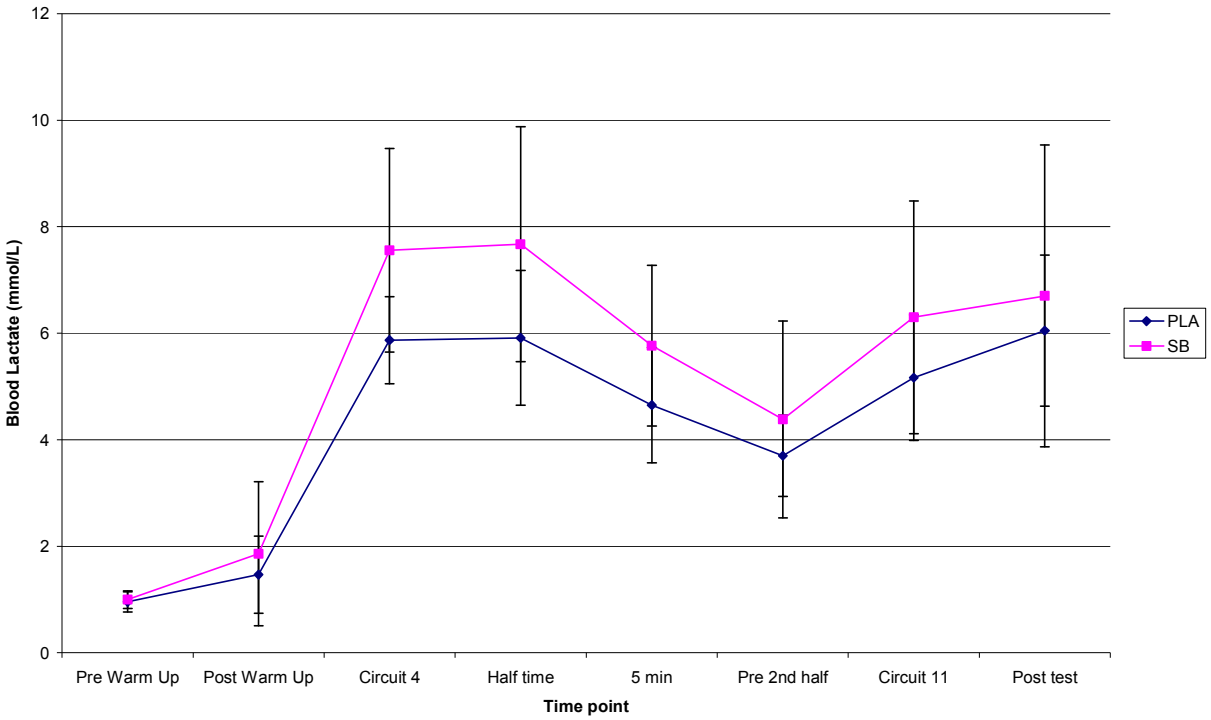


Figure 5.8: Comparison of blood lactate concentrations across time points for each supplementation protocol (females), (n=10, values denote mean ± SD) (*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.6. Partial Pressure of CO_2 (PCO_2)

5.3.6.1 Sub-Elite Male Data

No significant differences between supplementation protocols were observed at any time point for PCO_2 (*see Figure 5.9*). Pre-warm up PCO_2 values were identified as 6.24 ± 0.67 vs. 5.94 ± 0.45 kPa for SB and PLA supplementation, respectively (p=0.284). Post-test PCO_2 values were recorded as 5.64 ± 0.71 vs. 5.06 ± 0.63 kPa for SB and PLA supplementation respectively, displaying a tendency towards significance (p=0.089).

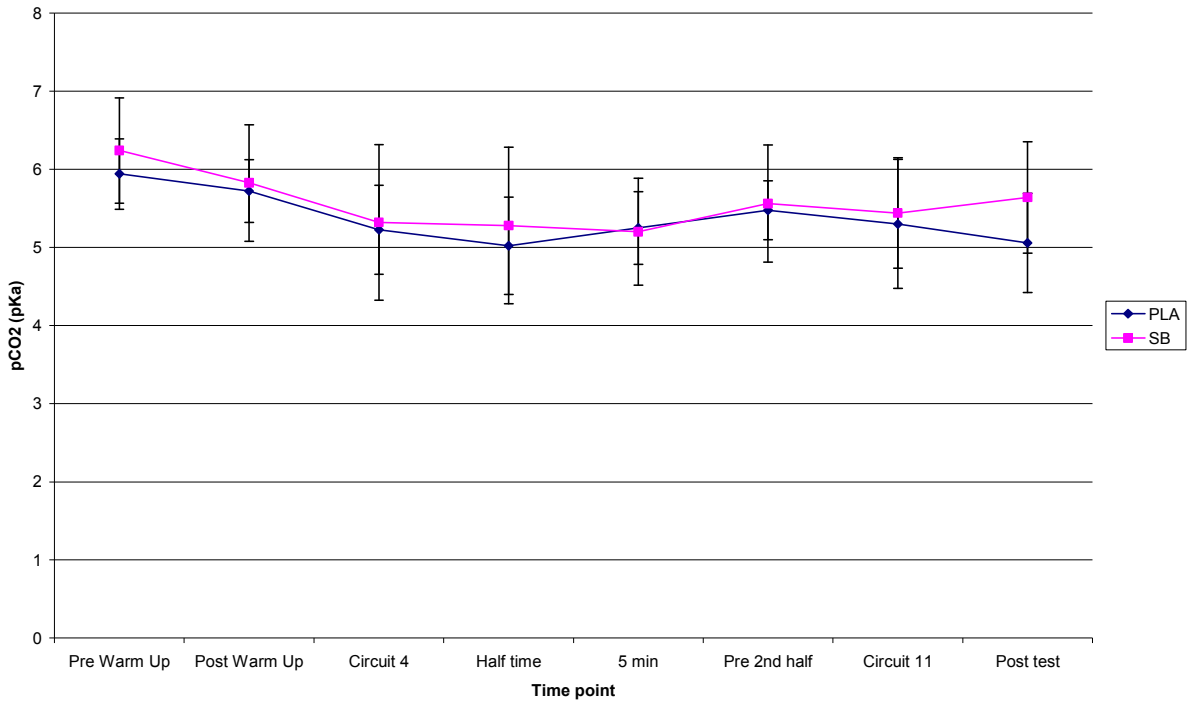


Figure 5.9: PCO_2 (kPa) across time points for each supplementation protocol (males), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.6.2 Elite Female Data

Similarly no significant differences in PCO_2 were observed between trials at any time point (*see Figure 5.10*). However, immediately prior to the 2nd half, the difference between SB and PLA trials was approaching a significant level (5.20 ± 0.16 vs. 4.5 ± 0.39 kPa for SB and PLA respectively; p=0.090).

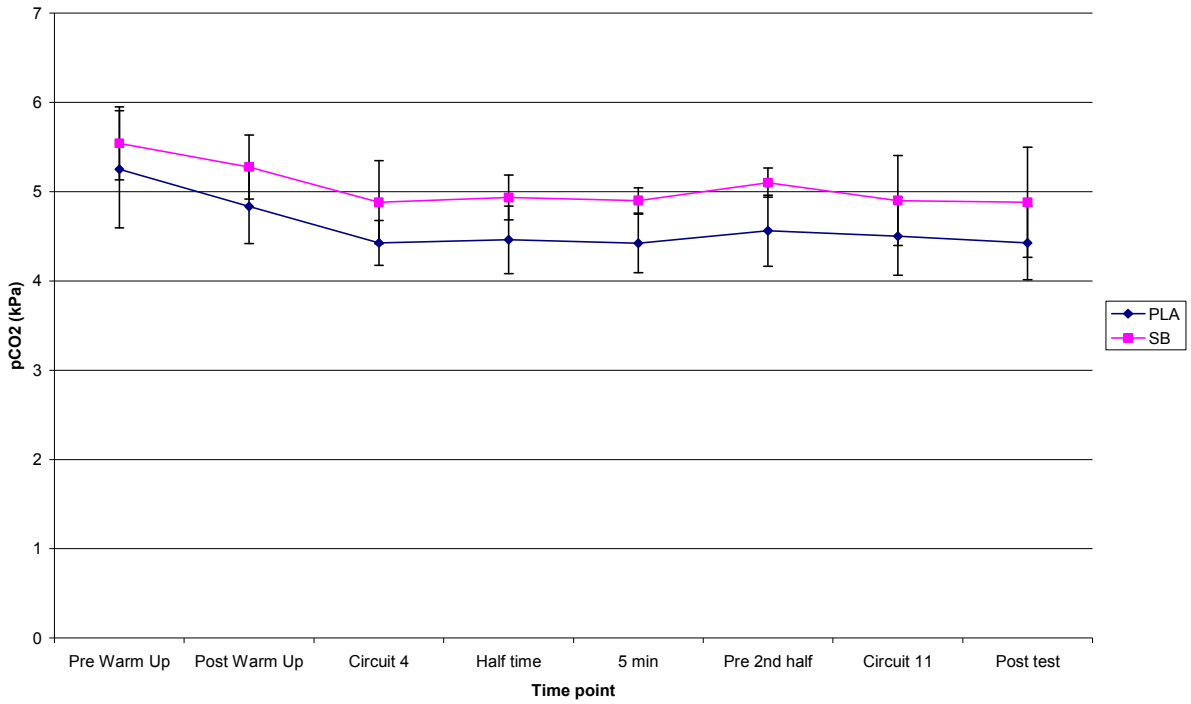


Figure 5.10: PCO_2 (kPa) across time points for each supplementation protocol (females), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.7. Partial Pressure of O_2 (PO_2)

5.3.7.1 Sub-Elite Male Data

No significant differences in PO_2 were detected at any time point between SB and PLA trials (*see Figure 5.11*). Pre-warm up PO_2 values were identified as 10.52 ± 1.06 vs. 10.68 ± 1.31 kPa for SB and PLA supplementation, respectively (p=0.763). Post-test PO_2 values were recorded as 10.58 ± 0.61 vs. 11.10 ± 0.73 kPa for SB and PLA supplementation, respectively (p=0.261).

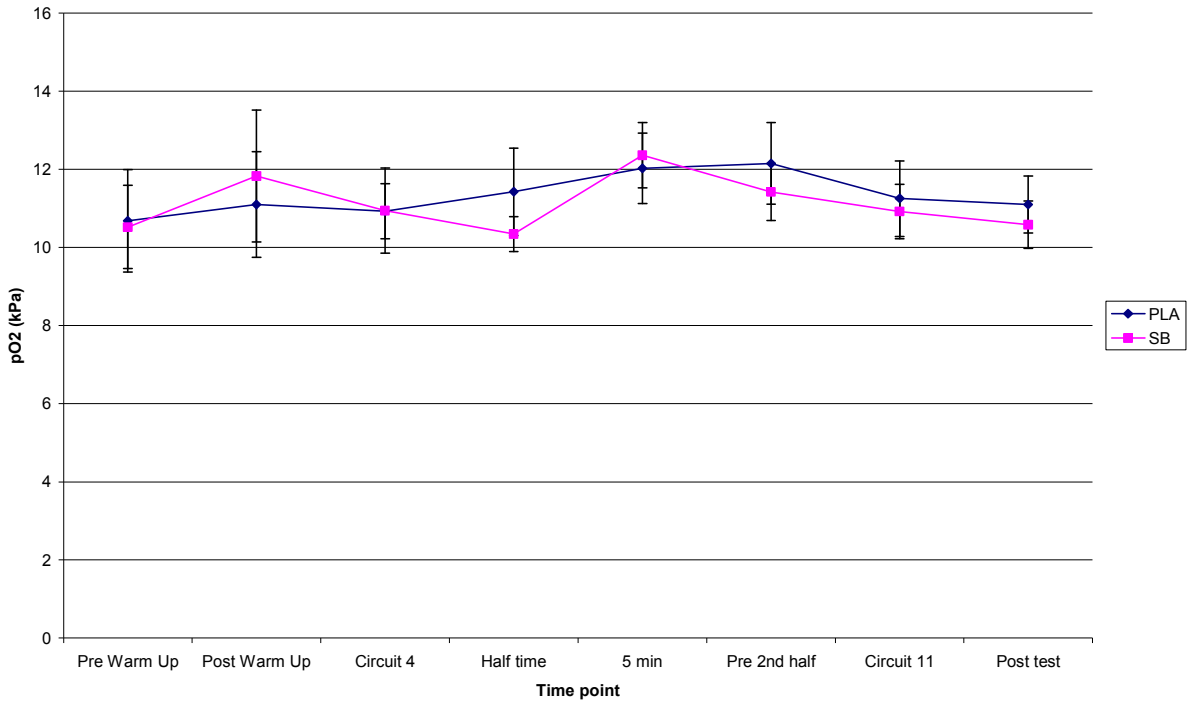


Figure 5.11: PO_2 (kPa) across time points for each supplementation protocol (males), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.7.2 Elite Female Data

As with the sub-elite male subjects, no significant differences in PO_2 were detected at any time point between SB and PLA trials for the elite females (*see Figure 5.12*).

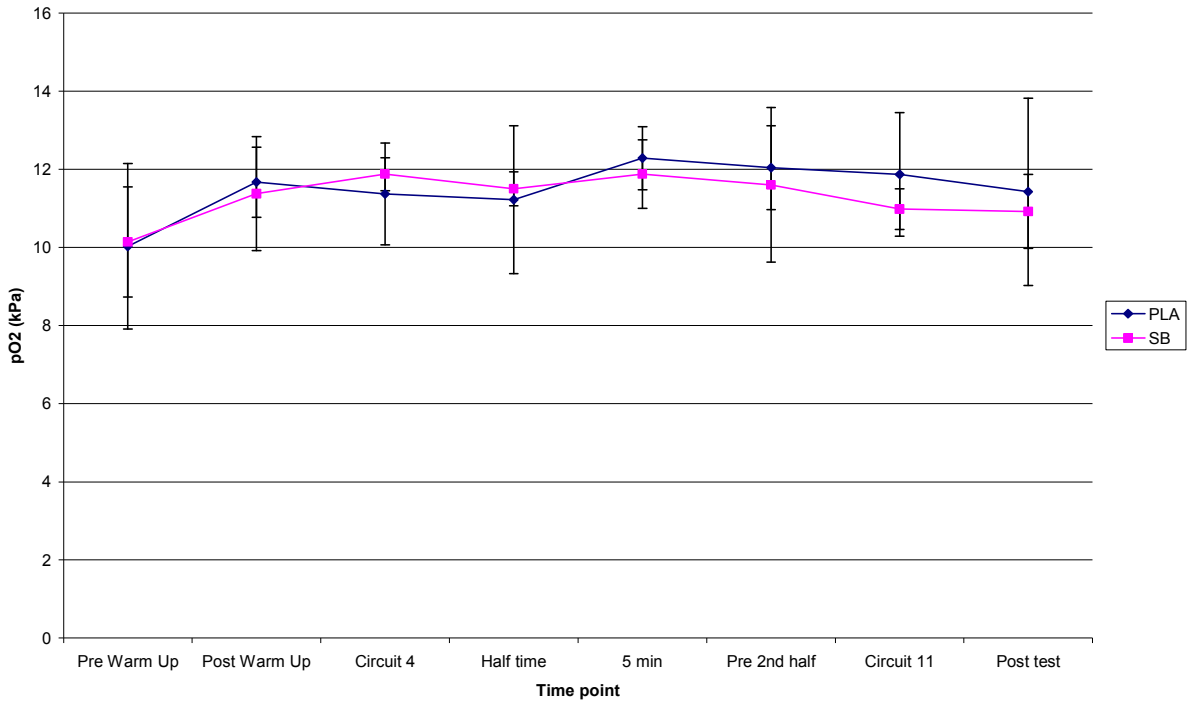


Figure 5.12: PO_2 (kPa) across time points for each supplementation protocol (females), (n=10, values denote mean ± SD)

(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.8. Performance Analysis Overview

Eight performance measures from the rugby-specific circuit protocol were analysed with the mean group and individual results for the 1st and 2nd halves outlined below. These performance measures included:

1. 20m sprint
2. Reactive agility speed (RAS) including its 3 components: RAS_15m sprint, RAS_reaction and RAS_total
3. Power 1: Vertical jump
4. Power 2: Weighted vertical jump
5. Defensive speed agility T drill
6. Tackle bag carry shuttle
7. Passing accuracy
8. Shuttle test

5.3.9. Mean Rugby-Specific Circuit Performance

The two conditions (SB and PLA) were compared using mean performance scores at each station of the circuit for 1st and 2nd half combined (all 14 circuits), mean scores for 1st half only, mean scores for 2nd half only, mean of scores recorded in the first two circuits, mean of scores recorded in the last two circuits, the difference between the mean of the first two circuits and the last two circuits.

5.3.9.1 Sub-Elite Male Data

From analysis of the mean performance results from all fourteen circuits throughout the entire 80 minute performance test (*see Figure 5.13*), it was determined that there were no significant differences in any element of performance between SB and PLA supplementation ($p>0.263$).

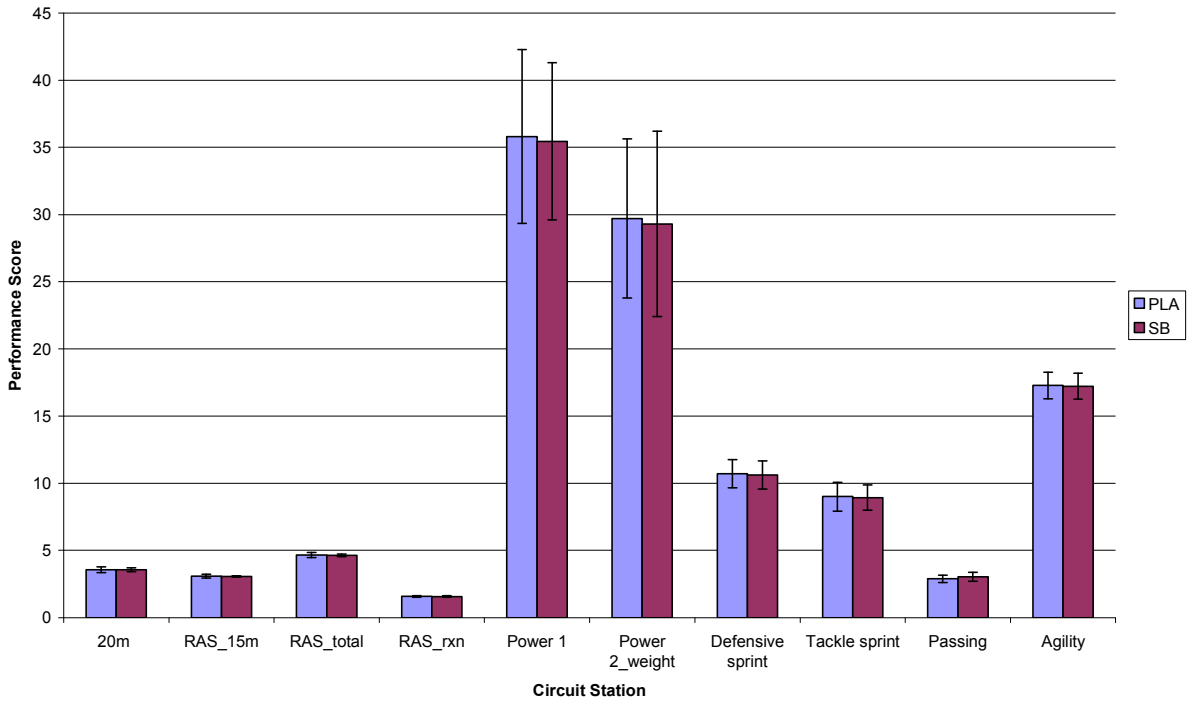


Figure 5.13: Mean performance scores at each station over all fourteen circuits of the 80-minute protocol for SB and PLA supplementation protocols (males), (n=10, values denote mean ± SD)

(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

Having observed no significant differences in performance between the two supplementation protocols over the course of the test as a whole, performance within each 40 minute half was then analysed separately. As shown in Figure 5.14 below, no significant differences in performance were identified between the two supplementation protocols. PLA supplementation seemed to record a slightly higher mean jump height for Power 2 (weighted vertical jump). However, this was not found to be significantly different to the SB condition ($p=0.060$ for Power 2 and $p>0.203$ for all other performance tasks).

