# The Role of Lactate Supplementation and Metabolism in Invasion Sport Performance


A thesis submitted for the degree of Doctor of Philosophy (PhD)

by

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# Declaration

Candidate's declarations:

I, Graham Alexander Thom, hereby certify that this thesis submitted in partial fulfilment of the requirements for the award of Doctor of Philosophy (PhD), Abertay University, is wholly my own work unless otherwise referenced or acknowledged. This work has not been submitted for any other qualification at any other academic institution.

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Date 01/09/2020

Supervisor's declaration:

I, Dr John Babraj hereby certify that the candidate has fulfilled the conditions of the Resolution and Regulations appropriate for the degree of Doctor of Philosophy in Abertay University and that the candidate is qualified to submit this thesis in application for that degree.

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# Certificate of Approval

I certify that this is a true and accurate version of the thesis approved by the examiners, and that all relevant ordinance regulations have been fulfilled.

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# Abstract

Lactate produced during exercise is no longer thought of as a waste product and it is now understood it plays an important role in exercise metabolism. Exercise intensity during invasion sports elicits a mean heart rate of approximately 85% $HR_{max}$ which equates to 80% $\dot{V}O_{2max}$ and blood lactate concentration greater than 4mmol·$l^{-1}$. It is likely that an athlete's ability to metabolise this lactate and maintain a lactate balance is a key determining factor in match performance. Supplemented lactate is utilised more rapidly and to a greater extent than glucose and helps maintain performance level for the repeated high intensity actions required throughout invasion sport match play.

Study 1 examined the effect of supplementing calcium lactate during simulated rugby union match play to determine whether sprint speed and intermittent sprint performance could be improved. Ten recreationally active participants were recruited for part one of the study to determine the rate at which orally consumed lactate solution appears in the blood. For part two of the study, seven amateur rugby union players (age: 25 ± 5 years, weight: 84 ± 6kg, and height: 180 ± 4cm) underwent the BURST under three conditions to compare water, glucose, and lactate. Sprint performance was measured through 17 x 15m maximal sprints over the course of the BURST. An increase in blood lactate concentration ($[La^-]b$) was evident within 10 minutes of ingestion and it was significantly ($p \leq 0.05$) elevated from 20 to 60 minutes post ingestion. There was no change in blood glucose concentration throughout the testing period. There was no significant ($p > 0.05$) difference in sprint time between conditions at any performance test, with total sprint time per block, per half, and across the full BURST protocol also showing no difference. There was no significant difference in decrement of sprint performance between the first and second ($p = 0.69$), or third and fourth ($p = 0.13$) exercise blocks, or between the first and second halves ($p = 0.59$). There was a significant difference in peak heart rate between condition in both the first (C = 179 ± 4, L = 172 ± 6, G = 179 ± 9; $p = 0.04$) and second (C= 178 ± 3 L = 170 ± 7, G = 176 ± 6; $p = 0.03$) halves of simulated match play. There was no significant difference in respiratory rate

between conditions in either the first (C = 44 ± 8, L = 38 ± 6, G = 47 ± 11; p =0.16) or second (C = 42 ± 17, L = 36 ± 6, G = 40 ± 19; p = 0.68) halves. Supplementation of 1% Wt/vol calcium lactate solution did not enhance sprint speed or significantly reduce the drop in sprint performance seen throughout a match. There was a trend for the decrement in performance to be less in the lactate condition and therefore, the use of calcium lactate may be recommended prior to rugby matches as an ergogenic aid to sustain sprint performance although any benefit is likely to be marginal.

Study 2 investigated the effect of sprint interval training on soccer specific performance indices and lactate kinetics. In youth soccer, 23% of the distance covered happens at speeds above maximal lactate steady state (MLSS) which suggests lactate kinetics may be important to soccer performance. Thirteen elite soccer academy players (age 15 ± 0.5y) underwent baseline testing (Wingate anaerobic Test (WAnT) with blood lactate measurements, incremental V̇O2peak and time to exhaustion test, 0-10m and 10-20m sprint performance, repeated 20m sprint performance, and vertical jump performance) before being allocated to control or SIT group. The control group maintained training whilst the SIT group carried out twice-weekly all-out effort cycle sprints consisting of 6 x 10s sprint with 80s recovery. Training elicited significant improvements in $\dot{V}O_{2peak}$ (pre: 54.89 ± 3.09ml· $kg^{-1}$·$min^{-1}$ post: 60.81 ± 5.73ml· $kg^{-1}$·$min^{-1}$; p = 0.001), TTE (pre: 655 ± 54s post: 688 ± 55s; p=0.001), 10 - 20m sprint time (pre: 1.29 ± 0.04s post: 1.25 ± .04s; p=0.02), and peak power during WAnT (pre: 12.4 ± 1.3 $W.kg^{-1}$ post: 15.3 ± 0.7$W.kg^{-1}$; p=0.003) which were not seen in the control group. The changes in performance were significantly correlated to changes in lactate kinetics (time to exhaustion: r=0.77, p = 0.04). Cycle-based SIT is an effective training paradigm for elite youth soccer players and the improvements in match specific fitness indices are associated with changes in lactate kinetics. These increased levels of lactate utilisation may facilitate a greater ergogenic benefit from supplemented lactate solution further enhancing its ability to mitigate the decline in sprint performance seen throughout invasion sport match play.

Study 3 examined the effect of calcium lactate supplementation of laboratory-based performance tests specific to field hockey. It also sought to determine whether lactate kinetic altering sprint interval training would enhance any ergogenic effect of the supplementation. Invasion sports such as field hockey require players to perform multiple repetitions of maximal effort sprints, with the volume and intensity of these playing a critical role in determining level of match performance. The drop in sprint performance over the duration of match play is linked to reduced substrate availability leading to fatigue, and there a various ergogenic aids used to mitigate this. One such supplement is lactate which has been shown to possibly maintain high-intensity exercise performance with the effect being amplified as cardiorespiratory fitness increases. Eleven amateur female field hockey players underwent baseline testing for CP, $\dot{V}O_{2peak}$, TTE, PP and RSA both with and without supplemented calcium lactate solution. This was followed by a control period where normal training and match play were maintained. Testing was then repeated before and after carrying out twice-weekly all-out effort cycle sprints consisting of 6 × 10 s sprint with 80 s recovery. There were no differences in performance between either condition at the baseline testing or following the control period ($p > 0.05$).

There was no change in any performance indicator following three-week control period ($p > 0.05$). Training elicited significant improvements in CP (Pre 2: 210 ± 2W; Post: 222 ± 25W, $p = 0.01$), $\dot{V}O_{2peak}$ (Pre 2: 40.27 ± 6.04 ml· $kg^{-1}$·$min^{-1}$; Post: 44.52 ± 4.11 ml· $kg^{-1}$·$min^{-1}$, $p = 0.02$), and TTE (Pre 2: 613 ± 99s; Post: 677 ± 118s, $p = 0.01$). There were no changes in either power at LT (Pre 2: 118 ± 21W; Post: 118 ± 18W, $p = 1.00$), or $[La^-]b$ at LT (Pre 2: 3.15 ± 0.64 mmol·$l^{-1}$; Post: 2.91 ± 0.47 mmol·$l^{-1}$, $p = 0.17$). With the exception of TTE ($p = 0.003$) which saw a significant detrimental effect of lactate supplementation, there were no differences between condition during post-training testing ($p > 0.05$). It is evident performing cycling-based SIT training twice-weekly in addition to the regular field hockey training sessions can help players develop aerobic and anaerobic capacity in a short period of time. It would appear supplementation of a 2% calcium lactate solution offers no ergogenic benefit for short-duration performance tests and may in fact have a detrimental effect on endurance capacity.

Together, these studies show that supplementation of calcium lactate solution is not an effective method to enhance physical performance during aerobic and anaerobic performance tests. It may provide a small mitigation to the decline in sprint performance seen over the course of invasion sport match play. Therefore, the use of calcium lactate could be recommended prior to invasion sport to help sustain sprint performance which is a critical component of match success. Although any benefit seen would be small, at an elite level these small changes may enhance overall team performance giving an advantage over oppositions.