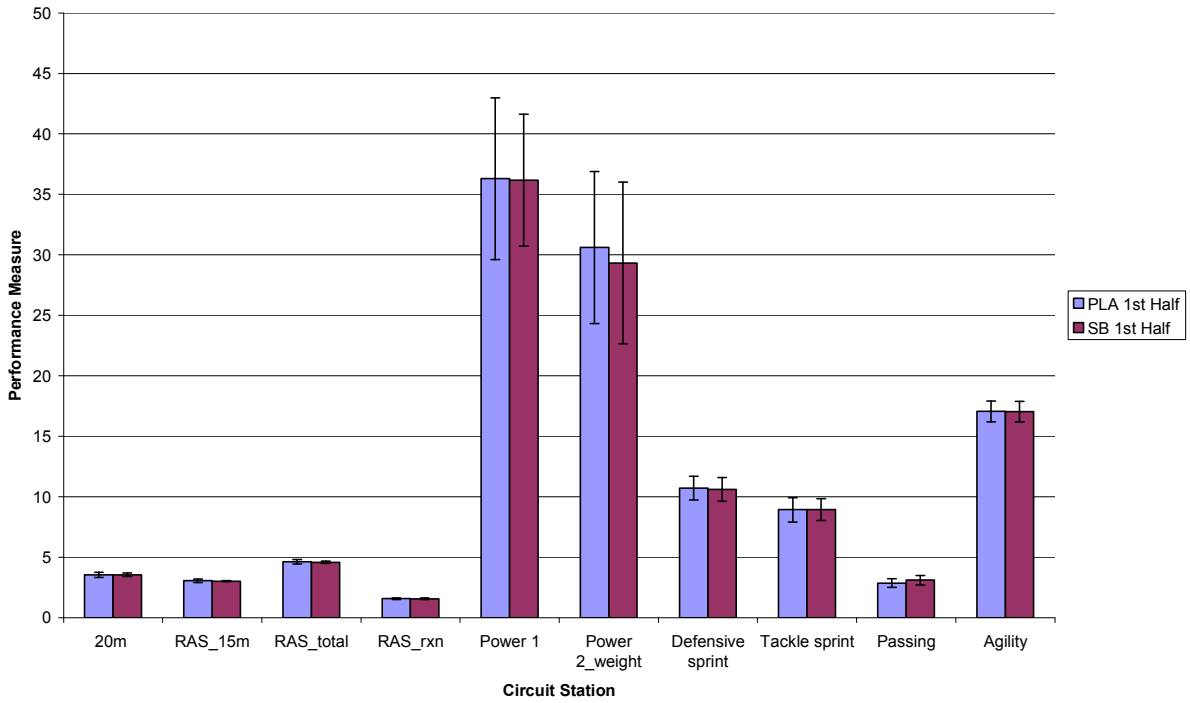


Figure 5.14: Mean 1st half performance scores for each task of the circuit for each supplementation protocol (males), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

As with the mean 1st half performance scores, no significant differences were detected between the two supplementation protocols upon analysis of the 2nd half performance, where p>0.286 for all performance tasks (*see Figure 5.15*).

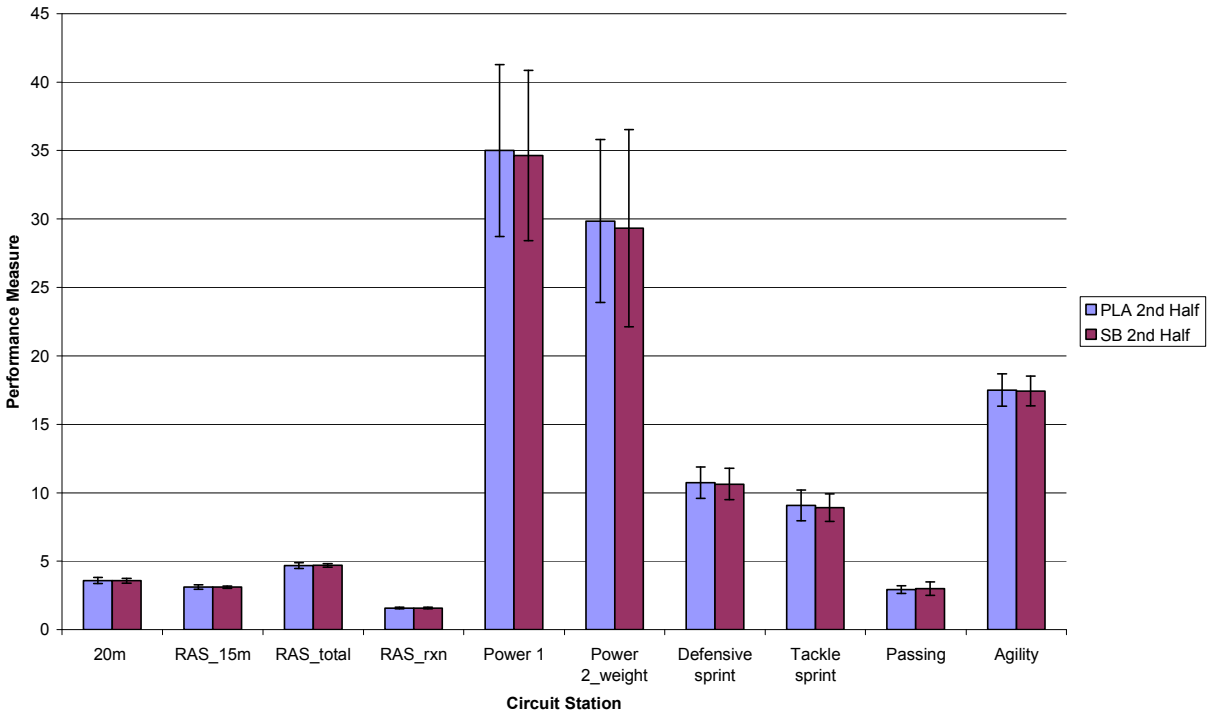


Figure 5.15: Mean 2nd half performance scores for each task of the circuit for each supplementation protocol (males), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

As a marker of fatigue index, the mean of the first two circuits and the mean of the last two circuits in both trials were investigated (*see Figure 5.16*). No significant differences in performance were found between the SB and PLA trials for the mean of the first two circuits across ($p>0.163$). Similarly, no significant differences were observed between supplementation protocols for the mean of the last two circuits ($p>0.347$).

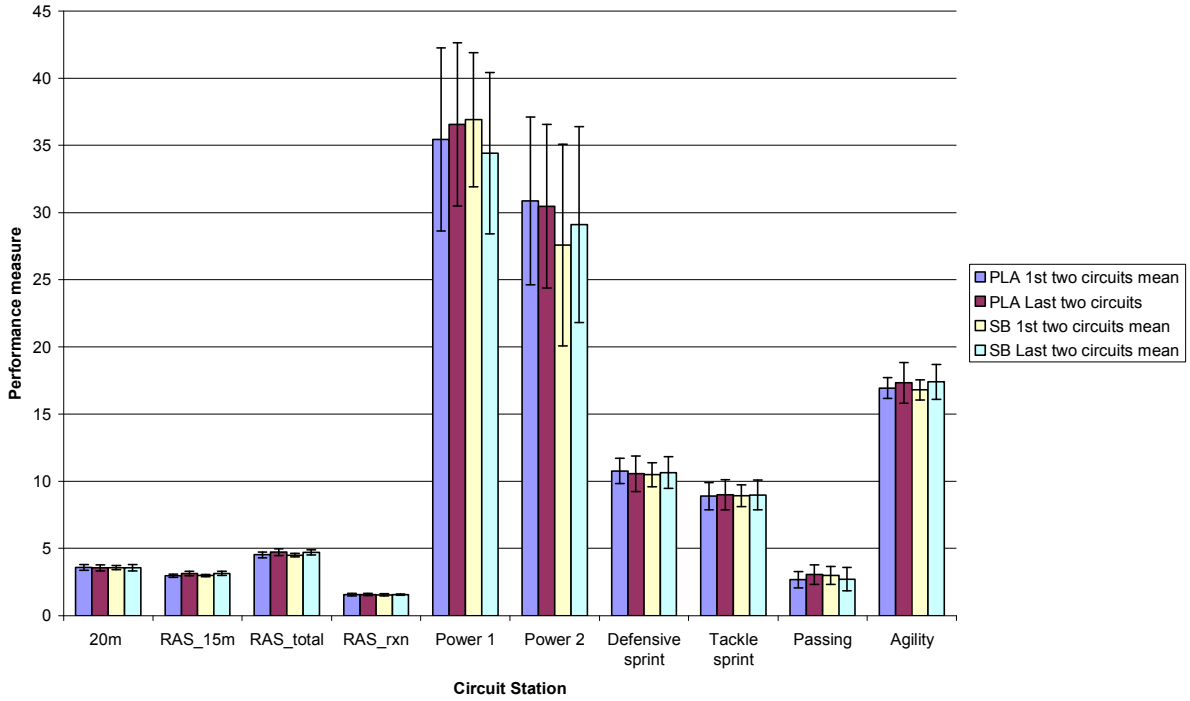


Figure 5.16: A comparison of the mean performance scores for the first two circuits and the last two circuits for both supplementation trials (males), (n=10, values denote mean ± SD)

(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

The difference between the mean of the first two circuits and the mean of the last two circuits was also investigated. Although a seemingly notable discrepancy between the mean of the first two and last two circuits appears for Power 2 in Figure 5.17, this difference did not reach significance (p=0.076). Therefore, no significant differences between the first two and last two circuits were identified across trials (p>0.118) for all performance tasks.

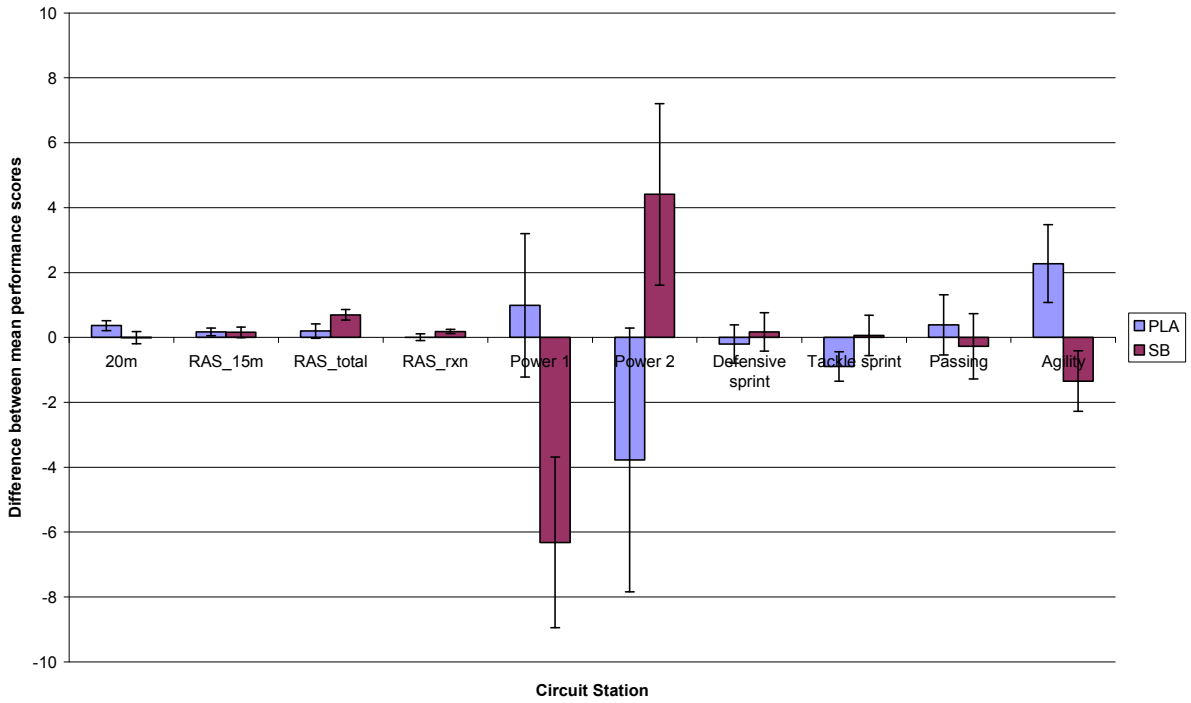


Figure 5.17: The difference between the mean of the first two circuits and the mean of the last two circuits across both supplementation trials (males), (n=10, values denote mean ± SD)

(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.9.2 Elite Female Data

Although the difference in weighted vertical jump height (Power 2) between the two trials appeared to be tending towards significance (p=0.062), jump height was not adjudged to be significantly higher with SB when compared to PLA supplementation (20.93 ± 2.91 vs. 20.35 ± 2.74cm). As a result, only one significant difference between the two supplementation protocols was identified across the mean of all fourteen circuits of the 80 minute performance test (*see Figure 5.18*). Passing accuracy was found to be significantly more accurate over the course of the test in the PLA trial as opposed to with SB supplementation (SB 2.68±0.62 vs. PLA 2.99 ± 0.55 accurate passes out of five; p=0.009).

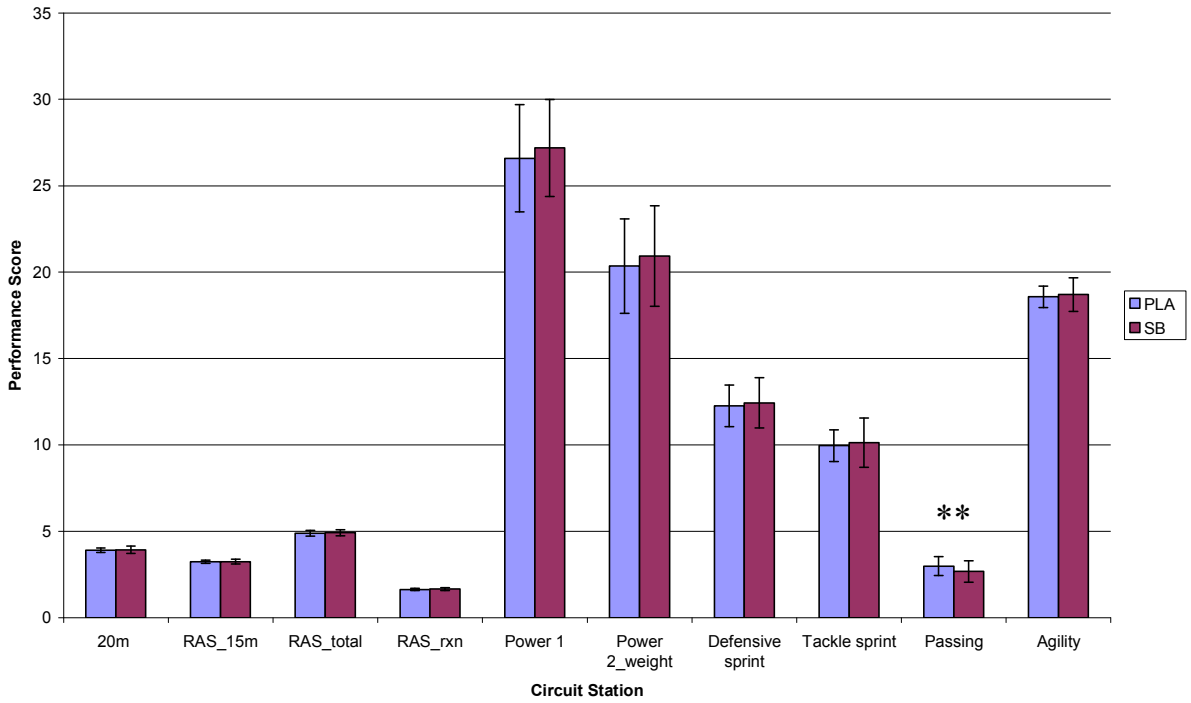


Figure 5.18: Mean performance scores at each station over all fourteen circuits of the 80-minute protocol for both supplementation protocols (females), (n=10, values denote mean ± SD)

(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

Following investigation of the mean scores for each station of the circuit in the first half and second half separately, just one point of significance in the each of the halves was observed between the two supplementation protocols. Within the 1st half (*see Figure 5.19*) the mean vertical jump height (Power 1) was found to be significantly greater with PLA supplementation when compared to SB. (SB 26.0 ± 2.9 vs. PLA 27.1 ± 3.3cm; p=0.010).

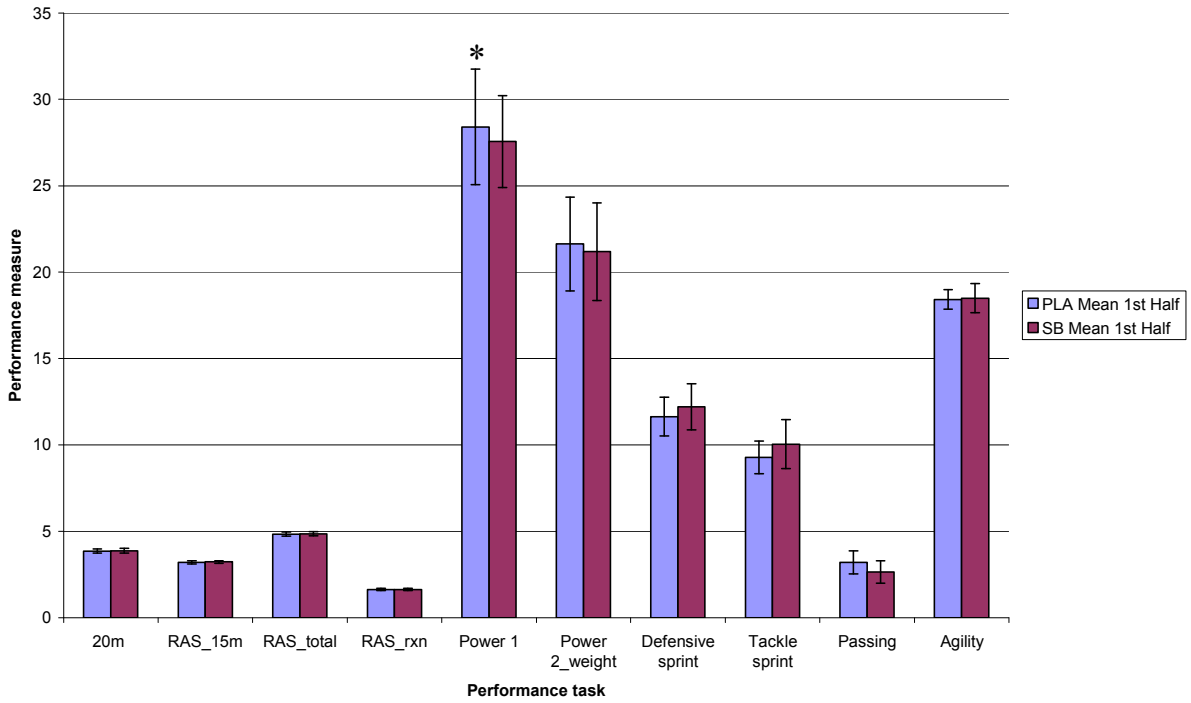


Figure 5.19: Mean 1st half performance scores for each task of the circuit for each supplementation protocol (females), (n=10, values denote mean ± SD) (*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

Within the 2nd half (*see Figure 5.20*), no significant differences between the mean results for the two supplementation protocols were observed with the exception of a significantly greater level of passing accuracy with the PLA protocol (3.1 ± 0.4 accurate passes out of five) in relation to SB supplementation (2.7 ± 0.6 accurate passes out of five), where p=0.022.

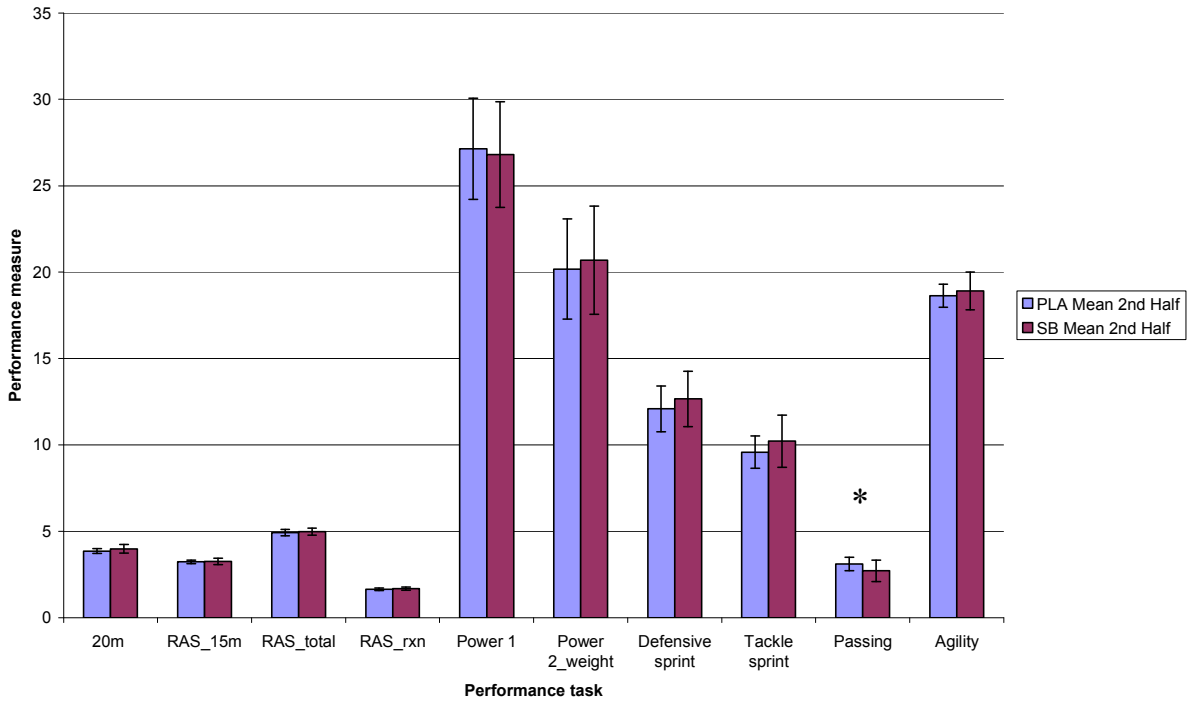


Figure 5.20: Mean 2nd half performance scores for each task of the circuit for each supplementation protocol (females), (n=10, values denote mean ± SD) (*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

As a marker of fatigue index, the mean of the first two circuits and the mean of the last two circuits in both trials were investigated (*see Figure 5.21*). No significant differences in performance were found between the mean of the first two circuits across SB and PLA trials. Similarly, no significant differences were observed between supplementation protocols for the mean of the last two circuits ($p>0.05$).

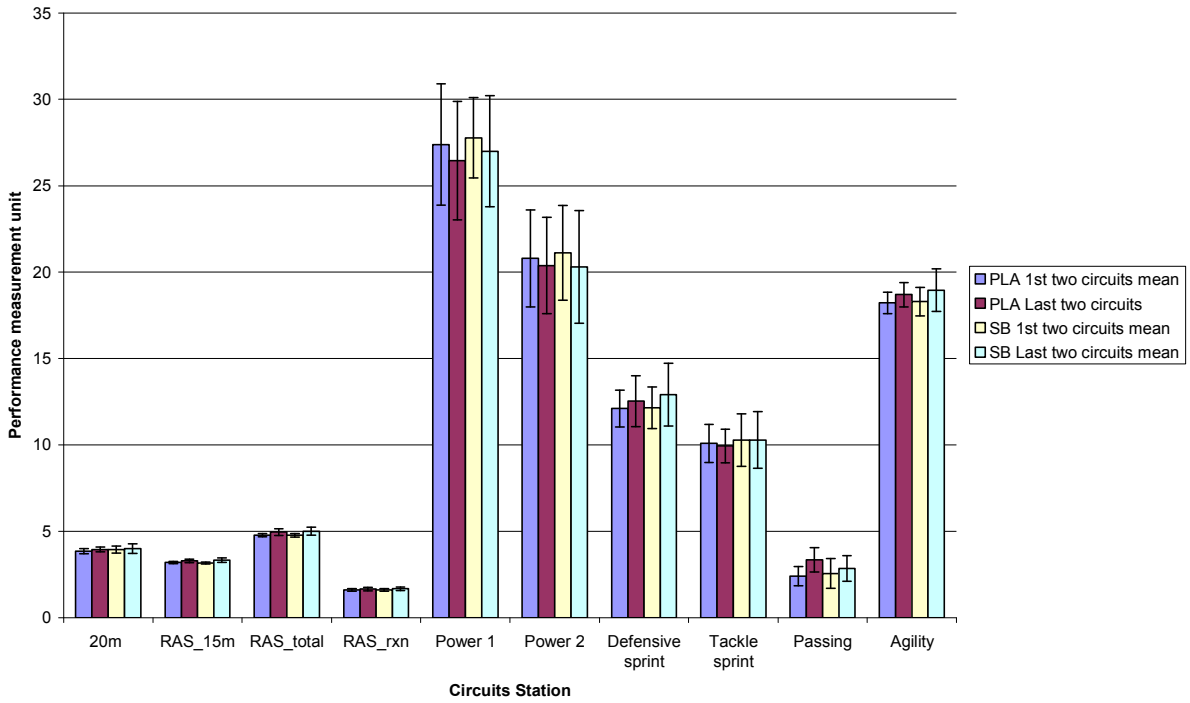


Figure 5.21: A comparison of the mean performance scores for the first two circuits and the last two circuits for both supplementation trials (females), (n=10, values denote mean ± SD)

(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

Subsequently, as illustrated in Figure 5.22, the difference between the mean of the first two circuits and the mean of the last two circuits was then explored. A significantly greater difference between the first two and last two circuits was identified with the SB trial when compared to the PLA trial for the defensive sprint T drill only (SB 0.75 ± 0.77 vs. PLA 0.43 ± 0.70; p=0.012). No other significant differences between the mean of the first two and the mean of the last two circuits were detected across trials.

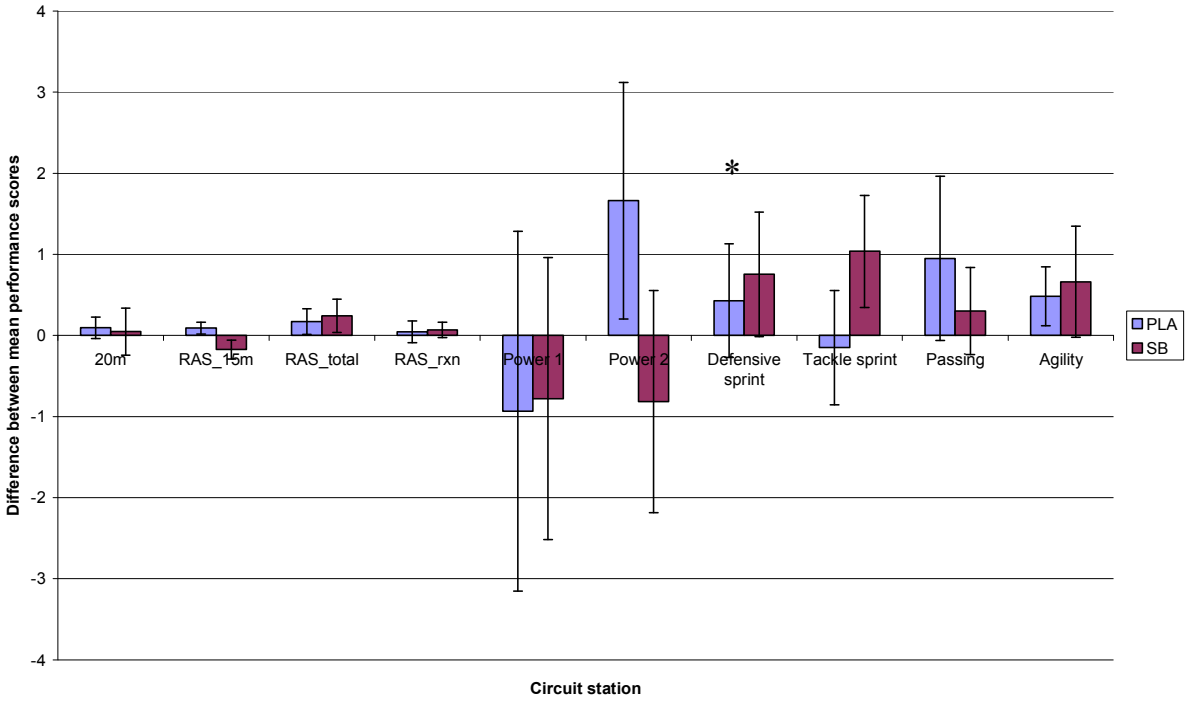


Figure 5.22: The difference between the mean of the first two circuits and the mean of the last two circuits across both the SB and PLA trials (females), (n=10, values denote mean ± SD)

(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.10. Individual Performance

5.3.10.1. Sub-Elite Male Data

Following individual analysis, out of the ten sub-elite male subjects, four subjects (40%) demonstrated an enhanced performance in some performance tasks with SB supplementation; two subjects (20%) experienced an increased level of some performance components with PLA supplementation; two subjects (20%) displayed elevated performance levels with both supplementation protocols for different performance tasks within the circuit; and the remaining two subjects (20%) showed no differences between the supplementation protocols. As in Section 4.3.14, means and standard deviations presented are based on all fourteen circuits completed by one individual subject.

Subject 8 encountered greater performance outcomes in six performance measures with SB when compared with PLA supplementation. 20m sprint (3.89 ± 0.13 vs. 4.03 ± 0.06s, respectively), RAS_15m (3.13 ± 0.04 vs. 3.37 ± 0.018s respectively), RAS_total (4.76 ± 0.06 vs. 5.09 ± 0.22s, respectively), Power 1 (29.13 ± 0.97 vs. 27.94 ± 1.18cm, respectively), the defensive T agility drill (12.09 ± 0.30 vs. 12.71 ± 0.58s, respectively) and the tackle bag carry shuttle (8.30 ± 0.23 vs. 8.71 ± 0.32s, respectively) all revealed more favourable mean performance scores with the experimental condition as opposed to the PLA.

Similarly, differences in performance were also found between the two supplementation protocols with Subject 1. SB supplementation elicited enhanced mean performance outcomes in RAS_15m (3.03 ± 0.08 vs. 3.18 ± 0.16s, respectively), RAS_total (4.47 ± 0.12 vs. 4.64 ± 0.19s, respectively), Power 2 (36.80 ± 2.43 vs. 32.09 ± 2.80cm respectively), the defensive T agility drill (9.92 ± 0.42 vs. 10.77 ± 0.40s, respectively) and the tackle bag carry shuttle (10.56 ± 0.50 vs. 11.18 ± 0.51s, respectively) when compared with PLA supplementation. Subject 3 also recorded faster RAS_15m times with SB supplementation in comparison to PLA (3.16 ± 0.15 vs. 3.23 ± 0.16s,

respectively). In addition, Subject 5 displayed a beneficial performance outcome with SB supplementation with respect to the PLA in the agility shuttle task (17.07 ± 0.23 vs. 17.53 ± 0.21s for each respective supplementation protocol).

In contrast, subject 6 and 9 exhibited superior performance outcomes with PLA as opposed to SB supplementation during certain elements of the performance test. Subject 6 demonstrated faster times in the 20m sprint (SB 3.64 ± 0.11 vs. PLA 3.51 ± 0.08s), RAS_total (SB 4.70 ± 0.20 vs. PLA 4.50 ± 0.10s), RAS_reaction (SB 1.61 ± 0.07 vs. PLA 1.50 ± 0.06s), the defensive T agility drill (SB 11.52 ± 0.45 vs. PLA 10.75 ± 0.28s) and the agility shuttle (SB 17.67 ± 0.56 vs. PLA 16.63 ± 0.55s), along with greater jump height in Power 1 (SB 31.90 ± 2.88 vs. PLA 34.33 ± 1.98cm) throughout the course of the PLA trial when compared to the SB trial. Comparably, the PLA supplementation protocol yielded greater results in Subject 9 in four of the same performance measures as Subject 6. 20m sprint times (SB 3.37 ± 0.13 vs. PLA 3.25 ± 0.06s), RAS_15m times (SB 3.01 ± 0.07 vs. PLA 2.93 ± 0.08s), RAS_total times (SB 4.62 ± 0.13 vs. PLA 4.48 ± 0.14s) and Power 1 jump height (SB 43.29 ± 1.89 vs. PLA 45.63 ± 2.35 s) were found to be improved with the PLA trial as opposed to the SB condition for this subject.

Subjects 4 and 7 experienced differences between the two trials, with performance enhancements being elicited by both PLA and SB supplementation with respect to each other for different elements of performance. Subject 4 recorded a greater mean RAS_reaction time (1.52 ± 0.03 vs. 1.58 ± 0.07s for SB and PLA, respectively) over the course of the 14 circuits in the SB trial when compared to the PLA. However, the same subject produced reduced sprint times for the 20m sprint (3.47 ± 0.05 vs. 3.38 ± 0.08s for SB and PLA, respectively), defensive T agility drill (10.43 ± 0.33 vs. 10.14 ± 0.21 for SB and PLA respectively) and agility shuttle (16.42 ± 0.31 vs. 16.06 ± 0.17 s for SB and PLA, respectively), along with significantly greater jump height in Power 2 (24.55 ± 1.42 vs. 25.86 ± 1.49 cm for SB and PLA, respectively) in the PLA trial as opposed to the SB trial.

Similarly, Subject 7 experienced enhanced jump height in Power 1 (SB 38.45 ± 2.84 vs. PLA 40.95 ± 2.61cm) with PLA when compared to the SB trial. However, SB supplementation, as opposed to PLA supplementation, resulted in enhanced performance in the defensive T agility drill (SB 9.17 ± 0.25 vs. PLA 9.59 ± 0.35s) and the agility shuttle task (SB 16.64 ± 0.62 vs. PLA 17.01 ± 0.42). Subjects 3 and 10 displayed no differences between the two supplementation protocols for any performance measure quantified.

5.3.10.2. Elite Female Data

Following individual analysis, out of the ten elite female subjects, three subjects (30%) demonstrated an enhanced performance in some performance elements with SB supplementation only; three subjects (30%) experienced an increased level of some performance components with PLA supplementation only; and the remaining four subjects (40%) displayed elevated performance levels with both supplementation protocols for different performance tasks within the circuit.

For Subject 6, SB supplementation elicited enhanced performance in the 20m sprint, RAS_15m and RAS_total, along with a higher vertical jump in Power 1 when compared to PLA supplementation. Similarly, Subject 5 experienced reduced sprint and reaction times for the 20m sprint and the RAS_reaction, respectively. In addition, SB supplementation resulted in a higher mean weighted jump height in Power 2 along with a faster agility shuttle speed when compared to the PLA protocol.

In contrast to these three subjects, Subject 3 demonstrated improved times with the PLA supplementation protocol for the 20m sprint, defensive T agility drill, tackle bag carry shuttle and agility shuttle. Subject 4 also recorded greater levels of performance in the PLA trial with respect to SB for the 20m sprint, RAS_reaction, defensive T agility drill, passing accuracy and the agility shuttle. Furthermore, mean performance enhancements were also observed in the PLA trial for Subject 8 during the 20m sprint, Power 1,

defensive T agility drill, tackle bag carry shuttle and the agility shuttle, when compared to SB supplementation.