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# List of Abbreviations

| | |
|---|---|
| A | Extravascular release of lactate from exercise metabolism |
| Acetyl-CoA | Acetyl coenzyme A |
| ADP | Adenosine diphosphate |
| ANOVA | Analysis of variance |
| AP | Average power output |
| ATP | Adenosine triphosphate |
| BIA | Bioelectrical impedance analysis |
| BURST | Bath University rugby shuttle test |
| C | Control condition |
| $Ca^{2+}$ | Calcium ion |
| CaMKII | Calmodulin-dependent protein kinase II |
| CG | Control group |
| CHO | Carbohydrate |
| CI | Confidence interval |
| CK | Creatine kinase |
| $CK_{mito}$ | Mitochondrial creatine kinase |
| CMJ | Counter movement jump |
| $CO_2$ | Carbon dioxide |
| COX | Cytochrome *c* oxidase |
| CP | Critical power |
| Cr | Creatine |
| CS | Citrate synthase |
| CV | Coefficient of variation |
| ETC | Electron transport chain |
| ES | Effect size |
| FAD | Flavin adenine dinucleotide |
| $FADH_2$ | Reduced form of FAD |
| G | Glucose condition |
| GPS | Global positioning system |
| GS | Glycogen synthase |

| | |
|---|---|
| GLUT-4 | Glucose transporter-4 |
| $H^+$ | Hydrogen ion |
| HIT | High intensity training |
| HIIT | High intensity interval training |
| HKII | Hexokinase II |
| HR | Heart rate |
| $HR_{max}$ | Maximum heart rate |
| IMF | Intermyofibrillar |
| INTRA | Intramyofibrillar |
| $k_1$ | Rate of lactate accumulation |
| $k_2$ | Rate of lactate clearance |
| L | Lactate condition |
| $La^-$ | Lactate anions |
| $[La^-]b$ | Blood lactate concentration |
| $[La^-]b_{max}$ | Maximum blood lactate concentration |
| LDH | Lactate dehydrogenase |
| LT | Lactate threshold |
| MCT | Monocarboxylate transporter |
| MICT | Moderate intensity continuous training |
| MLSS | Maximal lactate steady state |
| mRNA | Messenger ribonucleic acid |
| N | No lactate condition |
| $NAD^+$ | Nicotinamide adenine dinucleotide |
| NADH | Reduced form of $NAD^+$ |
| $O_2$ | Oxygen |
| PAR-Q | Physical activity readiness questionnaire |
| $PCO_2$ | Partial pressure of carbon dioxide |
| PCr | Phosphocreatine |
| PDH | Pyruvate dehydrogenase |
| PFK | Phosphofructokinase |
| PGC-1α | Peroxisome proliferator-activated receptor γ coactivator 1α |
| pH | A measure of acidity/alkalinity |
| $P_i$ | Inorganic phosphate |

| | |
|---|---|
| $PO_2$ | Partial pressure of oxygen |
| PP | Peak power output |
| PT | Performance test |
| Q | Cardiac output |
| $Q_{max}$ | Maximal cardiac output |
| RCT | Randomised controlled trial |
| RER | Respiratory exchange ratio |
| ROS | Reactive oxygen species |
| RR | Respiratory rate |
| RSA | Repeated sprint ability |
| SD | Standard deviation |
| SE | Standard error |
| SG | Sprint interval training group |
| SIT | Sprint interval training |
| SR | Sarcoplasmic reticulum |
| SS | Subsarcolemmal |
| SSG | Small sided games |
| SV | Stroke volume |
| $T[La^-]b_{max}$ | Time to reach maximum blood lactate concentration |
| TCA cycle | Tricarboxylic acid cycle |
| TP | Turn point |
| TT | Time trial |
| TTE | Time to exhaustion |
| TW | Total work |
| VJ | Vertical jump |
| $\dot{V}O_2$ | Oxygen uptake |
| $\dot{V}O_{2max}$ | Maximal oxygen uptake |
| $\dot{V}O_{2peak}$ | Peak oxygen uptake |
| W’ | Anaerobic work capacity |
| WAnT | Wingate anaerobic test |

# Chapter 1

# General Introduction

## 1.1 Research topic

### 1.1.1 Invasion sport

Invasion sports such as rugby union, soccer, and field hockey require players to be highly dynamic and demonstrate several different physical capabilities throughout match play (Polglaze et al., 2017). These sports involve prolonged low to moderate intensity physical activity interspersed with repeated bouts of high-intensity efforts of high-speed running and sprinting (Drust, Atkinson and Reilly, 2007; Faiss, Girard and Millet, 2013; Gabbett, Jenkins and Abernethy, 2012; Kotzamanidis et al., 2005; Lemos et al., 2017; Paul, Bradley and Nassis, 2015; Shojaeian, Moghadasi and Azizi, 2014; Wehbe, Hartwig and Duncan, 2014; Wisbey et al., 2010).

The ability to repeatedly perform sprinting actions either with (intermittent sprint exercise) or without (repeated sprint exercise) complete recovery throughout match play is an integral shared component of invasion sports and is argued to be a decisive element of success (Bishop et al., 2011; Faiss, Girard and Millet, 2013; Gabbett, 2010b; Girard, Mendez-Villanueva and Bishop, 2011; Girard, Brocherie and Millet, 2015; Spencer et al., 2004; Swaby, Jones et al., 2016). The frequency of these high-intensity bouts vary with work to rest ratio ranging from ~1:1 during the most intense periods of play to ~1:14 during less demanding periods (Cahill, Lamb et al., 2013; McLean, 1992; Randers et al., 2010; Swaby, Jones and Comfort, 2016; White and MacFarlane, 2013).

Invasion sport matches sometimes exceed 100 minutes, and players will fatigue as the match progresses (Mohr et al., 2010). Fatigue is defined as the skeletal muscles' capacity to generate force being diminished and importantly, is denoted by the point of decreased maximal force production, not the point of exhaustion or task failure (Allen and Westerblad, 2001; Enoka and Duchateau, 2008; Decorte et al., 2012). There are several mechanisms leading to muscular fatigue such as diminished phosphocreatine (PCr) availability to replenish adenosine triphosphate (ATP) levels (Wan et al., 2017), along with a concomitant increase in intracellular inorganic phosphate (Debold et al., 2016) and inhibition of calcium release from the sarcoplasmic reticulum (Allen, Lamb

and Westerblad, 2016). During invasion sport match play, muscle glycogen depletion is cited as the main factor of fatigue (Mohr, Krustrup and Bangsbo, 2005), and this manifests as a reduction in the number of sprints and high intensity actions (Aslan et al., 2012; Harley et al., 2010; Krustrup et al., 2006; Lovell et al., 2009; MacLeod, Bussell and Sunderland, 2007; Mendez-Villanueva et al., 2012; Stolen et al., 2005; Tee, Lambert and Coopoo, 2017), along with a reduction in total distance covered and the relative intensity of match play (Cunniffe et al., 2009; Rampinini et al., 2009; Rebelo et al., 2014; Roberts et al., 2008).

### 1.1.2 Supplementation

Differences between winning and losing teams can be very small in invasion sports (Schoeman and Coetzee, 2014) and teams look to enhance their chance of winning through increasing their level of performance (Gudmundsson and Horton, 2016). One of these methods is the use of ergogenic aids to improve performance or mitigate fatigue and is of interest to athletes, coaches and sports scientists (Glaister et al., 2018). Energy supply during invasion sports comes primarily in the form of stored muscle glycogen and blood glucose (Baker et al., 2015), and is an important aspect of maintaining performance (Azevedo et al., 2007). As matches progress, performance is seen to drop in the latter stages (Stolen et al., 2005) due to depletion of muscle glycogen leading to fatigue (Mohr, Krustrup and Bangsbo, 2005). It is therefore important to ensure an adequate supply of energy for athletes to maintain sprint performance throughout a match.

Many studies have been conducted with invasion sport athletes with the aim of enhancing sporting performance through supplementation including endurance capacity, and repeated and intermittent sprinting. Examples include, but are not limited to, caffeine (Carr et al., 2008), glucose (Lee et al., 2011), nitrate (Wylie et al., 2013), L-arginine (Birol et al., 2019) and sodium bicarbonate (Miller et al., 2016), the effects of which are explored in more detail in Section 2.4 of the literature review. One supplement which has received little attention is lactate, and to date there have been no studies investigating its use as an ergogenic aid to maintain or enhance invasion sport specific performance. Within the limited

literature available, lactate has been shown to be an effective ergogenic aid in some types of exercise (Azevedo et al., 2007; Morris et al., 2011, Northgraves et al., 2013; van Montfoort et al., 2004) although results are conflicting (Oliveira et al., 2017; Painelli et al., 2014; Swensen et al., 1994). Proposed mechanisms for this ergogenic effect include the preservation of muscle glycogen and blood glucose for use in later stages of exercise (Bryner et al., 1998; Fahey et al., 1991; Swensen et al., 1994) in a similar way to glucose supplementation (Nicholas et al., 1999; Russell, Benton and Kingsley, 2012). Another explanation is that the supplemented lactate activates aerobic metabolism by simulating the lactate produced endogenously during high intensity exercise (Gurd et al., 2005; Gurd et al., 2006) allowing more rapid resynthesis of PCr. This would benefit the invasion sport athlete as more rapid PCr regeneration is associated with a greater number of sprints and high intensity actions (Bishop and Edge, 2006; Redkva et al., 2018).

### 1.1.3 Lactate

Lactate produced through anaerobic glycolysis was historically labelled as a metabolic waste product, and it was thought to be responsible for the fatigue experienced during high intensity exercise (Azevedo et al., 2007; Gladden, 2004; Gollnick, Bayly and Hodgson, 1986; MacLaren and Morton, 2012; Powers and Howley, 2012). It is now clear however that lactate produced during anaerobic glycolysis plays an important role as an energy substrate (Bryner et al., 1998; Ellis, Simmons and Miller, 2009; Fahey et al., 1991; Gladden, 1991, 2000; Godfrey et al., 2009; Hamann, Kelley and Gladen, 2001; Handy, 2006; Hubbard, 1973; Jeppesen et al., 2013; Kelley et al., 2002; Swensen et al., 1994; van Hall et al., 2003). At rest and during exercise, lactate is predominantly produced and consumed in skeletal muscle (Bergman et al., 1999; Brooks et al., 1991; Donovan and Brooks, 1983; Stanley et al., 1986). It is produced continuously (Gladden, 2000) but levels remain low at rest or during low intensity exercise (Gollnick, Bayly and Hodgson, 1986). As exercise intensity increases, rate of anaerobic glycolysis also increases to meet the demand for ATP within the working muscles. The rate of lactate release becomes more evident at exercise intensities ≥85% maximal oxygen uptake ($\dot{V}O_{2max}$) with

blood lactate concentrations ([La$^-$]b) increasing exponentially in line with glycolysis (Brooks et al., 1991; Donovan and Brooks, 1983; Gladden, 2000). During invasion sport match play, the glycolytic reliant, repeated high intensity bouts are accompanied by elevated intra- and extracellular lactate concentrations. This lactate is rapidly transported and utilised either within the same muscle which produced it, or at another site in the body (Brooks, 1998; Brooks, 2002). These lactate shuttles are facilitated by specialist monocarboxylate transporter (MCT) proteins (Juel and Halestrap, 1999; Pilegaard et al., 1999) the expression of which can be enhanced through training (Baker, McCullagh and Bonen, 1998; Bonen et al., 1998; Juel et al., 2004; Thomas et al., 2005) thereby increasing the rate lactate is transported and utilised (Burgomaster et al., 2007; Bosquet et al., 2003; Holloway, Bliss and Hearon, 2017; van Hall et al., 2003).

### 1.1.4 Sprint interval training

This change in lactate kinetics is associated with increases in endurance capacity (Bergman et al., 1999) and has been reported following both moderate-intensity continuous training (MICT) (Messonier et al., 2006), and high-intensity interval training (HIIT) (Best et al., 2013; Gharbi, 2008; Jakeman, Adamson and Babraj, 2012). However, the intermittent training was shown to be more effective at increasing the rate of lactate clearance than continuous training (Gharbi et al., 2010). Following training which elevates levels of MCT1 and MCT4 (Pilegaard et al., 1999b), there is a rightward shift of the blood lactate curve during high-intensity exercise (Best et al., 2013; Jakeman, Adamson and Babraj, 2012) which is associated with increased exercise duration at high intensities (Messonnier et al., 2001, 2002). There has been no research to date investigating the effects of sprint interval training on lactate kinetics in adolescent invasion sport athletes.

## 1.2 Aims and hypotheses

It is clearly important for athletes, coaches, and practitioners to attempt to improve performance in invasion sports through maintenance or enhancement of the high-intensity actions critical to success. There is evidence to suggest the supplementation of lactate can enhance performance in various physical performance tests but there is currently no literature available on the ergogenic effects of supplementing lactate in an invasion sport specific context. Similarly, to date there has been no investigation on the effect of sprint interval training to improve lactate kinetics in invasion sport athletes and whether an improvement in this area would enhance the ergogenic effects of lactate supplementation. Therefore, the overarching aim of this PhD thesis is to determine the role of lactate supplementation and sprint interval training on performance indicators in invasion sports athletes. Collectively, the work will add to the current understanding of ergogenic aids to enhance invasion sport performance and the role lactate kinetics and metabolism play in this area.

The aims of this thesis are to determine whether, for invasion sport athletes;
i) supplementing lactate solution will preserve or enhance sprint performance to a greater degree than either a traditional glucose-based sports drink or water alone.
ii) sprint interval training is effective at increasing the rate at which lactate is transported and metabolised following high-intensity exercise.
iii) performance will increase with supplemented lactate after this type of training.

The main hypotheses are that;
i) supplemented lactate solution will lead to enhanced sprint speed and a greater preservation of sprint performance during intermittent high-intensity activity.
ii) SIT training will improve the ability to utilise lactate and improve related performance indicators for invasion sport athletes.
iii) invasion sport athletes will demonstrate greater power and endurance capabilities through ingestion of a lactate supplement following SIT.

## 1.3 Research design

To realise these aims, three experimental chapters are presented to test the hypotheses proposed. In studies 1 and 3, a randomised crossover design was employed which is seen as equivalent to the gold standard randomised controlled trial (RCT). Participants acting as their own control helps mitigate some potential inter-participant confounders, and sufficient statistical power can be achieved with fewer participants. The random order supplementation group was assigned during testing also helps limit the possibility of Type I or II errors through a learning effect as trials progress. Both these studies sought to determine the effect of lactate supplementation as an ergogenic aid in invasion sport performance with study 3 additionally investigating the change in this effect following SIT. In study 1, participants were amateur male rugby union players and in study three they were amateur female field hockey players. Study 2 employed a quasi-experimental design. This is similar to the traditional experimental design, but participants are not randomly allocated to intervention or control groups. In study 2, a true RCT model could not be used as this would be undesirable in a club environment so instead a cluster randomised control was used with elite level adolescent male players from the same age grade squad in different playing seasons assigned either training or control group. The study sought to determine the effect of SIT on lactate kinetics and performance indices in soccer.

## 1.4 Outline of thesis

The thesis is constructed as follows; Literature Review (Chapter 2), a General Methods (Chapter 3), three experimental studies (Chapter 4, 5, and 6), and a General Discussion (Chapter 7).

### 1.4.1 Chapter 2

This chapter provides an overview of the process used to identify relevant literature and how this was kept updated throughout the research period. It explores the current literature on; the physiological demands of invasion sports, the metabolic pathways for energy production and their role during high-

intensity exercise and invasion sport, acute supplementation of ergogenic aids with a specific focus on the use of lactate for this purpose, physiological and metabolic adaptations following MICT and HIIT and how this benefits invasion sport performance.

### 1.4.2 Chapter 3

This chapter details the methodology for each of the study chapters and provides a rationale for the procedures and testing employed. It also highlights the approach and rationale for the statistical testing used to analyse the data from each study.

### 1.4.3 Chapter 4

Supplementation has been used in invasion sports to enhance both maximal sprint performance and intermittent and repeated sprint ability. This study investigates the effect of supplementing calcium lactate before and during simulated rugby union match play in amateur adult male players. Performance was assessed through 17 x 15m sprints evenly spread throughout the protocol. Conditions were compared on time spent sprinting in each test and accumulative sprint times. Decline in sprint performance over the duration of the protocol was determined as a percentage increase in time from the baseline sprint.

### 1.4.4 Chapter 5

SIT has been employed to enhance performance indices in invasion sports and training has been shown to alter lactate kinetics. This study investigates the effect of a six-week SIT training intervention in adolescent soccer players. The effect of training was measured for both aerobic and anaerobic performance measures including peak oxygen uptake ($\dot{V}O_{2peak}$), time to exhaustion, Wingate Anaerobic Test (WAnT), 20m sprint speed, repeated sprint ability, and vertical jump performance. Changes in lactate kinetics were measured through extravascular release of lactate, rate of lactate accumulation, rate of lactate clearance, maximum blood lactate concentration, and time taken to reach maximum blood lactate concentration.

### 1.4.5 Chapter 6

Evidence suggests the usefulness of lactate as an ergogenic supplement may be enhanced as cardiorespiratory fitness increases. This study investigated the effect of supplementing calcium lactate on performance indices in amateur female field hockey players. The testing battery included peak oxygen uptake ($\dot{V}O_{2peak}$), time to exhaustion, critical power, and repeated sprint ability. A three-week SIT intervention was undertaken by the participants before repeating the testing to establish whether changes in aerobic and anaerobic performance would alter the magnitude of the ergogenic effect of supplemented lactate.

### 1.4.6 Chapter 7

This chapter provides a general discussion of the thesis. It discusses the main findings of the three studies and how they meet the aims and hypotheses of the thesis. It also provides an oversight of the practical implications of supplementation of lactate and training to alter lactate kinetics, potential limitations within the thesis, and recommendations for the direction of future research in this area.