The remaining four subjects experienced differences between the two trials, with performance enhancements being elicited by both PLA and SB supplementation with respect to each other for different elements of performance. Subject 1 recorded greater jump height in both Power 1 and Power 2 in the SB trial when compared to the PLA. However, the same subject produced faster sprint times for the agility shuttle and RAS_15m in the PLA trial as opposed to the SB trial. Subject 2 exhibited a faster mean defensive T agility time with PLA when compared to SB supplementation. However, SB supplementation produced enhanced performance in the tackle bag carry and agility shuttle. Similarly, Subject 9 generated a faster RAS_reaction with PLA when compared to SB supplementation. However, a faster mean tackle bag carry shuttle was recorded by the same subject with SB supplementation as opposed to PLA. Finally, Subject 10 obtained a greater mean passing accuracy in the PLA trial versus the SB trial. However, SB supplementation was associated with increased performance in Power 1, Power 2, defensive T agility drill and the agility shuttle, when compared to the PLA protocol.

5.3.11. Gastro-Intestinal (GI) Ratings

5.3.11.1. Sub-Elite Male Data

Although SB supplementation appeared to generate higher ratings of gastro-intestinal discomfort (*see Figure 5.23*), no significant mean differences were found between the two supplementation protocols at any time point throughout the performance test ($p>0.537$). As with the elite female subjects, the potential effect of GI discomfort experienced by subjects on performance during the trials was assessed using a linear regression analysis. Within the SB supplementation trial, the relationship between subjects' GI ratings and each element of the performance test was low and not considered to be statistically relevant ($p>0.306$). However, there was a significant association

between mean GI ratings and mean performance in the PLA supplementation trial. A significant relationship was found in the PLA supplementation condition between mean GI ratings and performance in the 20m sprint (p=0.020, r=0.749, r^2=0.562) RAS_15m (p=0.038, r=0.695, r^2=0.484), RAS_reaction (p=0.017, r=0.762, r^2=0.580), RAS_total (p=0.007, r=0.816, r^2=0.665), defensive T agility drill (p=0.004, r=0.843, r^2=0.710) and the agility shuttle (p=0.005, r=0.838, r^2=0.702).

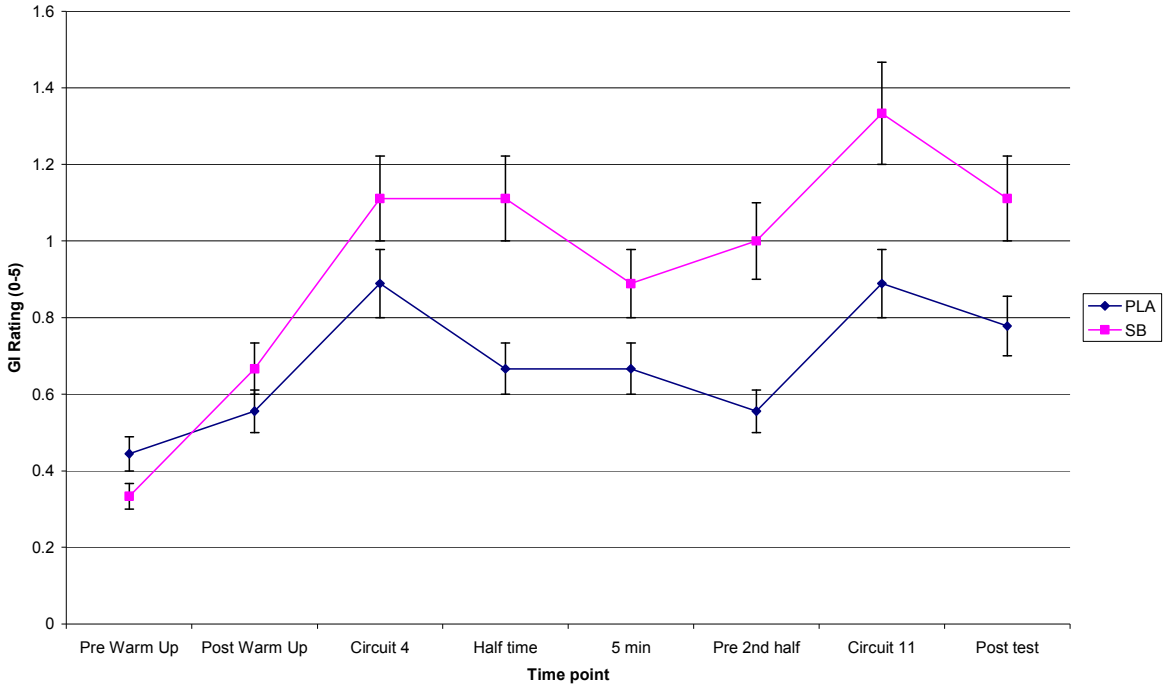


Figure 5.23: Mean GI ratings across each time point for each supplementation protocol (males), (n=10, values denote mean ± SD)

(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.11.2. Elite Female Data

Ratings of GI discomfort between the two trials were found to be relatively similar at the majority of time points (*see Figure 5.24*) and, consequently, no significant differences were found between the two supplementation protocols at seven of the eight time points. However, immediately following the fourth circuit of the 1st half (Circuit 4), SB supplementation demonstrated a significantly greater mean GI rating than the PLA trial (SB 0.5 ± 0.53 vs. PLA 0.1 ± 0.32 on a GI rating scale of 0-5), where p=0.037.

To assess the potential effect of GI discomfort experienced by subjects on performance during the trials, a linear regression analysis was conducted. Within the PLA trial, the relationship between subjects' GI ratings and each element of the performance test was not considered to be statistically relevant (p>0.093). However, there was a significant association between mean GI ratings and mean performance in the SB supplementation trial. A regression analysis was conducted between mean GI ratings and mean performance scores in each of the performance elements of the 80-minute rugby specific protocol performance test. A significant positive relationship was found between mean GI ratings and RAS_15m (r=0.678, r^2=0.460, p=0.031,), RAS_reaction, r=0.715, r^2=0.511, (p=0.020), RAS_total (, r=0.695, r^2=0.483, p=0.026), defensive T agility drill (r=0.826, r^2=0.681, p=0.003), tackle bag carry shuttle (r=0.776, r^2=0.603, p=0.014) and the agility shuttle (r=0.798, r^2=0.637, p=0.006) in the SB supplementation condition i.e. the greater the GI rating, the lower the performance level.

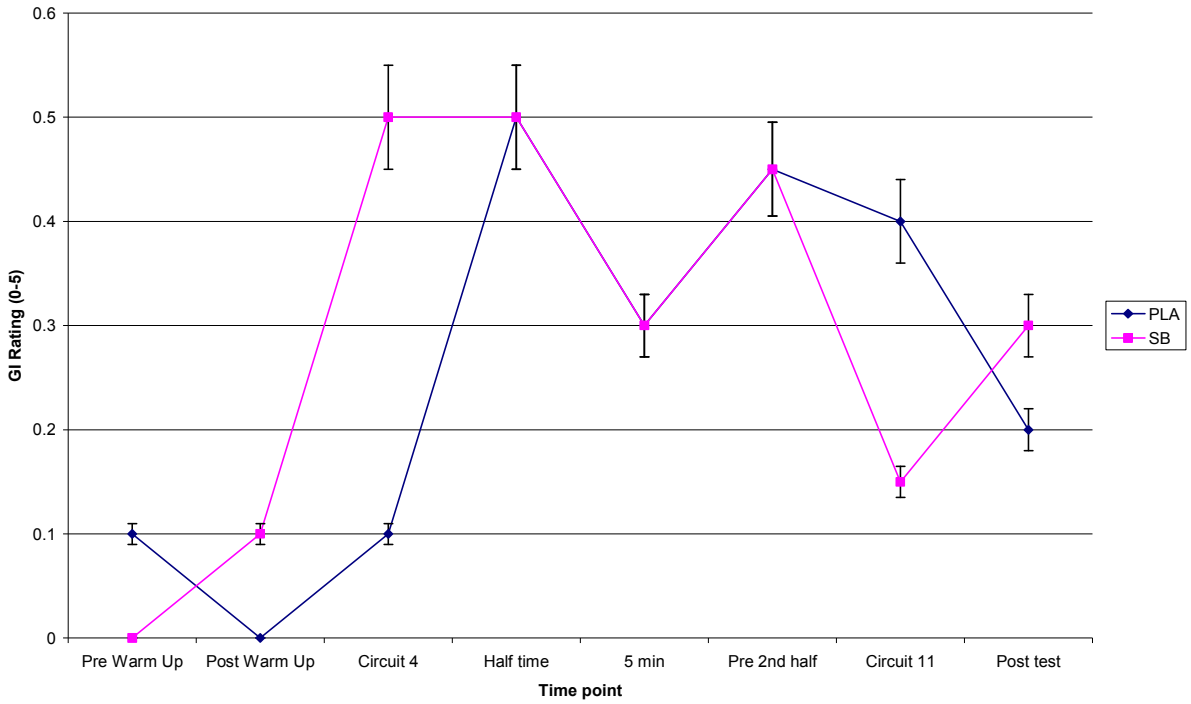


Figure 5.24: Mean GI ratings across each time point for each supplementation protocol (females), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.12. Muscle Soreness

5.3.12.1 Sub-Elite Male Data

No significant differences in perceived muscle soreness ratings were detected between the two supplementation protocols at any time point throughout the test (*see Figure 5.25*). Pre-warm up muscle soreness ratings of 0.9 ± 1.0 and 0.5 ± 0.7 on a muscle soreness scale of 0-10 were recorded for PLA and SB supplementation trials, respectively (p=0.104) The greatest muscle soreness ratings were recorded post-test, at which point mean ratings of 4.2 ± 2.3 and 4.2 ± 2.2 on a muscle soreness scale of 0-10 (Appendix C) were observed for PLA and SB supplementation trials, respectively (p=1.000).

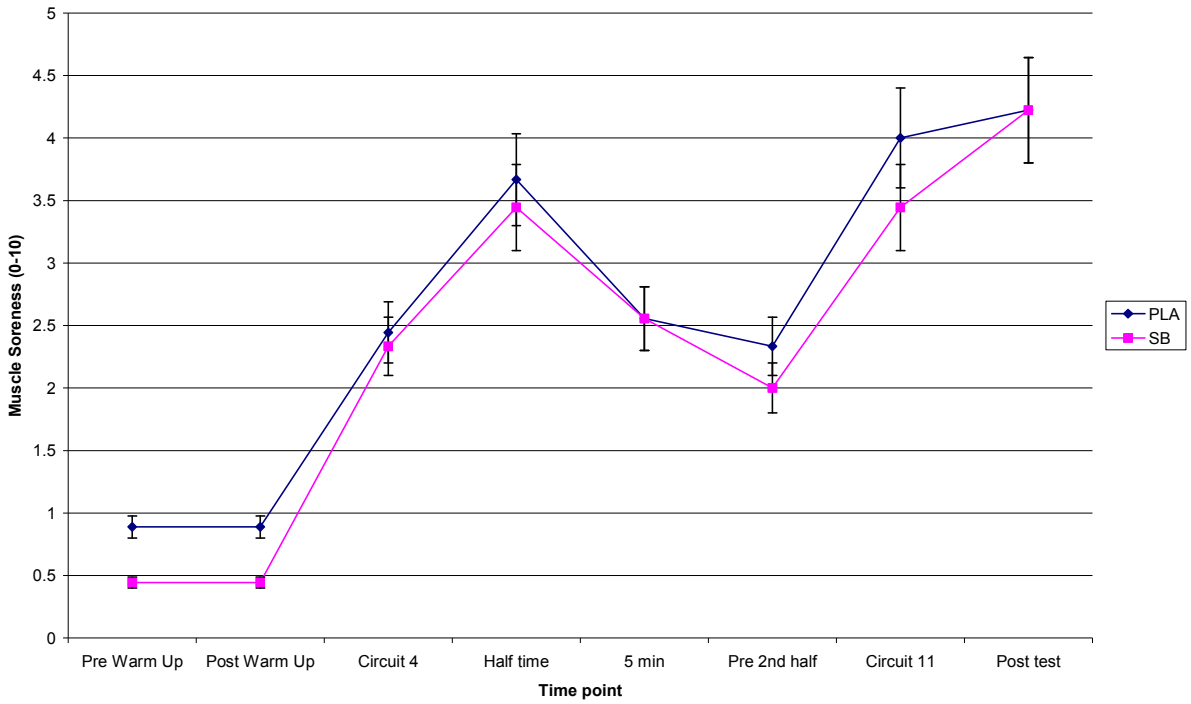


Figure 5.25: Mean muscle soreness ratings across each time point for each supplementation protocol (males), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.12.2 Elite Female Data

No significant differences in perceived muscle soreness ratings were detected between the two supplementation protocols at any time point throughout the test (*see Figure 5.26*). The greatest muscle soreness ratings were recorded post-test at which point mean ratings of 4.3 ± 2.2 and 4.0 ± 1.9 on a muscle soreness scale of 0-10 were observed for SB and placebo supplementation trials respectively (p=0.734).

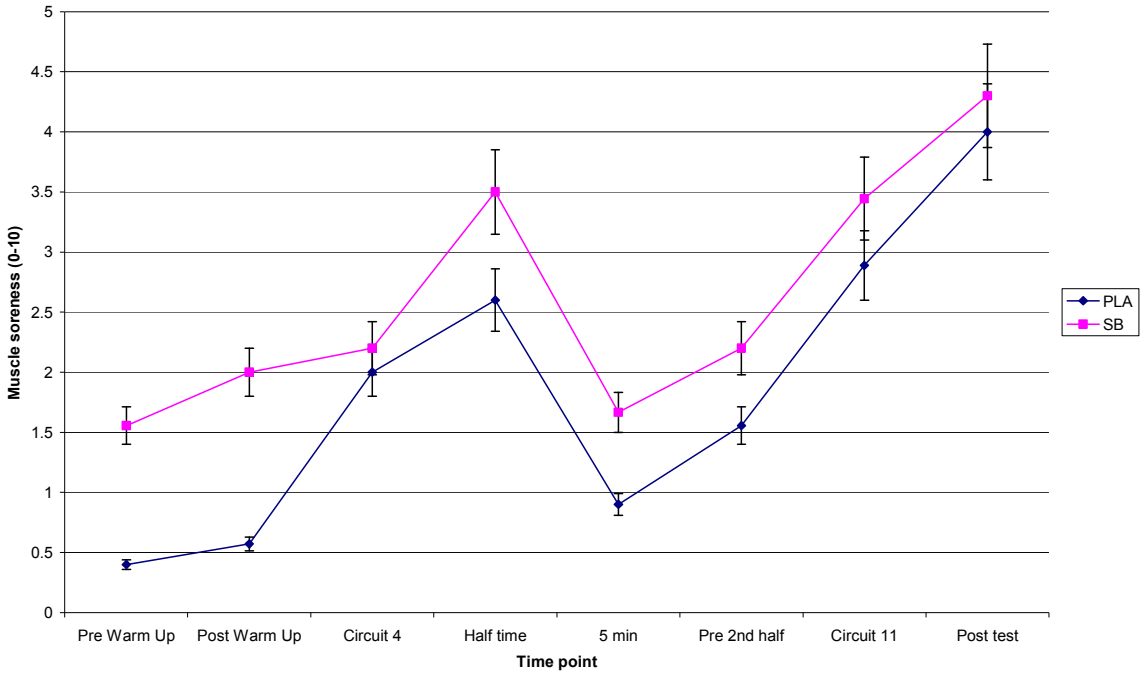


Figure 5.26: Mean muscle soreness ratings across each time point for each supplementation protocol (females), (n=10, values denote mean ± SD) (*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.13 RPE

5.3.13.1 Sub-Elite Male Data

Similarly, RPE exhibited no significant differences (*see Figure 5.27*) between the PLA and SB supplementation trials at any time point (p>0.141).

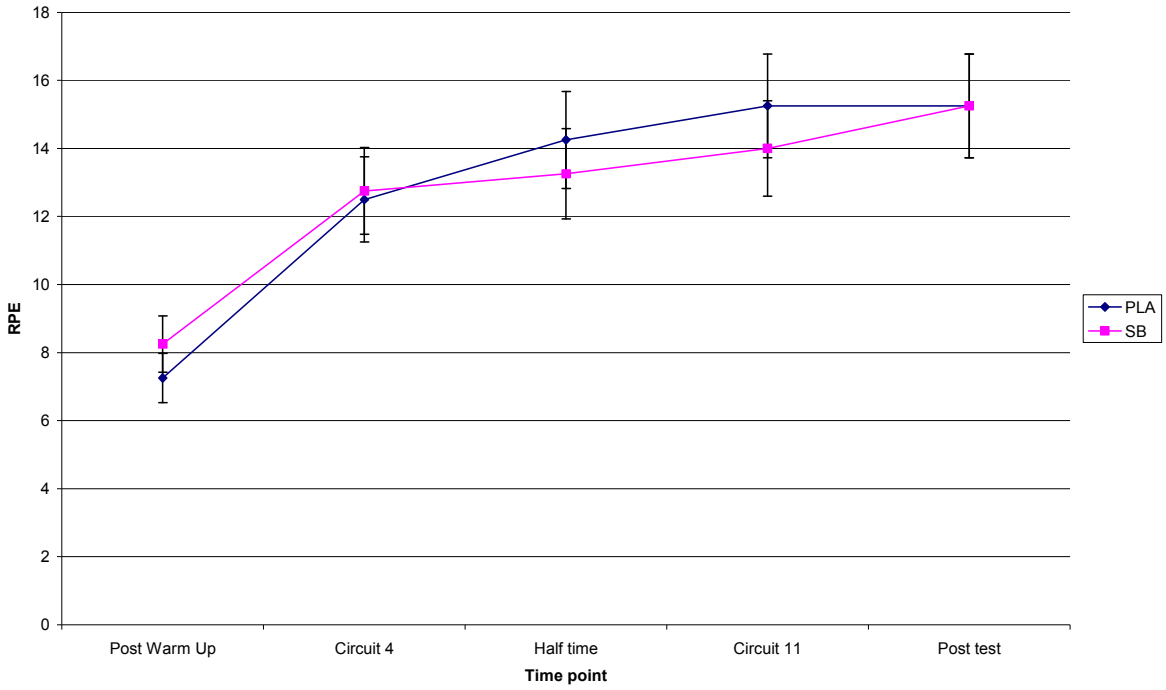


Figure 5.27: Mean RPE across each time point for each supplementation protocol (males), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.13.2 Elite Female Data

Similarly, RPE exhibited no significant differences (*see Figure 5.28*) between the PLA and SB supplementation trials at any time point (p>0.143).

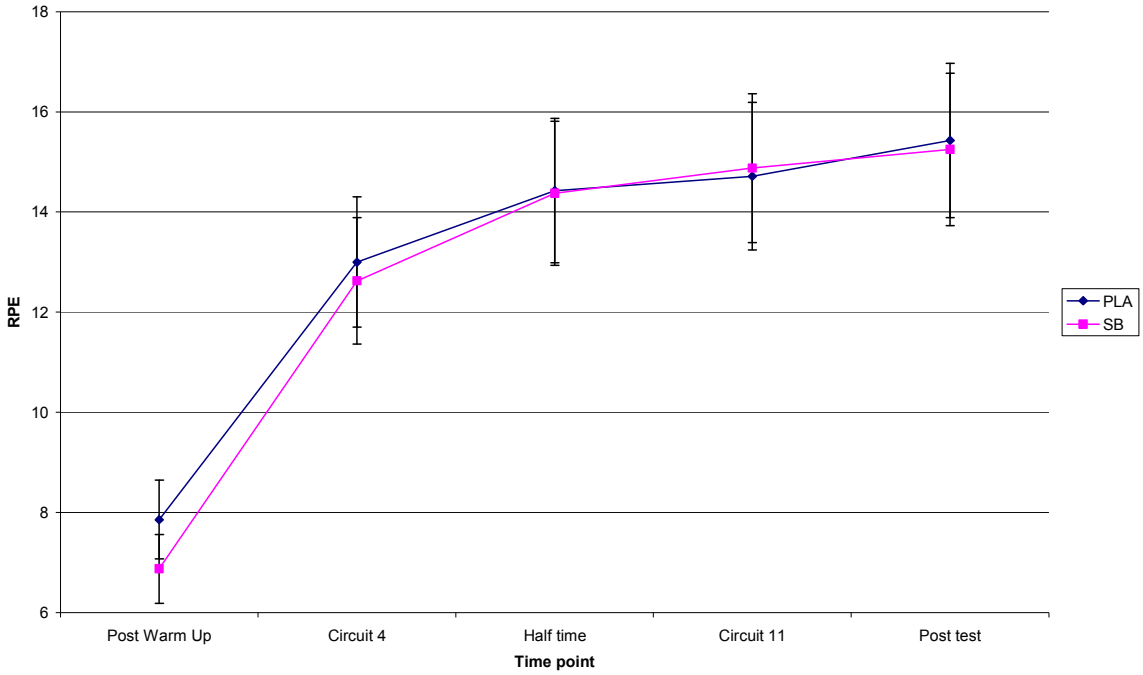


Figure 5.28: Mean RPE across each time point for each supplementation protocol (females), (n=10, values denote mean ± SD)
(*p<0.05, **p<0.01, significant difference between treatments, paired sample t-test)

5.3.14. Heart Rate

5.3.14.1 Sub-Elite Male Data

No significant differences in mean (SB 139 ± 13 vs. PLA 134 ± 11 bpm; p=0.266) or maximum (SB 183 ± 12 bpm vs. PLA 178 ± 9 bpm; p=0.198) recorded heart rates were observed between SB and PLA ingestion protocols over the course of the performance test.

5.3.14.2 Elite Female Data

Similarly, no significant differences in mean (SB 137 ± 9 vs. PLA 136 ± 9 bpm; p=0.884) or maximum (SB 177 ± 10 vs. PLA 177 ± 11 bpm; p=0.932) heart rates were observed between trials for the elite female group.

5.3.15. Summary

From statistical analysis, several points of significance were identified between SB and PLA trials ($p<0.05$). Within the blood variables measured, SB ingestion in sub-elite males when compared to PLA ingestion displayed a significantly higher level of $StdHCO_3^-$ at pre-warm up and post-warm up only; significantly elevated pH at all time points apart from following circuit 4 and at half time; significantly greater BE-Ecf at pre-warm up only; and significantly higher blood lactate concentrations at post-warm up, half time, 5 minutes into half time, post-circuit 11 and post-test. Elite females demonstrated a similar response with SB supplementation eliciting significantly greater $StdHCO_3^-$ levels at all time points with the exception of 5 minutes into half time and post-test; significant elevations in pH at pre-warm up, circuit 4, pre-2^{nd} half, circuit 11 and post-test; significantly greater BE-Ecf levels at pre-warm up, 5 minutes into half time, pre-2^{nd} half and following circuit 11; and no significant difference in blood lactate concentration but a trend towards significance at circuit 4 ($p=0.078$), when compared to PLA ingestion. No significant differences in PCO_2 or PO_2 were observed.

In terms of performance, sub-elite males recorded no significant differences in performance between trials. In the elite female group, across the mean of all fourteen circuits, PLA ingestion resulted in significantly greater passing accuracy when compared to SB ingestion ($p=0.009$). Following analysis of mean 1^{st} half performance, the elite females recorded a significantly greater jump height in Power 1 with PLA when compared to SB ingestion ($p=0.010$). Upon examination of 2^{nd} half mean performance, the elite female group displayed significantly greater passing accuracy with PLA as

opposed to SB ingestion ($p=0.022$). In addition, the difference between the mean of the first two and mean of the last two circuits was found to be significantly greater in the SB trial than the PLA trial ($p=0.012$).

GI disturbances were significantly greater with SB ingestion at one time point (circuit 4) in the elite female group ($p=0.037$). No significant differences in mean GI disturbance were recorded for males. A significant relationship was found between mean GI ratings and performance in the SB trial for the elite females. In contrast, the sub-elite males produced a significant relationship between mean GI ratings and performance in the PLA trial and not the SB trial. No significant differences in heart rate, RPE or muscle soreness were recorded between trials. A high degree of individual variability was also revealed. These results will be discussed in the following section.

5.4. Discussion

5.4.1. Introduction

Rugby union, along with many other team field sports, is characterised by the performance of repeated bouts of high intensity exercise interspersed with periods of active recovery of varying duration. The purpose of this study was to examine the effect of acute SB ingestion on this type of sport-specific performance. Results of this study indicated that, while acute SB ingestion had the capacity to induce pre-exercise alkalosis in both elite females and sub-elite males, conflicting findings were recorded in relation to the ergogenic benefit to rugby union-specific performance resulting from this perturbation in acid-base balance. This may indicate that, despite these positive ergogenic effects, the performance in activities typically associated with the specific physiological demands of rugby union, and other similar team field sports, may not benefit from acute SB ingestion.

5.4.2. Sport-Specific Simulated Match-Play

Previous research has suggested that the nature of the exercise protocol employed by research studies investigating SB ingestion may be critical in determining an ergogenic effect (Matson and Tran 1993). The mechanism by which SB ingestion is purported to operate is centred on an enhancement of blood buffering capacity (Siegler et al., 2010). This enhanced buffering capacity facilitates the removal of the excess H^+ accumulation associated with the provision of energy from glycolysis during high intensity exercise (Robergs et al., 2005). In order for SB ingestion to have the potential to enhance the buffering capacity and, consequently, performance, the type of exercise protocol utilised must involve a sufficient degree of anaerobiosis (Matson and Tran, 1993). Depending on the exercise intensity and duration required at any given time point within a rugby union match, all three energy systems contribute towards the provision of ATP for muscular contraction (Nicholas, 1997). However, the typical high intensity, short duration bouts

associated with rugby union ensure a heavy reliance on anaerobic glycolysis for energy production (Deutsch et al. 1998). Given the lack of significant findings in relation to the performance outcome in the current study, however, it is possible that the recovery periods within the sport of rugby union may render the potential effect of enhanced buffering unnoticeable (Cunniffe et al., 2009).

In an attempt to replicate the physiological demands associated with an 80 minute 15-a-side rugby union match, a specifically designed test protocol was developed as the performance test for this study. A thorough review of the scientific literature revealed only one other study (Cameron et al., 2010) examining the effects of SB ingestion on rugby-specific exercise. However, this exercise protocol involved just 9 minutes of rugby-specific activity, followed by 10 x 40m maximal intensity sprints every 30 seconds. Cameron et al. (2010) stated that this rugby-specific repeat-sprint performance test was designed to be applicable, based on time motion analysis, to both the sevens and 15-a-side codes of rugby union. However, given the stark differences between the physiological demands of rugby sevens and 15-a-side rugby union, the evidence within the literature does not support this supposition (Suarrez-Arrones et al., 2012; Cunniffe et al., 2009). The researchers (Cameron et al., 2010) conceded that the protocol, designed by a strength and conditioning trainer, did not involve the typical rest intervals associated with rugby union. Therefore, Cameron et al. (2010) concluded that the 9-minute rugby-specific training session represented 40-60 minutes of 15-a-side rugby union activity or, alternatively, the initial 9 minutes of a 14 minute rugby sevens match. While other studies have employed similar 80-minute rugby-specific protocols (Hamlin et al., 2008; Roberts et al., 2010), the current study is the first to the author's knowledge, to examine the effects of SB ingestion on such a protocol.

5.4.3. Subject Characteristics

Another distinctive aspect of the current research is the participation of elite female athletes. From examination of the literature up to 1993, a review by Matson and Tran (1993) calculated that the contribution of females athletes to research studies involving SB ingestion constituted just 3% of the total subject population (9 females out of a total of 295 subjects). In a more recent meta-analysis by Peart et al. (2012), data was presented for 47 females out of a total of 395 subjects across forty research articles (12% approximately). As a result, the recommendation to include female athletes in further SB investigations was suggested to examine the effect of SB ingestion on females and also to analyse any potential gender differences in response to SB ingestion (Matson and Tran, 1993; Peart et al., 2012). In addition, the aforementioned review paper (Matson and Tran, 1993), along with Peart et al. (2012) advocated the necessity for future research to explore the effect of SB ingestion on highly trained athletes in competitive performance. The current study aimed to address both of these issues through the recruitment of elite female subjects (currently playing at an international or interprovincial standard).

As previously mentioned (*see Section 5.2.1*), a total subject group of twenty athletes, comprising of ten elite females and ten sub-elite males participated in the current study. As cited in Study 1 (*see Section 3.4*) and 2 (*see Section 4.4*) of this work, sample size may have contributed towards the failure to demonstrate significant performance enhancements following SB supplementation. In order to attempt to curtail this issue, a larger contingent of subjects were recruited for the present study. Matson and Tran (1993) reported the sample size range within investigations involving SB ingestion to lie between 4 and 23, with a mean sample size of 8 ± 2 subjects. As a result, the sample size of twenty subjects selected for the current study sits near the upper limit within the range of previous studies completed.

5.4.4. Effects of SB Ingestion on Blood Parameters

The main findings of the current study demonstrated that, while both males and females responded relatively similarly to acute SB ingestion with regard to blood pH, $StdHCO_3^-$ and BE-Ecf (*see Section 5.3.2, 5.3.3 and 5.3.4. respectively*), significant differences in certain performance variables were identified (*see Section 5.3.9*). From these results it appears that 0.3g/kg BM of SB was sufficient to induce metabolic alkalosis in both males and females. In support of this data, previous research has exhibited a significantly higher alkalinity in trained males (Cameron et al., 2010) and elite female athletes (Tan et al., 2010). Within the current study, there was, however, some dissimilarity between males and females in terms of post-exercise blood lactate response. In accordance with the majority of the literature (Requena et al., 2005; Peart et al., 2012), the male subjects in the current study recorded significantly higher blood lactate concentrations with SB ingestion at several time points including post-warm up, half-time, 5 minutes into half-time, post-circuit 11 and post-test ($p<0.05$). This finding is widely supported in the research, particularly in investigations involving high intensity exercise with a heavy reliance on anaerobic metabolism (Bishop et al., 2004; Requena et al., 2005; Kupcis et al., 2012). Kupcis et al. (2012) reported peak blood lactate concentrations of 13.4 ± 1.7 vs. 11.9 ± 1.9 mmol/L following SB versus PLA supplementation prior to 2000m rowing performance; $p=0.001$). An increase in the activity of the lactate/H^+ co-transporter, promoting an increased efflux of lactate from the muscle cells, is purported to be the mechanism underpinning the increased post-exercise lactate concentrations observed with SB ingestion (Juel, 1998; Hollidge-Horvat et al., 2000).

Within the current study, although there was a trend towards significance at one point post-circuit 4 ($p=0.078$), no significant elevations in post-exercise blood lactate concentration were observed in the elite females with SB ingestion when compared to the PLA trial. These findings are contrary to those of previous research involving the effect of acute SB ingestion on elite female athletes in simulated match performance (Bishop et al., 2005; Tan et al., 2010). In light of the fact that blood lactate concentrations were tending towards significance at one point in the present study, sample size may be a

possible explanation for the inconsistency. However, the sample size utilised in the current study was higher than the average of 8 ± 2 subjects observed in a review of research involving SB ingestion (Matson and Tran, 1993). In addition, neither of the two aforementioned studies (Bishop et al., 2005; Tan et al., 2010) involved running as the mode of exercise. Rather, field hockey match-play was simulated using a cycle ergometer protocol (Bishop et al., 2005) and water polo performance was replicated with swimming (Tan et al., 2010). As a result, due to the diverging modes of activity used, these two studies may not be entirely comparable to the current studies findings. Furthermore, the periods of active recovery involved in the match simulation protocol may also have encouraged an accelerated clearance of blood lactate in the elite female group. However, this was not observed in their trained male counterparts in performance of an identical rugby match simulation protocol.

5.4.5. Effects of Metabolic Alkalosis on Performance

In relation to performance, acute SB ingestion resulted in no significant difference between trials for male subjects. Mean first half weighted vertical jump height appeared to be approaching significance ($p=0.060$) in favour of SB ingestion. However, no significant differences in performance were observed. Cameron et al. (2010) also found no significant improvement in exercise performance in a similar subject group to the present study (trained male rugby players). As previously discussed, the rugby-specific protocol undertaken by Cameron et al. (2010) varied significantly from the present study and involved a much shorter duration, thus making absolute comparisons between the two studies difficult. However, both studies (Cameron et al., 2010 and the present study) entailed high intensity, intermittent running performance in rugby union players and as such, similarities may be drawn. On the contrary to the findings of Cameron et al. (2010), Bishop and Claudis (2005) observed a positive ergogenic effect with acute SB ingestion on team field sport simulation. The field hockey simulation protocol consisted of 2 x 36 minute “halves” of intermittent repeat sprint activity. However, as previously mentioned, this protocol was carried out on a cycle ergometer.

Performance between the SB and PLA trials for the elite females presented conflicting results. From analysis of the mean of all fourteen circuits of the 80 minute performance test, passing accuracy was found to be significantly more accurate over the course of the test with PLA as opposed to SB supplementation (SB 2.68 ± 0.62 vs. PLA 2.99 ± 0.55) accurate passes out of five; p=0.009). Vertical jump height (Power 1) was also found to be significantly greater in the PLA trial than the SB trial (SB 26.0 ± 2.9 vs. PLA 27.1 ± 3.3cm; p=0.010). In addition, acute SB ingestion was found to elicit a significantly greater difference between the mean of the first two circuits and the mean of the last two circuits for the defensive T sprint (SB 0.75 ± 0.77 vs. PLA 0.43 ± 0.70; p=0.012).

Contradicting numerous studies reporting either significant performance enhancement or no difference between SB and PLA trials (Siegler et al., 2010; Carr et al., 2012; Bishop and Claudis, 2005), these results suggest that acute SB ingestion may have a detrimental effect on performance in relation to passing accuracy and lower body power. This may be partially accounted for by the significantly greater rating of GI disturbance recorded for the SB trial in comparison to PLA supplementation (SB 0.5±0.53 vs. PLA 0.1±0.32 on a GI rating scale of 0-5; p=0.037). However, this significant difference in GI disturbance was observed at just one time point (immediately following circuit 4) and as a result, may not be entirely responsible for the decrement in performance. Alternatively, as fatigue is multi-faceted, a separate factor may have contributed towards the significantly decreased performance in passing accuracy and lower body power. It may also be the case that SB ingestion did not cause a detriment, but rather that the PLA resulted in a performance enhancement. However, the substance utilised in this study (maltodextrin) has been used throughout the research as a valid placebo (Glaister et al., 2006; Abrams et al., 2005; Culbertson et al., 2010). As a result, the probability is that the GI symptoms were the dominant factor underlying the differences in performance.

The aforementioned results, outlining diminished performance with acute SB ingestion, are in contrast to previous studies (Peart et al., 2012; Carr et al., 2011). Another potential explanation for the negative effect may lie in the nature of the performance test. To the author's knowledge, no other published study has examined the effects of SB ingestion

on an 80-minute rugby-specific match simulation protocol involving a diverse range of tasks indicative of those involved in rugby match-play. Previous sport-specific research utilising SB ingestion has involved cycling (Bishop and Claudis, 2005), swimming (Tan et al., 2010) or straightforward running for an insufficient duration to simulate team field sport match-play (Cameron et al., 2010). Within a sport-specific field test such as the current protocol, there may be a risk of increased variability when compared to a more reliable, controlled laboratory experiment such as a Wingate cycle ergometer test. However, in order to investigate team field sport fully, the exercise protocol selected or designed must be specific to the sport in question. Another possible factor contributing towards the conflicting findings is the high degree of individual variability associated with SB ingestion. Although only GI disturbance was only observed to be significantly higher at one time point within the females, it remains a potential factor.

5.4.6. Effects of SB Ingestion on Recovery from High Intensity Intermittent Exercise

The present research also examines the effect of acute SB ingestion on recovery during the 10 minute half-time period imposed to simulate the recovery period during competitive match-play. Following an observation of the changes in acid-base balance during simulated soccer match-play, Russell et al. (2012) reported that blood pH and buffering capacity reduced throughout soccer-specific exercise but returned to normal during half time. The current study corroborated this data and demonstrated that, although pH did decrease in both SB and PLA trials during the "first half" of the simulated rugby match, the half-time recovery period returned pH to the respective pre-warm up levels in both males and females.

The elevated pH exhibited following SB ingestion, in relation to the PLA, prior to the warm up for the performance test (7.47 ± 0.03 vs. 7.38 ± 0.02; p=0.046 for females and 7.42 ± 0.03 vs. 7.37 ± 0.01; p=0.014 for males) was maintained following the half-time recovery period (7.46 ± 0.006 vs. 7.36 ± 0.001; p=0.001 for females and 7.43 ± 0.03 vs. 7.35 ± 0.01; p=0.007 for males). This signifies that the widely reported metabolic

alkalosis observed with SB ingestion prior to exercise may become evident once again after a brief recovery period (Siegler et al., 2010; Carr et al., 2011; Peart et al., 2012). It has been reported (Peart et al., 2012) that metabolic alkalosis, resulting in an enhanced buffering capacity, is the major mechanism by which SB ingestion is purported to act. The current study suggests that SB ingestion may have the potential to impact on subsequent performance following a half-time recovery period. These results, therefore, establish the potential foundation for the use of SB ingestion as a recovery strategy.