# Chapter 2

# Literature Review

## 2.1 Review process

Electronic database searches were performed using MEDLINE, PubMed, SPORTDiscus, Web of Science, CINAHL and Google Scholar. Initial review of the literature was conducted using available records from these sources in the English language up to 31 August 2012. Searches were repeated biannually throughout the duration of the thesis to ensure new publications were captured and the review contained the most up to date research as suggested in previous literature (Moher et al., 2008). The Cochrane Handbook suggests setting date limits from the time of the previous search when conducting an update (Bramer and Bain, 2017). However, there may be a difference in the time an article is published to when it becomes available on a database, so it is advisable an overlap period was used to mitigate this delay (Bramer and Bain, 2017), and this was set at two calendar years prior to the year the review was conducted. Searches were also carried out for available records on the databases when a new search term was introduced as the thesis progressed and new concepts or methods were introduced to ensure all relevant literature was captured in the search (Bramer and Bain, 2017; Garner et al., 2016). Reference lists from relevant sources were also examined for additional literature along with using forward citation tracking in Google Scholar.

The search terms covered the physiological demands of invasion sports, supplementation of lactate and other ergogenic aids, high-intensity interval training, repeated and intermittent sprint exercise, and exercise metabolism. Key words included; lactate, supplementation, sprint, invasion/team sports, performance, interval training, adaptations, lactate kinetics.

Where possible, the participants described in the literature were matched to age, ability, and sport specificity of the participants recruited within this thesis. Where no literature provided an exact match, this was stated, and conclusions were made based on the evidence available.

## 2.2 Sports science: research to practice for invasion sports

Within the parameters of the invasion sport environment, the difference between winning and losing is often small (Richards, Collins and Mascarenhas, 2012) and teams look to enhance their chance of winning by attempting to increase their level of performance (Gudmundsson and Horton, 2016). Across invasions sports, there are common attributes which are associated with good performance and small changes in these capacities can have a large effect on match performance and outcome (Comfort, Haigh and Matthews, 2012). These indices of performance are also normally seen to be more highly developed in elite athletes compared to amateur level players (Elferink-Gemser et al., 2006; Lacome et al., 2014; Roberts et al., 2008; Taylor et al., 2017). One of the most important of these capacities is the ability to repeatedly perform high-intensity or sprinting actions due to its influence on match outcomes (Austin, Gabbett and Jenkins, 2011a; Bishop et al., 2006, 2011; Gabbett, 2010b; Girard, Brocherie and Millet, 2015; Rampinini et al., 2007). To meet this demand, players must develop several physical attributes such as maximal running speed (Little and Williams, 2005), repeated sprint ability (RSA) (da Silva, Guglielmo and Bishop, 2010), and aerobic capacity (Reilly et al., 2000) and sports science research can add valuable knowledge on how to improve these capacities. For example, increasing maximal running speed through training equates to higher running speeds during match play (Al Haddad et al., 2015). There are also statistically significant ($p \leq 0.05$) correlations between back squat one repetition max (1RM) and short distance (~10-40m) sprint times (McBride et al., 2009; Styles, Matthews and Comfort, 2016; Wisløff et al., 2003) and increased muscular strength was associated with increased maximum running speed (Peñailillo et al., 2016). Care is required when programming training though as increased strength has also been associated with decreased change of direction speed in invasion sport athletes (Freitas et al., 2019). Despite this, maximal strength is also significantly positively correlated to power output such as vertical jump performance ($r = 0.78$, $p = 0.02$) (Wisløff et al., 2003) which is another indicator of performance during invasion sport (Arnason et al., 2004; Cunningham et al., 2018; Reilly and Borrie, 1992). It has been demonstrated there are significant correlations ($p = 0.02$) between sprint speed and vertical jump performance in

invasion sport athletes (Köklü et al., 2015). The Wingate anaerobic test (WAnT) is considered the gold standard for assessing anaerobic power in sports science laboratories (Herbert et al., 2015) and WAnT peak power and average power have both been shown to significantly correlate ($r = 0.59$ and $0.76$, $p \leq 0.05$) with vertical jump performance (Hoffman et al., 2000). However, there are only moderate correlations found between WAnT scores and sprint speed (Tharp et al., 1985). Training interventions which have been shown to significantly increase peak power during the WAnT have also reported no change in either running speed or vertical jump performance (Kelly et al., 2021) indicating some adaptations seen in a laboratory environment may not translate to a match environment. WAnT performance also does not correlate well to RSA (Aziz and Chuan, 2004) which is positively associated with match play performance in terms of high intensity running actions (da Silva, Guglielmo and Bishop, 2010; Gibson et al., 2013; Rampinini et al., 2007). Aerobic capacity is another performance indicator linked to performance in invasion sport and there is a strong correlation between aerobic capacity and high intensity running during matches ($r = 0.77$, $p = 0.001$) (Castagna et al., 2009; Moir, Krustrup and Bangsbo, 2003). Research interventions employing high intensity interval training can significantly increase maximal oxygen uptake in invasion sport athletes and this directly translates to match performance with significant increases in total distance covered, number of sprints, and average work intensity (Helgerud et al., 2001).

There are also many research projects which investigate training and competition separately so may lack a true reflection of the demands of the sport (Gómez-Carmona et al., 2020). For example, an intervention which found increased maximal and average running speed in invasion sport athletes during repeated sprint testing also then reported no change in these factors during simulated match play (Lara et al., 2014) suggesting care must be taken when transferring laboratory generated knowledge to practical application and reinforces the need for both situations to be tested before practical recommendations can be made. Overall invasion sport performance is also influenced by a wide range of interconnected factors outwith individual

physiological markers (D'Isanto et al., 2019). Factors for success included the efficient and correct individual and team decision making skills (Richards, Collins and Mascarenhas, 2012) and appropriate execution of team organisation and strategy (Lamas et al., 2014). There are also several psychological factors which influence team performance such as motor imagery (Mizuguchi et al., 2012) and goal setting (Weinberg, Stitcher and Richardson, 1994).

Determining the precise demands of invasion sport can also be problematic for sports scientists. While portable gas analysers can be used to assess oxygen uptake during invasion sports, their use within contact sports is limited and sport scientists are reliant on information gathered during simulated match play. Similarly, to determine the contribution of anaerobic metabolism during sport, blood lactate levels are recorded (Padulo et al., 2015). However, measurement of blood lactate during competitive matches is restricted to natural breaks in play (Deutsch et al., 1998). These breaks tend to occur after phases of intense play such as following scoring and therefore may lead to more greatly elevated lactate concentrations.

Knowledge translation/transfer also forms an important aspect of increasing the fit between the research knowledge base and application in the field, and often research funding criteria will require a tangible impact to be made in the discipline area (Greenhaugh and Wieringa, 2011; Schaillée et al., 2019, Verhagen et al., 2013). It seems fair to apply the scientific evidence from research to training programmes but there are several considerations when bridging the gap between sports science and the field, and historically this transfer has been limited (Bishop, 2008; Eisenmann, 2017; Fullagar et al., 2019). While there is a growing interest in sports science in the coaching community, Finch (2011) lists the three main reasons for the poor rate of knowledge transfer as research relevance failure, translation/adoption failure, and dissemination failure. Research relevance failure encompasses research which does to relate to 'real-world' application or is difficult to implement in a practical setting (Bishop, 2008; Fullagar et al., 2019). Dissemination failure is

when the research is not shared with the appropriate groups and therefore cannot be implemented in a practical setting. Translation/adoption failure involves research information getting to the right groups of people but findings are not understood or, through a reluctance of coaching staff to change their practices, acted upon appropriately (Halperin, 2018).

An evidence-based practice where sports science is combined with practical knowledge is recommended to help overcome the barriers between knowledge generation and practical implementation (Fullagar et al., 2019). Therefore, in order to ensure this thesis provides a valuable contribution to the field, it will address research questions with clear 'real-world' context and application and produce recommendations which can be easily understood and applied by practitioners.

## 2.3 Invasion sport

Invasion sport involves two opposing yet interacting teams of players competing against each other within a fixed area of play and for a given duration specific to that sport (Gudmundsson and Horton, 2016). This includes the sports of field hockey, basketball, handball, and lacrosse, as well as the various codes of football such as soccer, rugby union, and American football (Taylor et al. 2017). It is characterised by the invasion of the opposing team's end of the playing area in an attempt to score goals whilst simultaneously attempting to prevent them from doing the same (Lord et al., 2020). These types of sport are similar in that players are free to move around the playing area while attacking or defending (Gudmundsson and Horton, 2016), although goals are scored in different ways such as striking the ball in soccer and hockey, throwing the ball in basketball and netball, or carrying the ball over the line in American football and rugby union (Lord et al., 2020). To optimise training benefits, training stimulus should replicate the demands of match play (Cummins et al. 2013; Gabbett, 2010b; Harper, Carling and Kiely 2019; Smart et al., 2014; Vaz et al., 2014) which is why it is important to accurately understand the demands of the sport.