An analysis of the existing literature revealed that few studies have previously investigated the efficacy of SB ingestion in the context of recovery (Verbitsky et al., 1997; Siegler et al., 2010). Verbitsky et al. (1997) reported reduced fatigue and enhanced recovery with acute SB ingestion following supramaximal cycling. Verbitsky and colleagues (1997) separated two bouts of cycling at 117% VO_{2max} with 1 hour of SB ingestion of 0.4g/kg BM. Functional electrical stimulation was then applied to the quadriceps femoris. The resulting increased knee torque associated with SB ingestion suggested an enhancement of anaerobic metabolism in isometric contraction. In a more recent examination, Siegler et al. (2010) investigated the combination of SB ingestion with traditional recovery modalities. Following acute SB ingestion, performance of a series of 3 x 30 second Wingate tests was separated by 3 minutes of either active or passive recovery. The results of this study suggested that an amalgamation of pre-exercise SB ingestion with active recovery between exercise bouts may enhance acid-base recovery from high intensity exercise (Siegler et al., 2010). Siegler et al. (2010) proposed that the increased extracellular buffering capacity of the SB ingestion is complemented by the increased blood flow associated with active recovery, leading to improved high intensity intermittent performance. Both of the aforementioned studies (Verbitsky et al., 1997; Siegler et al., 2010), along with the current study support the potential benefit of SB ingestion in relation to recovery. However, further research is necessary to investigate the effects of SB ingestion on various recovery period durations, different modes of exercise and also the combination of SB ingestion with other recovery modalities such as ice bath immersion and compression therapy.

5.4.7. Summary

The findings of the current study substantiate the previously reported observations within the existing literature regarding the capacity of acute SB ingestion to induce pre-exercise alkalosis (Siegler et al., 2010; Bishop et al., 2010). This study also lends support to a potential role of acute SB ingestion in recovery from high intensity exercise which may have implications for half time recovery strategies. However, in terms of performance, the results appear both conflicting and inconclusive with regard to the effect of acute SB ingestion on simulated rugby-specific performance. Although previous research in the area of SB ingestion has reported positive ergogenic effects in isolated single bout performance, this may not transfer to team field sport performance. There is currently a dearth of research involving the effects of SB supplementation on team sport performance, particularly studies involving sport-specific match simulation protocols. As a result, further research is required to investigate the effects of acute SB ingestion on sport-specific simulated or actual team-sport performance involving the intensity, duration, movement patterns and mode of exercise relevant to that sport. With regard to the current study, the hypothesis was partially proven regarding the metabolic alkalosis and enhanced recovery of the blood parameters following the half time period. However, once again, as with the previous two studies, the prediction of enhanced performance as a result of this acute SB supplementation protocol did not come to fruition. Therefore, there is no evidence to suggest that this supplementation protocol may be beneficial for either elite female or sub-elite male rugby union players.

.

Chapter 6

Summary, Conclusions and Recommendations for Future Research

6.1. Summary

The aim of this research was to examine the effects of acute and chronic SB supplementation on high intensity, intermittent performance, recovery and subsequent performance in trained rugby union players. In pursuit of this aim, three separate, yet closely linked experiments were designed and implemented. The major findings of this work suggest that pre-exercise metabolic alkalosis may be induced following acute but not chronic SB ingestion. However, results are inconclusive regarding the efficacy of acute or chronic SB ingestion to enhance performance in high intensity, intermittent exercise indicative of the movement patterns and physiological demands associated with rugby union. Results also appear to indicate a high degree of individual variability, which in part may be due to potential gastro-intestinal side effects of SB ingestion.

Study 1 examined the effects of acute and chronic SB ingestion on high intensity intermittent performance. Following acute or chronic SB supplementation, subjects performed a 10 second Wingate test every 60 seconds for 6 minutes. Acute SB supplementation consisted of ingesting 0.1g SB/kg body mass at 90, 60 and 30 minutes prior to performance (0.3g/kg over 90 minutes). Chronic SB supplementation entailed SB ingestion of 0.3g/kg body per day for a period of 5 days prior to the performance test. Results from this study demonstrated the capacity of acute SB supplementation to maintain a significantly greater blood $StdHCO_3^-$ and blood pH at all time points when compared to all other supplementation protocols. Acute SB supplementation corresponded to a significant increase of 5.9mmol/L in mean $StdHCO_3^-$ (from 24.9 ± 1.3mmol/L at baseline to 30.8 ± 1.1mmol/L pre-test) and a significant increase of 0.06 in pH (from 7.39 ± 0.02 at baseline to 7.45 ± 0.019 pre-test) following ingestion and immediately prior to the performance test ($p<0.01$). However, no significant differences in performance were observed. Chronic SB ingestion failed to demonstrate a significant level of pre-exercise alkalosis. Although mean $StdHCO_3^-$ was significantly greater than acute PLA at one time point (following Sprint 1) during the chronic trial ($p=0.016$), no ergogenic effect of chronic SB ingestion was established. It was purported that training status and gastro-intestinal side effects may have contributed to the inconsistent findings

with respect to acute SB ingestion. In relation to chronic SB ingestion, it was suggested that the body's buffering capacity may have corrected for the acid-base balance disturbance caused by SB ingestion and returned pH to homeostasis levels prior to performance, hence no pre-exercise alkalosis was observed. In addition, the high degree of individual variability combined with the sample size (n=10) may have impeded a statistically significant finding.

As outlined above, Study 1 examined the effects of both acute and chronic SB ingestion on a controlled laboratory experiment (repeated Wingate tests). In contrast, Study 2 investigated the effects of chronic SB ingestion on a field-based running test protocol, designed to replicate the high intensity intermittent activity associated with rugby union sevens match-play. The specifically designed test protocol was a unique aspect of this study as, prior to this research, SB ingestion had not been examined in relation to rugby sevens. This chronic SB supplementation protocol, which consisted of SB ingestion of 0.3g/kg per day for 5 days, exhibited no significant differences in blood parameters or performance related variables between SB and PLA trials. In light of the fact that no significant shift in acid-base balance was detected prior to performance, it was unlikely that SB ingestion would elicit a significant effect on performance unless an alternative unknown physiological mechanism was responsible for the previously reported ergogenic effect of SB ingestion (Siegler et al., 2010). As a result, a higher chronic SB dose of 0.5g/kg per day, as used in previous research (McNaughton et a., 1999) may be required to examine the potential to maintain blood alkalinity after cessation of ingestion.

Similar to the previous investigation (Study 2), Study 3, also examined the effects of SB ingestion on a sport-specific match simulation performance protocol, this time concentrating on 15-a-side rugby union. However, Study 3 focuses on acute SB ingestion as opposed to the chronic SB ingestion protocol utilised in Study 2. In an attempt to minimise sample size as a potential explanation for non-significant findings, a larger sample size (n=20; 10 elite females and 10 sub-elite males) participated in this study. In addition, taking into account that approximately only 12% of subjects investigated in previous studies were female, ten elite female athletes were recruited, thus enabling the

evaluation of possible gender differences. Results of this study indicated that acute SB supplementation with 0.3g/kg body mass ingested over the 90 minutes prior to performance (0.1g/kg body mass taken at 90, 60 and 30 minutes pre-performance) had the capacity to induce pre-exercise alkalosis in both elite females and sub-elite males. However, conflicting findings were recorded in relation to the ergogenic benefit to rugby union-specific performance resulting from this perturbation in acid-base balance. Additionally, as evident from an elevated pH following the half-time period, this study also lends support to a potential role of acute SB ingestion in short-term recovery from high intensity intermittent exercise.

6.2. Study Implications and Conclusion

It was hypothesised that acute and chronic SB ingestion would instigate pre-exercise alkalosis, leading to an enhanced extracellular buffering capacity and, thus, improved performance of test protocols designed to simulate the playing requirements of a team field sport, such as rugby union. Overall, despite a high degree of intra-individual variability of results, neither acute nor chronic SB loading demonstrated an enhancement in mean performance. However, acute SB ingestion was deemed to be capable of eliciting a significant shift in acid-base balance. Adopting a chronic supplementation protocol may prove beneficial for athletes in terms of ameliorating the gastrointestinal side effects associated with SB ingestion. However, further scientific research is necessary to definitively confirm the ergogenic effect of chronic SB ingestion. Recommendations to athletes regarding SB supplementation must stress the large degree of individual variability in terms of gastrointestinal side affects that may accompany SB ingestion. Research involving repeated acute SB ingestion has also detected a high within-subject variability of gastrointestinal symptoms (Carr et al., 2012). This may have implications for athletes considering SB supplementation as it indicates that regardless of prior familiarisation with SB, the manifestation of gastrointestinal side affects may vary on different occasions. As a result, despite experimentation with SB ingestion in training, athletes may not be guaranteed a similar response to SB ingestion during competition.

From a practical perspective, athletes and coaches may consider the use of SB supplementation on an individual, case-by-case basis, as responses appear to be highly individual which in part may be due to the variability of GI distress observed. However, on the basis of the three studies conducted throughout this research, there is no evidence to support the use of acute or chronic SB supplementation to enhance performance in elite female or sub-elite male rugby players.

6.3. Recommendations for Future Research

Results from this research suggest that, although pre-exercise alkalosis may be induced by acute SB ingestion, this may not translate into a performance enhancement in high intensity intermittent exercise. However, further research into this area is necessary in order to devise accurate and precise practical recommendations for athletes and coaches. Initially, the precise physiological mechanism underpinning the efficacy of SB ingestion to improve performance requires further investigation to be fully substantiated. Although improvements in performance with SB ingestion have largely been attributed to an enhanced buffering capacity, the potential mechanisms contributing to performance enhancement in the absence of significant blood pH changes following chronic SB ingestion require further examination. Recent research (Street et al., 2005; Sostaric et al., 2006) has indicated that SB ingestion may reduce the exercise-induced increase in extracellular potassium (K^+). As a result, further clarification in relation to primary and potential secondary mechanisms involved is required. Future research involving SB ingestion may benefit from analysis of the acid-base balance of the urine to identify any compensatory action by the body to eliminate the excess base ingested. In addition, muscle biopsy sampling may be advantageous to examine the intracellular muscle buffering capacity rather than the extracellular environment alone.

Limited research has been undertaken regarding the optimal SB supplementation protocol, particularly with regard to chronic SB ingestion. As a result, further investigation into the dose, number of doses and timing of ingestion prior to exercise may

be required to identify the circumstances under which the maximum increase in $StdHCO_3^-$, pH and BE-Ecf may be observed. Future consideration should also be dedicated towards exploring the scope of SB ingestion as a potential recovery strategy. Finally, additional research is necessary to determine any potential differences in the response of trained and untrained subjects to SB ingestion. It has been purported that highly trained individuals may have an already enhanced buffering capacity, thus, limiting the potential effect of SB ingestion. However, the scientific evidence to corroborate this is not yet definitive. In addition, the present research is one of few investigations to include female athletes. As a result, future research should further investigate the effect of SB ingestion on female athletes.

Bibliography

Abrams, S. A., Griffin, I. J., Hawthorne, K. M., Liang, L., Gunn, S. K., Darlington, G. & Ellis, K. J. 2005. A combination of prebiotic short- and long-chain inulin-type fructans enhances calcium absorption and bone mineralization in young adolescents. *Am J Clin Nutr,* 82**,** 471-6.

Aerenhouts, D., Deriemaeker, P., Hebbelinck, M. & Clarys, P. 2011. Dietary acid-base balance in adolescent sprint athletes: a follow-up study. *Nutrients,* 3**,** 200-11.

Ahlborg, B., Bergstrom, J., Ekelund, L. G., Guarnieri, G., Harris, R. C., Hultman, E. & Nordesjo, L. O. 1972. Muscle metabolism during isometric exercise performed at constant force. *J Appl Physiol,* 33**,** 224-8.

Applegate, E. 1999. Effective nutritional ergogenic aids. *Int J Sport Nutr,* 9**,** 229-39.

Applegate, E. A. & Grivetti, L. E. 1997. Search for the competitive edge: a history of dietary fads and supplements. *J Nutr,* 127**,** 869S-873S.

Artioli, G. G., Gualano, B., Coelho, D. F., Benatti, F. B., Gailey, A. W. & Lancha, A. H., Jr. 2007. Does sodium-bicarbonate ingestion improve simulated judo performance? *Int J Sport Nutr Exerc Metab,* 17**,** 206-17.

Aschenbach, W., Ocel, J., Craft, L., Ward, C., Spangenburg, E. & Williams, J. 2000. Effect of oral sodium loading on high-intensity arm ergometry in college wrestlers. *Med Sci Sports Exerc,* 32**,** 669-75.

Baldari, C., Videira, M., Madeira, F. 2004. Lactate removal during active recovery related to the individual anaerobic and ventilatory thresholds in soccer players. *European Journal of Applied Physiology,* 93**,** 224-230.

Belcastro, A. N., & Bonen, A. 1975. Lactic acid removal rates during controlled and uncontrolled recovery exercise. *Journal of Applied Physiology,* 39**,** 932-936.

Bellinger, P. M., Howe, S. T., Shing, C. M. & Fell, J. W. 2012. The Effect of Combined Beta-Alanine and NaHCO3 Supplementation on Cycling Performance. *Med Sci Sports Exerc*.

Bennett, A. F., & Licht, P. 1972. Anaerobic metabolism during activity in lizards. *Journal of Comparative Physiology,* 81**,** 277-288.

Bishop, D. 2010. Dietary supplements and team-sport performance. *Sports Med,* 40**,** 995-1017.

Bishop, D. & Claudius, B. 2005. Effects of induced metabolic alkalosis on prolonged intermittent-sprint performance. *Med Sci Sports Exerc,* 37**,** 759-67.

Bishop, D., Edge, J., Davis, C. & Goodman, C. 2004. Induced metabolic alkalosis affects muscle metabolism and repeated-sprint ability. *Med Sci Sports Exerc,* 36**,** 807-13.

Bogdanis, G. C., Nevill, M. E., Boobis, L. H. & Lakomy, H. K. 1996. Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. *J Appl Physiol,* 80**,** 876-84.

Boning, D., Klarholz, C., Himmelsbach, B., Hutler, M. & Maassen, N. 2007. Extracellular bicarbonate and non-bicarbonate buffering against lactic acid during and after exercise. *Eur J Appl Physiol,* 100**,** 457-67.

Boobis, L., Williams, C. & Wootton, S.A. 1982. Human muscle metabolism during brief maximal exercise. *Journal of Physiology***,** 21-22.

Brooks, G. A. 2007. Lactate: link between glycolytic and oxidative metabolism. *Sports Med,* 37**,** 341-3.

Brooks, G. A. 2010. What does glycolysis make and why is it important? *J Appl Physiol,* 108**,** 1450-1.

Brown, S. P., Miller, W. C., & Eason, J. M. 2006. *Exercise physiology: Basis of human movement in health and disease,* Baltimore, USA, Lippincott, Williams and Wilkins.

Burke, L. 2004. ACSM and MSSE: nutrition and metabolism perspective. *Med Sci Sports Exerc,* 36**,** 179.

Burke, L. M. 2003. The IOC consensus on sports nutrition 2003: new guidelines for nutrition for athletes. *Int J Sport Nutr Exerc Metab,* 13**,** 549-52.

Burke, L. M. & Pyne, D. B. 2007. Bicarbonate loading to enhance training and competitive performance. *Int J Sports Physiol Perform,* 2**,** 93-7.

Calder, A. 1990. Restoration, Regeneration as Essential Components within Training Programs. Australian Institute of Sport.

Cameron, S. L., McLay-Cooke, R. T., Brown, R. C., Gray, A. R. & Fairbairn, K. A. 2010. Increased blood pH but not performance with sodium bicarbonate supplementation in elite rugby union players. *Int J Sport Nutr Exerc Metab,* 20**,** 307-21.

Cannon, W. B. 1932. *The Wisdom of the Body,* New York, Norton.

Carr, A. J., Hopkins, W. G. & Gore, C. J. 2011. Effects of acute alkalosis and acidosis on performance: a meta-analysis. *Sports Med,* 41**,** 801-14.

Carr, A. J., Slater, G. J., Gore, C. J., Dawson, B. & Burke, L. M. 2012. Reliability and Effect of Sodium Bicarbonate: Buffering and 2000-m Rowing Performance. *Int J Sports Physiol Perform,* 7**,** 152-60.

Cheetham, M. E., Boobis, L. H., Brooks, S. & Williams, C. 1986. Human muscle metabolism during sprint running. *J Appl Physiol,* 61**,** 54-60.

Costill, D. L., Verstappen, F., Kuipers, H., Janssen, E. & Fink, W. 1984. Acid-base balance during repeated bouts of exercise: influence of HCO3. *Int J Sports Med,* 5**,** 228-31.

Culbertson, J. Y., Kreider, R. B., Greenwood, M. & Cooke, M. 2010. Effects of beta-alanine on muscle carnosine and exercise performance: a review of the current literature. *Nutrients,* 2**,** 75-98.

Cunniffe, B., Proctor, W., Baker, J. S. & Davies, B. 2009a. An evaluation of the physiological demands of elite rugby union using Global Positioning System tracking software. *J Strength Cond Res,* 23**,** 1195-203.

Cunniffe, B., Proctor, W., Baker, J. S. & Davies, B. 2009b. An Evaluation of the Physiological Demands of Elite Rugby Union Using Global Positioning System Tracking Software. *Journal of Strength & Conditioning Research,* 23**,** 1195-1203.

Dantzer, R. a. K., K.W. 2007. Twenty years of research on cytokine-induced sickness behaviour. *Brain, behaviour and immunity.,* February; 21**,** 153–160.

Dawson, R., Jr., Biasetti, M., Messina, S. & Dominy, J. 2002. The cytoprotective role of taurine in exercise-induced muscle injury. *Amino Acids,* 22**,** 309-24.

Dennig, H., Talbott, J. H., Edwards, H. T. & Dill, D. B. 1931. EFFECT OF ACIDOSIS AND ALKALOSIS UPON CAPACITY FOR WORK. *J Clin Invest,* 9**,** 601-13.

Derave, W., Ozdemir, M. S., Harris, R. C., Pottier, A., Reyngoudt, H., Koppo, K., Wise, J. A. & Achten, E. 2007. beta-Alanine supplementation augments muscle carnosine content and attenuates fatigue during repeated isokinetic contraction bouts in trained sprinters. *J Appl Physiol,* 103**,** 1736-43.

Deutsch, M. U., Kearney, G. A. & Rehrer, N. J. 2007. Time - motion analysis of professional rugby union players during match-play. *J Sports Sci,* 25**,** 461-72.

Deutsch, M. U., Maw, G. J., Jenkins, D. & Reaburn, P. 1998. Heart rate, blood lactate and kinematic data of elite colts (under-19) rugby union players during competition. *J Sports Sci,* 16**,** 561-70.