### 2.3.1 Kinematics of invasion sports

Invasion sports involve prolonged periods of low to moderate intensity physical activity such as walking and jogging, interspersed with repeated bouts of high intensity efforts of high-speed running and sprinting which fluctuates from standing to rolling starts (Faiss, Girard and Millet, 2013; Gabbett, Jenkins and Abernethy, 2012; Lemos et al., 2017; Paul, Bradley and Nassis, 2015; Wehbe, Hartwig and Duncan, 2014). These movements are multi-directional and depending on the sport, there will be a greater or lesser requirement for sagittal plane high intensity running and sprinting, lateral shuffling and cutting, and vertical jumping movements (Taylor et al., 2017). Understanding these demands has traditionally been done through motion analysis with most researchers using an observational technique (Drust, Atkinson and Reilly, 2007). From these data, it can be determined what type of activity (walking, jogging, running etc.) the athlete is undertaking, along with its duration and frequency to estimate the total workload over the course of a match. Information such as total distance covered, percentage of time spent at given speeds, and work to rest ratios are calculated, but this does not offer an accurate reflection of the true physiological demands placed on the athlete (Wehbe, Hartwig and Duncan, 2014). Relatively recent advances in technology are allowing sports scientists to collect more detailed information using wearable global positioning system (GPS) technology with integrated accelerometers that can provide additional data on the number and magnitude of accelerations, changes of direction and other high force production activities such as tackling and jumping (Wehbe, Hartwig and Duncan, 2014) which are metabolically demanding (Duthie, Pyne and Hooper, 2003; Kempton et al., 2015). GPS data also provides insight into the specific and positional demands of invasion sport (Cummins et al., 2013).

During competitive matches, athletes cover several thousand metres over the duration of play. Total distance covered provides a useful metric to compare the running demands of different playing positions within a sport. Midfield players in field hockey and soccer cover more distance than defenders and strikers (Abbott, Brickley and Smeeton, 2018; Gabbett, 2010a), and in rugby union,

backs cover more distance than forwards during a match (Suárez-Arrones et al., 2012; Cunniffe et al., 2009). There are, however, limitations when comparing the reported total distance covered both within and between sports. Total distance covered varies with age, sex, and ability level (Taylor et al., 2019). Elite athletes cover a greater total distance than non-elite players (Lacome et al., 2014; Roberts et al., 2008; Taylor et al., 2017), and care should be taken when comparing data from different studies to ensure participants are from a similar demographic to avoid over- or under-estimating demands. For example, elite male soccer players cover up to 12000m and it would be unreasonable to expect this from youth players who can cover as little as 2186m depending on age and ability (Taylor et al., 2017). At an international level, rugby union players can cover as much as 7500m (Lacome et al., 2014), whereas for teams in the English Premiership, total distance covered is 5866m (Roberts et al., 2008).

Another confounding factor is the size of the playing area, and the duration of match play. Larger playing areas and longer duration matches lend themselves to greater total distances, but this does not mean a player covering a shorter distance was necessarily working at a lower intensity if they also played for a shorter period. To help mitigate this issue, relative intensities can be calculated using metres covered per minute played ($m \cdot min^{-1}$) and when this is done, a more accurate representation of the demands of each sport is seen.

In addition to providing a meaningful comparison between sports, relative intensity can also provide a useful indication of fatigue with players often covering less total and relative distances in the latter stages of a match (Cunniffe et al., 2009; Rampinini et al., 2009). In hockey, players competing for between 32 and 45 minutes performed at a pace of 124.6 - 175.3 $m \cdot min^{-1}$, but when playing time was extended beyond 45 minutes, relative intensity dropped to 114 .3 - 152.3 $m \cdot min^{-1}$ (Morencos et al. 2019). In rugby, Higham et al. (2012) demonstrated that when substitutions are made, these new players covered 24% more distance per minute they played compared to athletes who played the full match.

### 2.3.2 High-intensity exercise during invasion sports

The ability to frequently perform repeated high-intensity or sprinting actions without complete recovery is an integral component of invasion sports (Bishop, Girard and Mendez-Villanueva, 2011; Faiss, Girard and Millet, 2013; Girard, Mendez-Villanueva and Bishop, 2011; Hamlin et al., 2017; Spencer et al., 2004), and is argued to be a decisive element of success (Austin, Gabbett and Jenkins, 2011a; Bishop et al., 2006, 2011; Gabbett, 2010b; Girard, Brocherie and Millet, 2015; Rampinini et al., 2007). This can be divided broadly into two main categories of intermittent sprint exercise and repeated sprint exercise (Girard, Mendez-Villanueva and Bishop, 2011). Intermittent-sprint exercise comprises short duration sprints of approximately ten seconds or less followed by a recovery period greater than 60s allowing near complete recovery and no reduction in subsequent sprint performance. Repeated sprint exercise can be defined as sprints of ten seconds or less repeated with less than 60s recovery between sprints (Girard, Mendez-Villanueva and Bishop, 2011).

Players change activity approximately every 2 - 4s (Taylor et al., 2017) including performing multiple high- and very-high-intensity accelerations and decelerations (Harper, Carling and Kiely 2019) making this element of invasion sport one of the most demanding for athletes (Polglaze and Hoppe 2019). GPS data provides additional information on the number and magnitude of accelerations, changes of direction, and other high force production activities such as tackling and jumping (Wehbe, Hartwig and Duncan, 2014) which are also metabolically demanding for players (Duthie, Pyne and Hooper, 2003; Kempton et al., 2015). Across invasion sports, all players (except for goalkeepers) will perform multiple bouts of high intensity work with sprinting accounting for 1-10% of total distance covered or 1-3% of effective playing time (Girard, Mendez-Villanueva and Bishop, 2011). While there is a commonality for these actions to be performed multiple times, the number varies within sports depending on playing positions, age, and sex (Al Haddad et al., 2015; Mancha-Triguero et al., 2021).

Care must be taken when grouping invasion sports for activity profiles as there are some differences between them. For example, in elite male rugby union, number of repeated high-intensity bouts ranged from 2 to 21 per player (Austin, Gabbett, and Jenkins, 2011b) whereas, in elite male field hockey, Spencer et al. (2004) reported only 17 repeated high-intensity activity bouts for all playing positions. Both these studies defined a repeated high-intensity exercise bout as a passage of play resulting in three or more individual bouts within 21 seconds during the same passage of play. However, Spencer and colleagues (2004) are reporting on only sprinting activity because field hockey is a non-contact sport whereas rugby union includes contact activities such as tackling, rucking and scrummaging which are also high intensity in nature (Austin, Gabbett, and Jenkins, 2011b; Deusch et al., 2007; Duthie et al., 2006).

Another limitation when comparing data both within and between invasion sports is the lack of uniformity when defining aspects such as movement speed zones (Cummins et al., 2013). In a systematic review of 81 studies, Taylor and colleagues (2017) reported sprinting was defined across a range of invasion sports by different authors as running speeds greater than between 5.28 and 8.33 $m \cdot s^{-1}$. Even within the literature on youth soccer alone there are differences. Sprinting is defined by Buchheit et al. (2010a) as a running speed >5.31 $m \cdot s^{-1}$ for Under-13 to Under 18 players, whereas Aslan and colleagues (2012) define sprinting under three separate categories for youth players (mean age 17.6 ± 0.58 years). These are categorised as; low intensity sprint - 5.03 to 5.83 $m \cdot s^{-1}$, moderate intensity sprint - 5.84 to 6.67 $m \cdot s^{-1}$, high intensity sprint - >6.67 $m \cdot s^{-1}$. These differences whilst small can make comparison between studies problematic. A similar situation exists in the literature for field hockey where sprinting is defined as running speeds >5.56 $m \cdot s^{-1}$ (Vescovi et al., 2014, 2015) and >5.83 $m \cdot s^{-1}$ (Morencos et al. 2019) for elite level female players. In real terms, this is the difference between completing 20m in 3.60s and 3.43s which makes a large difference when competing for a ball. More work is required within the field of sports science to unify these measurements although that is outwith the scope of this project.

### 2.3.3 Metabolic demands of invasion sport

Invasion sport match play last 60 - 90 minutes or more meaning there is a need for energy provision over a prolonged period of time. While invasion sports such as soccer are considered to be predominantly aerobic in nature (Drust, Atkinson and Reilly, 2007), due to the intermittent nature of these sports, both the aerobic and anaerobic energy systems are utilised, with approximately 29% anaerobic contribution to overall game play (Osgnach et al., 2010; Paul, Bradley and Nassis, 2015; Stolen et al., 2005). Again, there is variation within and between sports such as forwards in rugby union being more reliant on anaerobic glycolysis than backs because of the non-running high-intensity work such as scrummaging and mauling they perform (Deutsch, Keearney and Rehrer, 2007). Field hockey players will perform low intensity work for ~85 - 88% of playing time with the remaining 12 - 15% consisting of high-intensity running and sprinting greater than ~4m·s$^{-1}$ (Harry and Booyen, 2020). In soccer however, time spent performing high-intensity running and sprinting greater than ~4m·s$^{-1}$ accounts for 20% of match play (Aslan et al., 2012; Buchheit et al., 2017; Castagna et al., 2010; Hill-Haas et al., 2010; Russell et al., 2011; Strøyer, Hansen and Klausen, 2004) with the additional requirement of maintaining this for 30 minutes longer than field hockey matches.