Dickson, S. L. 1995. Understanding the oxyhemoglobin dissociation curve. *Crit Care Nurse,* 15**,** 54-8.

Douroudos, II, Fatouros, I. G., Gourgoulis, V., Jamurtas, A. Z., Tsitsios, T., Hatzinikolaou, A., Margonis, K., Mavromatidis, K. & Taxildaris, K. 2006. Dose-related effects of prolonged NaHCO3 ingestion during high-intensity exercise. *Med Sci Sports Exerc,* 38**,** 1746-53.

Driller, M. W. a. b., Gregory, J. R. b., Williams, A. D. a. & Fell, J. W. a. The effects of serial and acute NaHCO3 loading in well-trained cyclists. *Journal of Strength & Conditioning Research*.

Duthie, G., Pyne, D. & Hooper, S. 2003. Applied physiology and game analysis of rugby union. *Sports Med,* 33**,** 973-91.

Edge, J., Bishop, D. & Goodman, C. 2006. Effects of chronic NaHCO3 ingestion during interval training on changes to muscle buffer capacity, metabolism, and short-term endurance performance. *J Appl Physiol,* 101**,** 918-25.

Fabiato, A. & Fabiato, F. 1978. Effects of pH on the myofilaments and the sarcoplasmic reticulum of skinned cells from cardiace and skeletal muscles. *J Physiol,* 276**,** 233-55.

Favero, T. G., Zable, A. C., Bowman, M. B., Thompson, A. & Abramson, J. J. 1995. Metabolic end products inhibit sarcoplasmic reticulum Ca2+ release and [3H]ryanodine binding. *J Appl Physiol,* 78**,** 1665-72.

Filley, G. F. 1971. *Acid-Base and Blood Gas Regulation,* Philadelphia, Lea & Febiger.

Fletcher, W. M., & Hopkins, F.G. 1907. Lactic acid in amphibian muscle. *Journal of Physiology, London,* 35**,** 247-303.

Fuller, C. W., Taylor, A. & Molloy, M. G. 2010. Epidemiological study of injuries in international Rugby Sevens. *Clin J Sport Med,* 20**,** 179-84.

Gabbett, T. J. 2006. A comparison of physiological and anthropometric characteristics among playing positions in sub-elite rugby league players. *J Sports Sci,* 24**,** 1273-80.

Gabbett, T. J. 2007. Physiological and anthropometric characteristics of elite women rugby league players. *J Strength Cond Res,* 21**,** 875-81.

Gaitanos, G. C., Nevill, M. E., Brooks, S. & Williams, C. 1991. Repeated bouts of sprint running after induced alkalosis. *J Sports Sci,* 9**,** 355-70.

Gaitanos, G. C., Williams, C., Boobis, L. H. & Brooks, S. 1993. Human muscle metabolism during intermittent maximal exercise. *J Appl Physiol,* 75**,** 712-9.

Gao, J. P., Costill, D. L., Horswill, C. A. & Park, S. H. 1988. Sodium bicarbonate ingestion improves performance in interval swimming. *Eur J Appl Physiol Occup Physiol,* 58**,** 171-4.

George, K. P. & MacLaren, D. P. 1988. The effect of induced alkalosis and acidosis on endurance running at an intensity corresponding to 4 mM blood lactate. *Ergonomics,* 31**,** 1639-45.

Gladden, L. B. 2008. Current trends in lactate metabolism: introduction. *Med Sci Sports Exerc,* 40**,** 475-6.

Glaister, M., Lockey, R. A., Abraham, C. S., Staerck, A., Goodwin, J. E. & McInnes, G. 2006. Creatine supplementation and multiple sprint running performance. *J Strength Cond Res,* 20**,** 273-7.

Goldfinch, J., Mc Naughton, L. & Davies, P. 1988. Induced metabolic alkalosis and its effects on 400-m racing time. *Eur J Appl Physiol Occup Physiol,* 57**,** 45-8.

Gracely, R. H., McGrath, P., & Dubner, R. 1978. Ratio scales of sensory and affective verbal pain descriptors. *Pain,* 5**,** 5-18.

Greenhaff, P. L., Gleeson, M., Whiting, P. H. & Maughan, R. J. 1987. Dietary composition and acid-base status: limiting factors in the performance of maximal exercise in man? *Eur J Appl Physiol Occup Physiol,* 56**,** 444-50.

Guthrie, H. 1983. *Introductory Nutrition,* Mosby, London.

Guyton, A. C., & Hall, J.E. 1996. *Textbook of Medical Physiology,* Philadelphia, USA, W.B. Saunders Company.

Hamlin, M. J., Hinckson, E. A., Wood, M. R. & Hopkins, W. G. 2008. Simulated rugby performance at 1550-m altitude following adaptation to intermittent normobaric hypoxia. *J Sci Med Sport,* 11**,** 593-9.

Harris, R. C., Tallon, M. J., Dunnett, M., Boobis, L., Coakley, J., Kim, H. J., Fallowfield, J. L., Hill, C. A., Sale, C. & Wise, J. A. 2006. The absorption of orally supplied beta-alanine and its effect on muscle carnosine synthesis in human vastus lateralis. *Amino Acids,* 30**,** 279-89.

Harrison, R. A. 1995. Acid-base balance. *Respir Care Clin N Am,* 1**,** 7-21.

Hawley, J. A. & Reilly, T. 1997. Fatigue revisited. *J Sports Sci,* 15**,** 245-6.

Heil, D. P., Jacobson, E. A. & Howe, S. M. 2012. Influence of an alkalizing supplement on markers of endurance performance using a double-blind placebo-controlled design. *J Int Soc Sports Nutr,* 9**,** 8.

Higham, D. G., Pyne, D. B., Anson, J. M. & Eddy, A. 2012. Movement patterns in rugby sevens: Effects of tournament level, fatigue and substitute players. *J Sci Med Sport,* 15**,** 277-82.

Hill, C. A., Harris, R. C., Kim, H. J., Harris, B. D., Sale, C., Boobis, L. H., Kim, C. K. & Wise, J. A. 2007. Influence of beta-alanine supplementation on skeletal muscle carnosine concentrations and high intensity cycling capacity. *Amino Acids,* 32**,** 225-33.

Hollidge-Horvat, M. G., Parolin, M. L., Wong, D., Jones, N. L. & Heigenhauser, G. J. 2000. Effect of induced metabolic alkalosis on human skeletal muscle metabolism during exercise. *Am J Physiol Endocrinol Metab,* 278**,** E316-29.

Hooper, S. L., Mackinnon, L. T., Howard, A., Gordon, R. D. & Bachmann, A. W. 1995. Markers for monitoring overtraining and recovery. *Med Sci Sports Exerc,* 27**,** 106-12.

Horswill, C. A., Costill, D. L., Fink, W. J., Flynn, M. G., Kirwan, J. P., Mitchell, J. B. & Houmard, J. A. 1988. Influence of sodium bicarbonate on sprint performance: relationship to dosage. *Med Sci Sports Exerc,* 20**,** 566-9.

Hulikova, A., Harris, A. L., Vaughan-Jones, R. D. & Swietach, P. 2012. Acid-extrusion from tissue: the interplay between membrane transporters and pH buffers. *Curr Pharm Des,* 18**,** 1331-7.

Hultman, E. S., H. 1983. Energy metabolism and contraction force of human skeletal muscle in situ during electrical stimulation. *Journal of Physiology***,** 525-532.

Ibanez, J., Pullinen, T., Gorostiaga, E., Postigo, A. & Mero, A. 1995. Blood lactate and ammonia in short-term anaerobic work following induced alkalosis. *J Sports Med Phys Fitness,* 35**,** 187-93.

Jensen, F. B. 2004. Red blood cell pH, the Bohr effect, and other oxygenation-linked phenomena in blood O2 and CO2 transport. *Acta Physiol Scand,* 182**,** 215-27.

Johansen, L. & Quistorff, B. 2003. 31P-MRS characterization of sprint and endurance trained athletes. *Int J Sports Med,* 24**,** 183-9.

Jones, N. L., Sutton, J. R., Taylor, R. & Toews, C. J. 1977. Effect of pH on cardiorespiratory and metabolic responses to exercise. *J Appl Physiol,* 43**,** 959-64.

Joyce, S., Minahan, C., Anderson, M. & Osborne, M. 2012. Acute and chronic loading of sodium bicarbonate in highly trained swimmers. *Eur J Appl Physiol,* 112**,** 461-9.

Juel, C. 1998. Muscle pH regulation: role of training. *Acta Physiol Scand,* 162**,** 359-66.

Juel, C. & Pilegaard, H. 1998. Lactate/H+ transport kinetics in rat skeletal muscle related to fibre type and changes in transport capacity. *Pflugers Arch,* 436**,** 560-4.

Kellman, M. 2002. *Enhancing recovery: Preventing underperformance in athletes,* Champaign, IL, Human Kinetics.

Kendrick, I. P., Harris, R. C., Kim, H. J., Kim, C. K., Dang, V. H., Lam, T. Q., Bui, T. T., Smith, M. & Wise, J. A. 2008. The effects of 10 weeks of resistance training combined with beta-alanine supplementation on whole body strength, force production, muscular endurance and body composition. *Amino Acids,* 34**,** 547-54.

Kozak-Collins, K., Burke, E. R. & Schoene, R. B. 1994. Sodium bicarbonate ingestion does not improve performance in women cyclists. *Med Sci Sports Exerc,* 26**,** 1510-5.

Kravitz, L. 2005. Lactate: Not guilty as charged. . *IDEA Fitness Journal,* 2**,** 23-25.

Kuotedakis, S. 1985a. Lactic acid removal and heart rate frequencies during recovery after strenuous rowing exercise. *British Journal of Sports Medicine,* 19**,** 199-202.

Kuotedakis, Y., & Sharp, N.C. 1985b. Lactic acid removal and heart rate frequencies during recovery after strenuous rowing exercise. *British Journal of Sports Medicine,* 19**,** 199-202.

Kupcis, P. D., Slater, G. J., Pruscino, C. L. & Kemp, J. G. 2012. Influence of sodium bicarbonate on performance and hydration in lightweight rowing. *Int J Sports Physiol Perform,* 7**,** 11-8.

Lavender, G. & Bird, S. R. 1989. Effect of sodium bicarbonate ingestion upon repeated sprints. *Br J Sports Med,* 23**,** 41-5.

Linderman, J., Kirk, L., Musselman, J., Dolinar, B. & Fahey, T. D. 1992. The effects of sodium bicarbonate and pyridoxine-alpha-ketoglutarate on short-term maximal exercise capacity. *J Sports Sci,* 10**,** 243-53.

Lindh, A. M., Peyrebrune, M. C., Ingham, S. A., Bailey, D. M. & Folland, J. P. 2008. Sodium bicarbonate improves swimming performance. *Int J Sports Med,* 29**,** 519-23.

M, R. & M, K. Changes in acid-base balance during simulated soccer match-play. *Journal of Strength & Conditioning Research*.

Malucelli E, I. S., Manners DN, Testa C, Martinuzzi A, Barbiroli B, Lodi R. 2011. The role of pH on the thermodynamics and kinetics of muscle biochemistry: an in vivo study by (31)P-MRS in patients with myo-phosphorylase deficiency. *Biochim Biophys Acta.***,** 1244-9.

Martin, N., Zoeller, R.F., Robertson, R.J., & Lephart, S.M. 1998. The comparative effects of sports massage, active recovery, and rest in promoting blood lactate clearance after supramaximal leg exercise. *Journal of Athletic Training,* 33**,** 30-35.

Martini, F. 2006. *Fundamentals of Anatomy and Physiology,* San Francisco, USA, Pearson Education.

Marx, J. O., Gordon, S. E., Vos, N. H., Nindl, B. C., Gomez, A. L., Volek, J. S., Pedro, J., Ratamess, N., Newton, R. U., French, D. N., Rubin, M. R., Hakkinen, K. & Kraemer, W. J. 2002. Effect of alkalosis on plasma epinephrine responses to high intensity cycle exercise in humans. *Eur J Appl Physiol,* 87**,** 72-7.

Matson, L. G. & Tran, Z. V. 1993. Effects of sodium bicarbonate ingestion on anaerobic performance: a meta-analytic review. *Int J Sport Nutr,* 3**,** 2-28.

Matsuura, R., Arimitsu, T., Kimura, T., Yunoki, T. & Yano, T. 2007. Effect of oral administration of sodium bicarbonate on surface EMG activity during repeated cycling sprints. *Eur J Appl Physiol,* 101**,** 409-17.

Maughan, R. J. & Burke, L. M. 2011. Practical nutritional recommendations for the athlete. *Nestle Nutr Inst Workshop Ser,* 69**,** 131-49.

Maughan, R. J., Greenhaff, P. L., Leiper, J. B., Ball, D., Lambert, C. P. & Gleeson, M. 1997. Diet composition and the performance of high-intensity exercise. *J Sports Sci,* 15**,** 265-75.

Mc Naughton, L. & Thompson, D. 2001. Acute versus chronic sodium bicarbonate ingestion and anaerobic work and power output. *J Sports Med Phys Fitness,* 41**,** 456-62.

McArdle, W. D., Katch, F.I., & Katch, V.L. 2010. *Exercise Physiology: Nutrition, Energy and Human Performance,* Baltimore, USA, Lippincott, Williams and Wilkins.

McKenzie, D. C. 1988. Changes in urinary pH following bicarbonate loading. *Can J Sport Sci,* 13**,** 254-6.

McLean, D. A. 1992. Analysis of the physical demands of international rugby union. *J Sports Sci,* 10**,** 285-96.

McNaughton, L., Backx, K., Palmer, G. & Strange, N. 1999. Effects of chronic bicarbonate ingestion on the performance of high-intensity work. *Eur J Appl Physiol Occup Physiol,* 80**,** 333-6.

McNaughton, L. R. 1992a. Bicarbonate ingestion: effects of dosage on 60 s cycle ergometry. *J Sports Sci,* 10**,** 415-23.

McNaughton, L. R. 1992b. Sodium bicarbonate ingestion and its effects on anaerobic exercise of various durations. *J Sports Sci,* 10**,** 425-35.

McNaughton, L. R., Siegler, J. & Midgley, A. 2008. Ergogenic effects of sodium bicarbonate. *Curr Sports Med Rep,* 7**,** 230-6.

Medbo, J. I., Gramvik, P., Jebens, E. 1999. Aerobic and anaerobic energy release during 10 and 30 s bicycle sprints. *Acta Kinesiol Univ Tartuensis***,** 122-46.

Medbo, J. I. & Sejersted, O. M. 1985. Acid-base and electrolyte balance after exhausting exercise in endurance-trained and sprint-trained subjects. *Acta Physiol Scand,* 125**,** 97-109.

Mero, A. A., Keskinen, K. L., Malvela, M. T. & Sallinen, J. M. 2004. Combined creatine and sodium bicarbonate supplementation enhances interval swimming. *J Strength Cond Res,* 18**,** 306-10.

Murphree, R. 2004. Stress: Catalyst for the onset of disease. *Health News,* 2**,** 1-8.

Muthayya, N. M. 2002
Human Physiology, New Dehli, India., Jaypee brothers medical publishers.

Nakamaru, Y. & Schwartz, A. 1970. Possible control of intracellular calcium metabolism by [H+]: sarcoplasmic reticulum of skeletal and cardiac muscle. *Biochem Biophys Res Commun,* 41**,** 830-6.

Nicholas, C. W. 1997. Anthropometric and physiological characteristics of rugby union football players. *Sports Med,* 23**,** 375-96.

Parkhouse, W. S., McKenzie, D. C., Hochachka, P. W. & Ovalle, W. K. 1985. Buffering capacity of deproteinized human vastus lateralis muscle. *J Appl Physiol,* 58**,** 14-7.

Peart, D. J., Siegler, J. C. & Vince, R. V. 2012. Practical Recommendations for Coaches and Athletes: A Meta-Analysis of Sodium Bicarbonate use for Athletic Performance. *J Strength Cond Res*.

Pederson, T. H., Nielson, O.B., Lamb, G.D, & Stephenson, D.G. 2004. Intracellular acidosis enhances the excitability of working muscle. *Science,* 305**,** 1144-1147.

Potteiger, J. A., Webster, M. J., Nickel, G. L., Haub, M. D. & Palmer, R. J. 1996. The effects of buffer ingestion on metabolic factors related to distance running performance. *Eur J Appl Physiol Occup Physiol,* 72**,** 365-71.

Price, M., Moss, P. & Rance, S. 2003. Effects of sodium bicarbonate ingestion on prolonged intermittent exercise. *Med Sci Sports Exerc,* 35**,** 1303-8.

Price, M. J. & Simons, C. 2010. The effect of sodium bicarbonate ingestion on high-intensity intermittent running and subsequent performance. *J Strength Cond Res,* 24**,** 1834-42.

Pruscino, C. L., Ross, M. L., Gregory, J. R., Savage, B. & Flanagan, T. R. 2008. Effects of sodium bicarbonate, caffeine, and their combination on repeated 200-m freestyle performance. *Int J Sport Nutr Exerc Metab,* 18**,** 116-30.

Putnam, R. W. 1979. The role of lactic acid accumulation in muscle fatigue of two species of anurans, Xenopus laevis and Rana pipiens. *Journal of Experimental Biology,* 82**,** 35-51.

Quarrie, K. L., Handcock, P., Toomey, M. J. & Waller, A. E. 1996. The New Zealand rugby injury and performance project. IV. Anthropometric and physical performance comparisons between positional categories of senior A rugby players. *Br J Sports Med,* 30**,** 53-6.

Requena, B., Zabala, M., Padial, P. & Feriche, B. 2005. Sodium bicarbonate and sodium citrate: ergogenic aids? *J Strength Cond Res,* 19**,** 213-24.

Rienzi, E., Reilly, T. & Malkin, C. 1999. Investigation of anthropometric and work-rate profiles of Rugby Sevens players. *J Sports Med Phys Fitness,* 39**,** 160-4.

Riggs, T. E., Shafer, A. W. & Guenter, C. A. 1973. Acute changes in oxyhemoglobin affinity. Effects on oxygen transport and utilization. *J Clin Invest,* 52**,** 2660-3.

Robergs, R., Hutchinson, K., Hendee, S., Madden, S. & Siegler, J. 2005. Influence of pre-exercise acidosis and alkalosis on the kinetics of acid-base recovery following intense exercise. *Int J Sport Nutr Exerc Metab,* 15**,** 59-74.

Roberts, S. P., Stokes, K. A., Weston, L. & Trewartha, G. 2010. The Bath University Rugby Shuttle Test (BURST): a pilot study. *Int J Sports Physiol Perform,* 5**,** 64-74.