Elite athletes in invasion sports cover a greater total distance than non-elite players showing the importance of high levels of aerobic fitness as playing standard improves (Austin, Gabbett and Jenkins, 2011a; Lacome et al., 2014; Mohr et al., 2008; Roberts et al., 2008). Cardiorespiratory fitness is also linked to repeated sprint ability (RSA) with greater oxidative capacity allowing superior maintenance of high intensity efforts through replenishment of muscle phosphocreatine (PCr) concentrations (Bishop and Edge, 2006; Hamilton et al., 1991).

Despite the absolute differences between sports for total distance covered and volume of high-intensity work, heart rate (HR) response is similar between sports and ability levels. Throughout match play, average heart rate is approximately 85% maximum heart rate ($HR_{max}$) which equates to 70 - 80% maximal oxygen uptake ($\dot{V}O_{2max}$) (Bangsbo, Mohr and Krustrup, 2006; Castagna et al., 2010; Coutts, Reaburn and Abt, 2003; Cunniffe et al., 2009),

although, in rugby union, some position specific demands can result in up to 72% of the game being played above 85% $HR_{max}$ (Duthie, Pyne and Hooper, 2003) and approximately 20% of the match above 95% maximum heart rate ($HR_{max}$) (Deutsch et al., 1998). Maximal lactate steady state (MLSS) is associated with a mean $[La^-]b$ of 4 $mmol \cdot l^{-1}$ (Billat et al., 2003), and an oxygen uptake ($\dot{V}O_2$) equivalent to 70 - 90% of $\dot{V}O_{2max}$ (Jones and Vanhatalo, 2017; Poole et al., 2016) suggesting that many players will be at, or above, MLSS intensity throughout matches and as such will experience elevated blood lactate concentrations ($[La^-]b$.

Recorded blood lactate during game play has been shown to be 4 - 8 $mmol \cdot l^{-1}$ in soccer (Aslan et al., 2012), 5.8 - 9.8 $mmol \cdot l^{-1}$ in rugby union (McLean, 1992), and 5.2 - 5.8 $mmol \cdot l^{-1}$ in field hockey (Kusnanik, Rahayu and Rattray, 2018) showing a common reliance on anaerobic glycolysis during periods of high intensity activity across the three sports. Aslan et al. (2012) demonstrated that 23% of distance travelled during a soccer match occurs at running speeds above the onset of blood lactate accumulation (set at 4 $mmol \cdot l^{-1}$). Blood lactate concentration ($[La^-]b$) is also greater in elite level players compared to non-elite players due to the greater number of high-intensity activities performed at that level (Mohr, Krustrup and Bangsbo, 2003). It has been suggested that the distance players cover at high intensity may reflect their ability to maintain lactate balance as assessed by lactate threshold (Edwards et al., 2003). This indicates a player's ability to metabolise lactate could be a key determinant of performance and therefore, improving lactate metabolism through training may be beneficial for invasion sport performance.

There is a decrease in $[La^-]b$ between first and second half of game play that coincides with a significant ($p < 0.01$) drop in high-intensity running and sprinting between halves (MacLeod, Bussell and Sunderland, 2007; Stolen et al., 2005). This suggests a link to neuromuscular fatigue which can also increase the risk of injury (Harper, Carling and Kiely 2019). Therefore, maintaining substrate availability may help prevent this drop in high-intensity activity and sprint performance, and possibly help mitigate the risk of injury.

## 2.4 Energy metabolism

During exercise such as invasion sport play, there is a requirement to provide the working muscle with sufficient energy to allow contractions to occur. This energy is derived from the hydrolysis of adenosine triphosphate (ATP) in the presence of ATPase. ATP can be generated both aerobically (oxidative phosphorylation) and anaerobically (phosphocreatine or anaerobic glycolysis) and the extent of contribution from each energy system depends on the intensity and duration of the exercise (Figure 2.1 and 2.2). During invasion sports such as soccer, it is estimated approximately 71% of total energy consumed is produced aerobically with the remainder produced through a combination of the anaerobic pathways (Osgnach et al., 2010; Paul, Bradley and Nassis, 2015; Stolen et al., 2005). This plays an important role during the numerous high-intensity actions performed throughout a match (Mohr, Krustrup and Bangsbo, 2003).

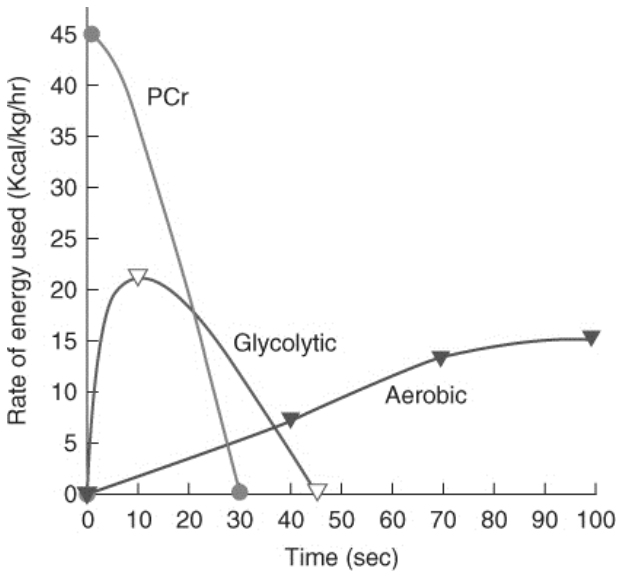


***Figure 2.1 Energy continuum (MacLaren and Morton, 2012)***

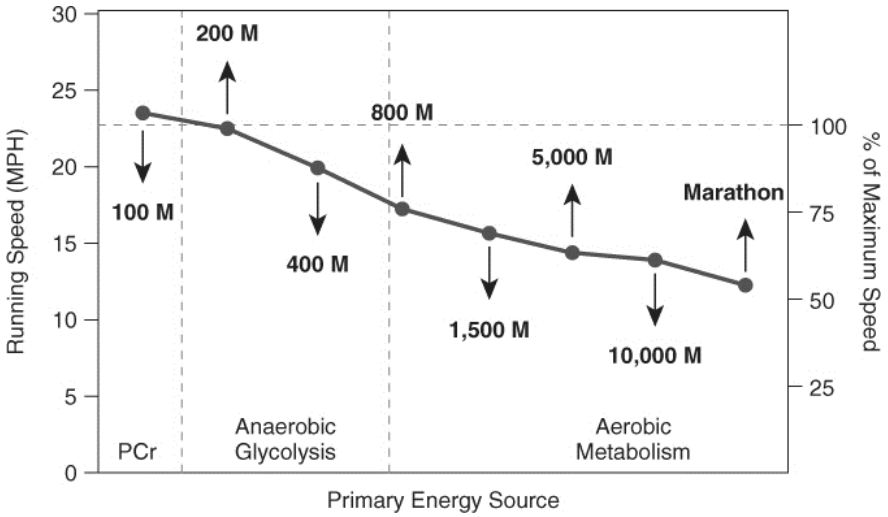


***Figure 2.2 Primary energy sources for different distances at maximal effort (MacLaren and Morton, 2012)***

### 2.4.1 Phosphocreatine

At the onset of any physical activity, intramuscular phosphocreatine (PCr), the breakdown of which is catalysed by the enzyme creatine kinase (CK), is the predominant source of ATP. In human skeletal muscle it is estimated that PCr stores equate to 80 mmol·kg dry muscle$^{-1}$ (Glaister, 2005) and ATP resynthesis is achieved through the donation of a phosphate to adenosine diphosphate (ADP) producing ATP and creatine (Cr) (Figure 2.3). Following a high intensity bout, there is a bi-exponential replenishment of PCr with ~50% in first 30s (Gaitanos et al., 1993), ~75% within the first minute, and full recovery between three and five minutes (MacLaren and Morton, 2012). Acute exercise also leads to an elevation in CK concentration within the cell (Hazar et al., 2015; Xu et al., 2016). ATP produced aerobically within the mitochondria is broken down in a reaction catalysed by a second form of CK associated with the mitochondria ($CK_{mito}$), and this provides the dephosphorylated Cr with a phosphate to create PCr (MacLaren and Morton, 2012).

The rate at which PCr is replenished plays an important role in invasion sports with athletes who exhibit more rapid PCr regeneration also performing more high intensity actions and a greater number of sprints during match play (Bishop and Edge, 2006; Redkva et al., 2018). There is a strong association between the rate at which ATP can be generated aerobically and the ability of an athlete

to perform repeated bouts of high intensity work (Bogdanis et al., 1996; McKenna et al., 1997; Parolin et al., 1999; Trump et al., 1996). In invasion sport athletes, there are significant correlations ($r = -0.57$, $p < 0.05$) between maximal oxygen uptake and rate of fatigue during repeated sprint exercise Gharbi et al., 2015).