Roberts, S. P., Trewartha, G., Higgitt, R. J., El-Abd, J. & Stokes, K. A. 2008. The physical demands of elite English rugby union. *J Sports Sci,* 26**,** 825-33.

Robinson, K., & Verity, L.S. & . 1987. Effect of induced alkalosis on rowing ergometer performance during repeated 1-mile workouts
Medicine and Science in Sports and Exercise, 19.

Sacco, G. M. 2005. *Journal of Behavioural and Neuroscience Research,* Vol. 3

Sale, C., Hill, C. A., Ponte, J. & Harris, R. C. 2012. beta-alanine supplementation improves isometric endurance of the knee extensor muscles. *J Int Soc Sports Nutr,* 9**,** 26.

Sale, C., Saunders, B. & Harris, R. C. 2010. Effect of beta-alanine supplementation on muscle carnosine concentrations and exercise performance. *Amino Acids,* 39**,** 321-33.

Sale, C., Saunders, B., Hudson, S., Wise, J. A., Harris, R. C. & Sunderland, C. D. 2011. Effect of beta-alanine plus sodium bicarbonate on high-intensity cycling capacity. *Med Sci Sports Exerc,* 43**,** 1972-8.

Seyle, H. 1936. A syndrome produced by diverse noxious agents. *Nature,* Nature.

Shephard, R. J. 2005. Chronic Fatigue Syndrome: A brief review of functional disturbances and potential therapy. *Journal of Sports Medicine and Physical Fitness,* 45 381-92.

Siegler, J. C. & Gleadall-Siddall, D. O. 2010. Sodium bicarbonate ingestion and repeated swim sprint performance. *J Strength Cond Res,* 24**,** 3105-11.

Siegler, J. C. & Hirscher, K. 2010. Sodium bicarbonate ingestion and boxing performance. *J Strength Cond Res,* 24**,** 103-8.

Siegler, J. C., Keatley, S., Midgley, A. W., Nevill, A. M. & McNaughton, L. R. 2008. Pre-exercise alkalosis and acid-base recovery. *Int J Sports Med,* 29**,** 545-51.

Siegler, J. C., Marshall, P., Bray, J. & Towlson, C. 2011. Sodium bicarbonate supplementation & ingestion timing: does it matter? *J Strength Cond Res*.

Siegler, J. C., Marshall, P. W. M., Bray, J. & Towlson, C. 2012. Sodium Bicarbonate Supplementation and Ingestion Timing: Does It Matter? *Journal of Strength & Conditioning Research,* 26**,** 1953-1958.

Siegler, J. C., McNaughton, L. R., Midgley, A. W., Keatley, S. & Hillman, A. 2010a. Metabolic alkalosis, recovery and sprint performance. *Int J Sports Med,* 31**,** 797-802.

Siegler, J. C., Midgley, A. W., Polman, R. C. & Lever, R. 2010b. Effects of various sodium bicarbonate loading protocols on the time-dependent extracellular buffering profile. *J Strength Cond Res,* 24**,** 2551-7.

Smith, L. L. 2000. Cytokine hypothesis of overtraining: a physiological adaptation to excessive stress. . *Journal of Medical Science in Sports and Exercise.,* 32**,** 317-331.

Spencer, M., Bishop, D., Dawson, B. & Goodman, C. 2005. Physiological and metabolic responses of repeated-sprint activities:specific to field-based team sports. *Sports Med,* 35**,** 1025-44.

Spriet, L. L., Lindinger, M. I., Heigenhauser, G. J. & Jones, N. L. 1986. Effects of alkalosis on skeletal muscle metabolism and performance during exercise. *Am J Physiol,* 251**,** R833-9.

Spriet, L. L., Lindinger, M. I., McKelvie, R. S., Heigenhauser, G. J. & Jones, N. L. 1989. Muscle glycogenolysis and H+ concentration during maximal intermittent cycling. *J Appl Physiol,* 66**,** 8-13.

Staempfli, H. R. & Constable, P. D. 2003. Experimental determination of net protein charge and A(tot) and K(a) of nonvolatile buffers in human plasma. *J Appl Physiol,* 95**,** 620-30.

Stephens, T. J., McKenna, M. J., Canny, B. J., Snow, R. J. & McConell, G. K. 2002. Effect of sodium bicarbonate on muscle metabolism during intense endurance cycling. *Med Sci Sports Exerc,* 34**,** 614-21.

Stevenson, A., Oliver, A., Roger, C., & Stephen, B.W. Year. Point of care testing of arterial/earlobe capillary blood gases: validation of a portable blood gas analyser. *In:* European Respiratory Society, 2004 Glasgow, UK.

Stone, M. a. S., M. 2003. Recovery-adaptation: strength and power sports. . *Olympic Coach,* 15**,** 12-15.

Suarez-Arrones, L., Nunez, F., Portillo, J. & Mendez-Villanueva, A. 2011a. Match running performance and exercise intensity in elite female Rugby Sevens. *J Strength Cond Res*.

Suarez-Arrones, L. J., Nunez, F. J., Portillo, J. & Mendez-Villanueva, A. 2011b. RUNNING DEMANDS AND HEART RATE RESPONSES IN MEN RUGBY SEVENS. *J Strength Cond Res*.

Sukkar, M. Y., El-Munshid, H.A. and Ardawi, M.S.M. 1994. *Concise Human Physiology,* Oxford, Blackwell Science.

Sutton, J. R., Jones, N. L. & Toews, C. J. 1981. Effect of PH on muscle glycolysis during exercise. *Clin Sci (Lond),* 61**,** 331-8.

Suzuki, Y., Ito, O., Mukai, N., Takahashi, H. & Takamatsu, K. 2002. High level of skeletal muscle carnosine contributes to the latter half of exercise performance during 30-s maximal cycle ergometer sprinting. *Jpn J Physiol,* 52**,** 199-205.

Tallon, M. J., Harris, R. C., Boobis, L. H., Fallowfield, J. L. & Wise, J. A. 2005. The carnosine content of vastus lateralis is elevated in resistance-trained bodybuilders. *J Strength Cond Res,* 19**,** 725-9.

Tan, F., Polglaze, T., Cox, G., Dawson, B., Mujika, I. & Clark, S. 2010. Effects of induced alkalosis on simulated match performance in elite female water polo players. *Int J Sport Nutr Exerc Metab,* 20**,** 198-205.

Thies, R., & Barron, K.W. 1992. *Physiology,* New York, Springer-Verlag.

Trabelsi, K., Rebai, H., El-Abed, K., Stannard, S. R., Khannous, H., Masmoudi, L., Sahnoun, Z., Hakim, A., Fellman, N. & Tabka, Z. 2011. Effect of ramadan fasting on body water status markers after a rugby sevens match. *Asian J Sports Med,* 2**,** 186-94.

Trivedi, B. & Danforth, W. H. 1966. Effect of pH on the kinetics of frog muscle phosphofructokinase. *J Biol Chem,* 241**,** 4110-2.

Ui, M. 1966. A role of phosphofructokinase in pH-dependent regulation of glycolysis. *Biochim Biophys Acta,* 124**,** 310-22.

Verbitsky, O., Mizrahi, J., Levin, M. & Isakov, E. 1997. Effect of ingested sodium bicarbonate on muscle force, fatigue, and recovery. *J Appl Physiol,* 83**,** 333-7.

Wilkes, D., Gledhill, N. & Smyth, R. 1983. Effect of acute induced metabolic alkalosis on 800-m racing time. *Med Sci Sports Exerc,* 15**,** 277-80.

Yoshida, T. & Watari, H. 1993. Changes in intracellular pH during repeated exercise. *Eur J Appl Physiol Occup Physiol,* 67**,** 274-8.

Yquel, R. J., Arsac, L. M., Thiaudiere, E., Canioni, P. & Manier, G. 2002. Effect of creatine supplementation on phosphocreatine resynthesis, inorganic phosphate accumulation and pH during intermittent maximal exercise. *J Sports Sci,* 20**,** 427-37.

Zabala, M., Peinado, A. B., Calderon, F. J., Sampedro, J., Castillo, M. J. & Benito, P. J. 2011. Bicarbonate ingestion has no ergogenic effect on consecutive all out sprint tests in BMX elite cyclists. *Eur J Appl Physiol,* 111**,** 3127-34.

Zabala, M., Requena, B., Sanchez-Munoz, C., Gonzalez-Badillo, J. J., Garcia, I., Oopik, V. & Paasuke, M. 2008. Effects of sodium bicarbonate ingestion on performance and perceptual responses in a laboratory-simulated BMX cycling qualification series. *J Strength Cond Res,* 22**,** 1645-53.

Appendices

Appendix A1

Informed Consent Form

I. **Research Study Title:** The effects of acute versus chronic sodium bicarbonate ($NaHCO_3$) supplementation on high intensity, intermittent sprint performance.
University Department: School of Health and Human Performance
Principal investigator: Dr. Giles Warrington
Other Investigator: Paula Fitzpatrick

II. **Clarification of the purpose of the research**
Ingestion of sodium bicarbonate ($NaHCO_3$), among other buffering substances, has been suggested to delay the onset of fatigue and improve performance during high intensity exercise. However any potential performance enhancement due to $NaHCO_3$ ingestion appears to be highly individual and may involve side affects such as nausea and gastrointestinal discomfort. Therefore it may be beneficial to examine the effects of short-term $NaHCO_3$ loading in the days preceding exercise performance to offset any possible gastro-intestinal issues during performance. The purpose of this study is to investigate $NaHCO_3$ loading and monitor the effects both on performance and on the incidence of gastro-intestinal problems.

III. **Confirmation of particular requirements as highlighted in the Plain Language Statement**
I will be asked to visit Dublin City University (DCU) for testing on five separate days. I will be asked to take part in one of four strategies following a habituation visit:: (1) supplement with $NaHCO_3$ 90 minutes before the performance test, (2) supplement with a placebo 90 minutes before the performance test, (3) supplement with $NaHCO_3$ every day for five days before the performance test, (4) supplement with a placebo every day for five days before the performance test. The performance test will be a high intensity repeat sprint cycle ergometer test. I will also be asked to complete brief questionnaires on muscle soreness and blood samples will also be taken at various intervals throughout the testing process. This entire process must be completed on four separate occasions using a different nutritional strategy each time. Total time requirements on each day will be no more than 120 minutes. There will be no less than one week between each session.

Participant – please complete the following (Circle Yes or No for each question)

Have you read or had read to you the Plain Language Statement	*Yes/No*
Do you understand the information provided?	*Yes/No*
Have you had an opportunity to ask questions and discuss this study?	*Yes/No*
Have you received satisfactory answers to all your questions?	*Yes/No*

IV. **Confirmation that involvement in the Research Study is voluntary**
I understand that I may withdraw from the testing procedures at any time. No penalty will be incurred for failure to complete all stages of the research study.

V. **Advice as to arrangements to be made to protect confidentiality of data, including that confidentiality of information provided is subject to legal limitations**
Confidentiality is an important issue during data collection. My identity, or other personal information, will not be revealed or published. I will be assigned an ID number under which all personal information will be stored in a secure file and saved in password protected file in a computer at DCU. The investigators alone will have access to the data.
Confidentiality of information provided can only be protected within the limitations of the law. It is possible for data to be subject to subpoena, freedom of information claim or mandated reporting by some professions.

VII. **Signature:**
I have read and understood the information in this form. My questions and concerns have been answered by the researchers, and I have a copy of this consent form. Therefore, I consent to take part in this research project

Participants Signature: _____

Name in Block Capitals: _____

Witness: _____

Appendix A2

Informed Consent Form

I. **Research Study Title:** The effects of chronic sodium bicarbonate ($NaHCO_3$) supplementation on simulated rugby sevens specific performance.
University Department: School of Health and Human Performance
Principal investigator: Dr. Giles Warrington
Other Investigator: Paula Fitzpatrick

II. **Clarification of the purpose of the research**
Ingestion of sodium bicarbonate ($NaHCO_3$), among other buffering substances, has been suggested to delay the onset of fatigue and improve performance during high intensity exercise. However any potential performance enhancement due to $NaHCO_3$ ingestion appears to be highly individual and may involve side affects such as nausea and gastrointestinal discomfort. Therefore it may be beneficial to examine the effects of short-term $NaHCO_3$ loading in the days preceding exercise performance to offset any possible gastro-intestinal issues during performance. The purpose of this study is to investigate $NaHCO_3$ loading and monitor the effects both on performance and on the incidence of gastro-intestinal problems.

III. **Confirmation of particular requirements as highlighted in the Plain Language Statement**
I will be asked to visit Dublin City University (DCU) for testing on three separate days. I will be asked to take part in one of two strategies following a habituation visit: (1) supplement with $NaHCO_3$ every day for five days before the performance test, (2) supplement with a placebo every day for five days before the performance test. The performance test will be a rugby sevens specific repeated sprint agility test. I will also be asked to complete brief questionnaires on muscle soreness and blood samples will also be taken at various intervals throughout the testing process. This entire process must be completed on two separate occasions using a different nutritional strategy each time. Total time requirements on each day will be no more than 45 minutes. There will be no less than one week between each session.

Participant – please complete the following (Circle Yes or No for each question)

Have you read or had read to you the Plain Language Statement	*Yes/No*
Do you understand the information provided?	*Yes/No*
Have you had an opportunity to ask questions and discuss this study?	*Yes/No*
Have you received satisfactory answers to all your questions?	*Yes/No*

IV. **Confirmation that involvement in the Research Study is voluntary**
I understand that I may withdraw from the testing procedures at any time. No penalty will be incurred for failure to complete all stages of the research study.

V. **Advice as to arrangements to be made to protect confidentiality of data, including that confidentiality of information provided is subject to legal limitations**
Confidentiality is an important issue during data collection. My identity, or other personal information, will not be revealed or published. I will be assigned an ID number under which all personal information will be stored in a secure file and saved in password protected file in a computer at DCU. The investigators alone will have access to the data.
Confidentiality of information provided can only be protected within the limitations of the law. It is possible for data to be subject to subpoena, freedom of information claim or mandated reporting by some professions.

VII. **Signature:**

I have read and understood the information in this form. My questions and concerns have been answered by the researchers, and I have a copy of this consent form. Therefore, I consent to take part in this research project

Participants Signature: _____

Name in Block Capitals: _____

Witness: _____

Appendix A3

Informed Consent Form

I. **Research Study Title:** The effects of acute sodium bicarbonate ($NaHCO_3$) supplementation on high intensity intermittent performance using a simulated 80 minute rugby specific test protocol.
University Department: School of Health and Human Performance
Principal investigator: Dr. Giles Warrington
Other Investigator: Paula Fitzpatrick

II. **Clarification of the purpose of the research**
Ingestion of sodium bicarbonate ($NaHCO_3$), among other buffering substances, has been suggested to delay the onset of fatigue and improve performance during high intensity exercise. However any potential performance enhancement due to $NaHCO_3$ ingestion appears to be highly individual and may involve side affects such as nausea and gastrointestinal discomfort. Therefore it may be beneficial to examine the effects of short-term $NaHCO_3$ loading in the days preceding exercise performance to offset any possible gastro-intestinal issues during performance. The purpose of this study is to investigate $NaHCO_3$ loading and monitor the effects both on performance and on the incidence of gastro-intestinal problems.

III. **Confirmation of particular requirements as highlighted in the Plain Language Statement**
I will be asked to visit Dublin City University (DCU) for testing on three separate days. I will be asked to take part in one of two strategies following a habituation visit: (1) supplement with $NaHCO_3$ 90 minutes before the performance test, (2) supplement with a placebo 90 minutes before the performance test. The performance test will be a rugby specific repeated sprint agility test. I will also be asked to complete brief questionnaires on muscle soreness and blood samples will also be taken at various intervals throughout the testing process. This entire process must be completed on two separate occasions using a different nutritional strategy each time. Total time requirements on each day will be no more than 180 minutes. There will be no less than one week between each session.

Participant – please complete the following (Circle Yes or No for each question)

Have you read or had read to you the Plain Language Statement	*Yes/No*
Do you understand the information provided?	*Yes/No*
Have you had an opportunity to ask questions and discuss this study?	*Yes/No*
Have you received satisfactory answers to all your questions?	*Yes/No*

IV. **Confirmation that involvement in the Research Study is voluntary**
I understand that I may withdraw from the testing procedures at any time. No penalty will be incurred for failure to complete all stages of the research study.

V. **Advice as to arrangements to be made to protect confidentiality of data, including that confidentiality of information provided is subject to legal limitations**
Confidentiality is an important issue during data collection. My identity, or other personal information, will not be revealed or published. I will be assigned an ID number under which all personal information will be stored in a secure file and saved in password protected file in a computer at DCU. The investigators alone will have access to the data.
Confidentiality of information provided can only be protected within the limitations of the law. It is possible for data to be subject to subpoena, freedom of information claim or mandated reporting by some professions.

VII. **Signature:**

I have read and understood the information in this form. My questions and concerns have been answered by the researchers, and I have a copy of this consent form. Therefore, I consent to take part in this research project

Participants Signature: _____

Name in Block Capitals: _____

Witness: _____

Appendix B

SCHOOL OF HEALTH AND HUMAN PERFORMANCE
DUBLIN CITY UNIVERSITY

General Health Questionnaire

Name:…….. Occupation:……

Address:…….

Telephone: (Home)…….. (Work):……..

Do you have, or have you ever suffered from: -Diabetes? Yes / No
-Asthma? Yes / No
-Epilepsy? Yes / No

Have you ever had pains in your chest or heart? Yes / No

Do you ever feel faint or have spells of dizziness? Yes / No

Do you have or have you ever had high blood pressure? Yes / No

Do you have a muscle, back or joint problem that could be aggravated by physical activity or made worse with exercise? Yes / No

Do you have any current injuries? Yes / No

In the past week, have you suffered from any illness which required you to be in bed or off work for one day or more? Yes / No

Do you smoke? If yes, how many per day? Yes / No

Do you drink? If yes, how many units per week? Yes / No

Is there a good physical reason not mentioned here why you should not carry out laboratory testing? Yes / No

For females only - date of last menstrual period: _____

Please provide any further information concerning any condition/complaints which you suffer from and any medication which you may be taking by prescription or otherwise:

……

Date:_____Signature:_____

Authorising Signature: _____

Appendix C

Muscle Soreness Scale

0 A complete absence of pain
1 Extremely weak pain
2 Weak/Distracting pain
3 Mild/Annoying pain
4 Slightly moderate pain/Uncomfortable
5 Moderate pain/Irritating
6 Barely Strong pain/Distressing
7 Strong pain/Agonizing
8 Intense pain/Intolerable
9 Very Intense pain/Unbearable
10 Extremely Intense pain/Excruciating