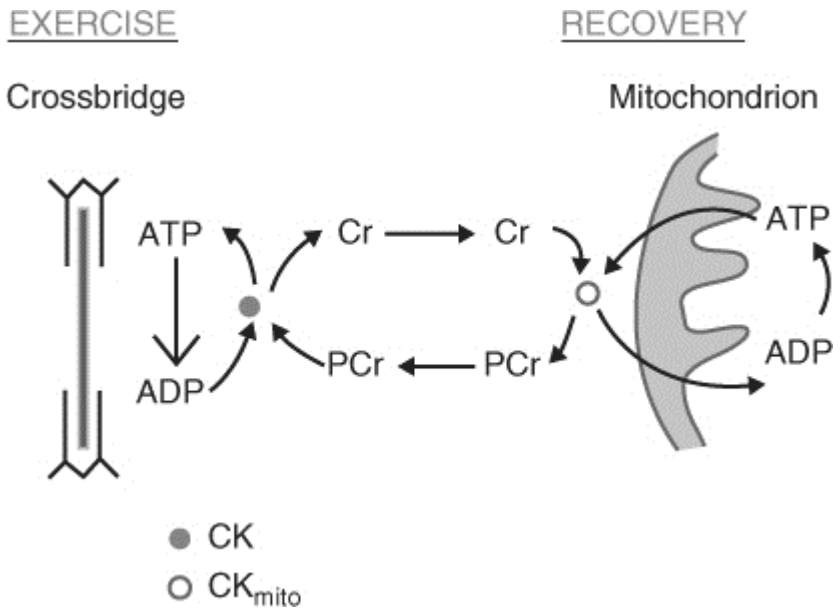


***Figure 2.3 PCr shuttle (MacLaren and Morton, 2012)***

### 2.4.2 Anaerobic glycolysis

Anaerobic glycolysis occurs in the sarcoplasm of the muscle cell and utilises either locally stored muscle glycogen or blood glucose (Adeva et al., 2013) to produce a net gain of two or three ATP respectively (Powers and Howley, 2012). While energy supplied from this process is rapid, it is not particularly efficient in terms of the amount of ATP generated. Intramuscular glycogen stores are much greater than that of PCr (~300 mmol·kg$^{-1}$ dry muscle compared to ~80 mmol·kg$^{-1}$ dry muscle) (Glaister, 2005) but vary with training status reaching up to 800 mmol·kg$^{-1}$ dry muscle in trained individuals (Hearris et al., 2018). It is argued glycogen stored within the working muscles is the most important energy substrate during invasion sport match play (Bangsbo 1994).

80% of CHO reserves found in skeletal muscle is in the form of glycogen with an upper limit of 4g per 100g wet muscle (Hansen et al., 1999). Glycogen is produced in glycogenesis which is the polymerisation of glucose and is catalysed by the enzyme glycogen synthase (GS) (Roach, 2002).

Glycogen levels are depleted during exercise and replenished afterwards. However, the manner in which it is utilised is complex and varies greatly depending on factors such as the exercise undertaken (intensity, duration and mode) and the training status of the individual (Marchand et al., 2007; Nielsen et al., 2011).

Glycogen is stored in three distinct locations within the muscle fibre (Nielsen et al., 2009) and these are; within the myofibrils (intramyofibrillar (INTRA) glycogen), between the myofibrils (intermyofibrillar (IMF) glycogen), and in the subsarcolemmal space just beneath the surface of the fibre (SS glycogen).

The subcellular localisation of glycogen may provide a substrate for specific cellular functions (Philp, Hargreaves and Baar, 2012). IMF glycogen represents the greatest quantity within the fibre and is situated close to the sarcoplasmic reticulum (SR), t-tubules and mitochondria (Nielsen et al., 2009). Its presence is positively correlated with the half-relaxation time of unfatigued tetanic contraction which indicates a role in SR Ca2+ reuptake (Nielsen et al., 2010). IMF glycogen provides the energy for sodium-potassium adenosine triphosphatase and the SR calcium ATPase which initiates the repolarisation of the t-tubules (Philp, Hargreaves and Baar, 2012). INTRA glycogen is positioned to provide CHO for cross-bridge cycling and is depleted preferentially during high-intensity exercise (Nielsen et al., 2011). It is also associated with force maintenance during repeated tetanic contraction (Philp, Hargreaves and Baar, 2012). As levels of INTRA glycogen reduce, so does calcium (Ca2+) release rate (Ørtenblad et al., 2011), suggesting a link between low glycogen levels and fatigue (Nielsen et al., 2010, 2011).

Glycogen utilisation and resynthesis both during and after fatiguing exercise, appears to be dependent on its subcellular location, with INTRA glycogen being both metabolised (Nielsen et al., 2011) and resynthesised (Marchand et al., 2007) preferentially to either IMF or SS glycogen. This is true for both Type I and Type II fibres. Nielsen et al. (2011) suggest the preferential depletion of

INTRA glycogen may mediate a signal for forceful muscle contraction to halt by reducing the Ca2+ release rate. The preserved IMF glycogen then serves as a buffer of energy for reuptake of Ca2+ into the SR. The role of SS glycogen is less clear although Philp, Hargreaves and Baar (2012) suggest that due to its sensitivity to exercise and nutrition, it could play a role in intracellular signalling. Relative glycogen distribution within Type I and II skeletal muscle fibres is similar, with respective concentrations of 77% and 84% for IMF glycogen, 12% and 8% for INTRA glycogen, and 11% and 8% for SS glycogen (Nielsen et al., 2009). In both untrained, obese (Nielsen et al., 2010a) and recreationally active (lean) (Nielsen et al., 2010b) individuals, there are equal absolute levels of INTRA and SS glycogen between Type I and Type II fibres. However, in elite cross-country skiers, it was shown that Type I fibres contained ~80% more INTRA glycogen and ~30% more SS glycogen than Type II fibres for both upper (triceps brachii) and lower (vastus lateralis) limbs (Nielsen et al., 2011), suggesting a long-term training adaptation.

The glycolytic pathway involves a complex series of reactions requiring several enzymes (Figure 2.4). One of these enzymes is phosphofructokinase (PFK) which controls the conversion of fructose 6-phosphate to fructose 1,6-diphosphate (Figure 2.4) and is believed to be the rate limiting factor controlling anaerobic glycolysis during maximal effort exercise (Alves and Sola-Penna, 2003; Hearris et al., 2018). PFK levels are comparatively high in Type II muscle fibres (Bouchard et al., 1986) allowing these fibres to produce high-power outputs anaerobically. Mohr et al. (2016) found that in invasion sports, total sprint distance and the average duration of individual sprints during high intensity periods of match play was significantly correlated ($r = 0.46 - 0.48$; $p < 0.05$) to PFK activity levels. Likewise, Iaia et al. (2011) associated PFK levels with several sprint indicators including RSA. It has also been shown that sprint training significantly increases PFK activity levels (Fournier et al., 1982) which should then lead to an increase in the glycolytic capacity of the muscle during sporting activities. Indeed, following eight weeks of sprint training there was a greater contribution of glycogen to energy production during the Wingate Anaerobic Test (WAnT) (Barnett et al., 2004).

During glycolysis, two pairs of hydrogen ions ($H^+$) are stripped from the glucose substrate 3-phosphoglyceraldehyde and bind to nicotinamide adenine dinucleotide ($NAD^+$) to form NADH (Figure 1.4). Under normal respiration, NADH is oxidised in further processes involving the additional $H^+$ being transported into the mitochondria and thereby maintaining the redox balance, with $NAD^+$ freely available for continued glycolysis. During low intensity exercise at ~40% maximal oxygen uptake ($\dot{V}O_{2max}$), NADH levels are seen to decrease but with the $NAD^+$/NADH ratio largely unchanged (Sahlin, Katz and Henriksson, 1987). As exercise intensity increases beyond 75% $\dot{V}O_{2max}$, such as it is during invasion sport match play (Bangsbo, Mohr and Krustrup, 2006), NADH production exceeds the cell's $H^+$ transport capacity leading to a 'backing up' of the electron transport chain (ETC) (White and Schenk, 2012). Therefore, for the continuation of glycolysis and further pyruvate metabolism to be possible, $NAD^+$ must be regenerated (Powers and Howley, 2012). This reaction is catalysed by the enzyme lactate dehydrogenase (LDH) and results in the formation of lactate through the reduction of pyruvate in further anaerobic metabolism (Adeva et al., 2013). LDH is abundant in the cytosol of cardiac and skeletal muscle cells, and concentrations increase following acute exercise (Donovan and Brooks, 1983; van Hall, 2000; Xu et al., 2016). LDH exists as one of two types: M (muscle) form which predominates in skeletal muscle, and H (heart) form found predominantly in cardiac muscle (van Hall, 2000). Tissue with high oxidative metabolism (cardiac and Type I skeletal muscle fibres), and therefore high lactate oxidation capability, are associated with the H form of LDH which favours conversion of lactate to pyruvate. Conversely, highly anaerobic tissue (Type II skeletal muscle fibres) is more likely to contain M form LDH which exhibits a greater exchange of pyruvate to lactate (van Hall, 2000). Exercise training develops the capacity of skeletal muscle to oxidise NADH (White and Schenk, 2012) through the increase in mitochondrial size and density (Hearris et al., 2018) allowing higher $H^+$ transportation rates and therefore reduced lactate accumulation.

#### *2.4.2.1 Lactate*

The terms lactic acid and lactate are often used interchangeably but they are two different molecules (Powers and Howley, 2012). The lactic acid produced during anaerobic glycolysis is very rapidly, and almost completely (99%), dissociated into lactate ($La^-$) anions and hydrogen ions ($H^+$) (Agüera et al., 1995; Gladden, 2004; Powers and Howley, 2012). This lactate was once thought to be a metabolic waste product responsible for the fatigue experienced during high intensity exercise (Azevedo et al., 2007; Gladden, 2004; Gollnick, Bayly and Hodgson, 1986; MacLaren and Morton, 2012; Powers and Howley, 2012). There is now however, a great deal of evidence that lactate is an important energy substrate (Azevedo et al., 2007; Bryner et al., 1998; Ellis, Simmons and Miller, 2009; Fahey et al., 1991; Gladden, 1991, 2000; Godfrey et al., 2009; Hamann, Kelley and Gladen, 2001; Handy, 2006; Hubbard, 1973; Jeppesen et al., 2013; Kelley et al., 2002; Morris et al., 2011; Swensen et al., 1994; van Hall et al., 2003; van Montfoort et al., 2004).

*Endogenous Lactate Production and Metabolism*

Although definitely not the only site, skeletal muscle is the predominant producer and consumer of lactate (Bergman et al., 1999), and this is true during both exercise and at rest (Brooks et al., 1991; Donovan and Brooks, 1983; Stanley et al., 1986). Anaerobic glycolysis still occurs within individual muscles at rest which results in a net release of lactate (Gladden 2000) with no evidence supporting the idea that there is an inadequate supply of $O_2$ (Svedahl and MacIntosh, 2003).

In resting humans, the concentration of lactate in the blood is low and stable (Brooks et al., 1999a; Stanley et al., 1986) with arterial lactate concentrations of approximately 0.5 - 1mmol·$l^{-1}$ (Chatham, Des Rosier and Forder, 2001; Gollnick, Bayly and Hodgson, 1986). Lactate is released from muscles continuously, but at times such as during recovery from exercise, or even during prolonged duration low to moderate intensity exercise, skeletal muscle may show a net uptake of lactate (Gladden, 2000). During exercise of an intensity ≤40% $\dot{V}O_{2max}$ (low intensity) there is little or no increase in $[La^-]b$ (Gollnick, Bayly and Hodgson, 1986) because rate of appearance is matched by rate of disposal

(Bang, 1936; Brooks et al., 1991; Stanley et al., 1986). Only individuals who have a low aerobic capacity demonstrate a notable rise in $[La^-]b$ up to an exercise intensity of approximately 50 - 60% $\dot{V}O_{2max}$ (Connett, Gayeski and Honig, 1986; Gollnick, Bayly and Hodgson, 1986). As exercise intensity increases beyond 60% $\dot{V}O_{2max}$, the appearance of lactate in the blood becomes more evident (Agüera et al., 1995; Bang, 1936; Gollnick, Bayly and Hodgson, 1986) and whilst working at a high intensity ≥85% $\dot{V}O_{2max}$, there is an exponential increase in the appearance of blood lactate directly linked with the requirement for anaerobic glycolysis (Brooks et al., 1991; Donovan and Brooks, 1983; Gladden, 2000; Gollnick, Bayly and Hodgson, 1986; Stanley et al., 1986). Following 30s of supramaximal exercise such as the WAnT, $[La^-]b$ can increase by approximately 1000% (Weinstein et al., 1998).

Lactate is simultaneously produced and consumed continuously both at rest and during exercise by skeletal muscle (Bang, 1936; Depocas, Minaire and Chatonnet, 1969; Donovan and Brooks, 1986; Gladden, 2004). Lactate produced within one muscle fibre can be oxidised within that fibre, an adjacent fibre, or within a different muscle entirely (Brooks, 2000; Brooks et al., 1991). In normal conditions, resting lactate turnover is ~1mmol·kg·$h^{-1}$ (Handy, 2006). Approximately 50% of lactate metabolism in resting mammals is accounted for by oxidation (Depocas, Minaire and Chatonnet, 1969) and a further 20% by gluconeogenesis (Bergman et al., 2000; Brooks et al., 1999b; Donovan and Brooks, 1983; Mazzeo et al., 1986; Stanley et al., 1986). Of the lactate removed through oxidation at rest, 80% is removed by skeletal muscle (Kelley et al., 2002).

*In situ* research on muscle tissue in mammals has demonstrated that contracting muscle lactate consumption and oxidation are dependent on $[La^-]b$, and this is true for both skeletal and cardiac muscle tissue in humans (Brooks et al., 1991; Brooks et al., 1998; Stanley et al., 1986).

Circulating $[La^-]b$ becomes slightly elevated at the onset of low to moderate intensity exercise due to the working muscle having a greater lactate release than uptake (Bang, 1936). If this intensity is maintained, $[La^-]b$ gradually falls, often returning to resting levels (Bang, 1936) as muscles that originally had a

net release of lactate change to a net uptake (Brooks, 2000; Gladden, 1991) as consumption and oxidation increase in line with [$La^-$]b. At this point, disposal through oxidation and gluconeogenesis is 80% and 20% respectively (Brooks et al., 1991; Brooks et al., 1992; Stanley et al., 1986).

Despite intramuscular partial pressure of oxygen ($PO_2$) dropping at the onset of exercise, it remains above that which would be considered hypoxic even as exercise intensity increases to $\dot{V}O_{2max}$ (Richardson et al., 1998), and lactate will be produced even when there is no restriction in entry to the ETC (Connett, Gayeski and Honig, 1986). The initial rise in lactate output through dependence on glycolytic metabolism at the onset of exercise (Hill, Long and Lupton, 1924; MacLaren and Morton, 2012), may be due to a delay in the activation of oxidative metabolism rather than a shortage of oxygen (Gurd et al., 2006; Svedahl and MacIntosh, 2003). This includes the PDH complex which controls entry of carbohydrate-derived substrates into the TCA cycle (Gurd et al., 2006), and also fat metabolism, which is a major contributor to skeletal muscle fuelling during low intensity exercise (Helge et al., 2008).

Post-exercise differences observed between individuals who had demonstrated similar increases in [$La^-$]b, can be attributed to differences in clearance rates rather than rate of production (Donovan and Brooks, 1983; Mazzeo et al., 1986). In animals which were trained in cardiovascular endurance, metabolic clearance of accumulated blood lactate was 37% and 107% greater during low and high intensity exercise respectively (Donovan and Brooks, 1983). The significant increase in [$La^-$]b during high intensity exercise may be produced through a combination of factors. Larger Type IIa/x fibres, which are more dependent on glycolytic energy production, are recruited to cope with increasing force production requirements (Jones, Campbell and Pringle, 2004). They rely on glycolysis because they have fewer and smaller mitochondria (Ingier, 1979), lower levels of aerobic enzymes (Peter et al., 1972), and lower capillary density (Ingier, 1979) compared to Type I fibres. Once the rate of glycolysis outstrips the rate at which the TCA cycle metabolises pyruvate, there is a greater transformation of pyruvate to lactate to allow continued glycolysis (Carter, Jones and Doust, 2000). It was long held that in order to metabolise endogenously produced lactate, it would first have to interact with LDH to convert it back to

pyruvate in the sarcoplasm. The reformed pyruvate would in turn enter the mitochondria and begin oxidation in the TCA cycle (Adeva et al., 2013). However, some evidence suggests that the presence of LDH in the mitochondria of skeletal muscle cells (Brooks et al., 1999a; Brooks, 2002b; Dubouchaud et al., 2000), cardiac muscle cells (Brooks et al., 1999b; Brooks, 2002b), liver (Brooks, 2002b), and astrocyte cells of the brain (Lemire, Mailloux and Appanna, 2008) allows lactate to enter the mitochondria without prior conversion to pyruvate.

Another argument supporting the idea that lactate is an important energy substrate, is the data collected by Löfberg et al. (2001) through their research in mitochondrial myopathies. They found that individuals with impaired mitochondrial function exhibited a higher than usual [$La^-$]b during exercise suggesting they were unable to transport into, and therefore oxidise, lactate in the mitochondria of the working muscle.

*Lactate shuttles and whole-body kinetics*

Lactate has a low molecular weight and is exchanged quickly between tissue compartments, with movement across cell membrane barriers being facilitated by various transport mechanisms, or shuttles, and unlike glucose, does not require insulin (Brooks, 1998).

The lactate shuttle concept initially proposed by Brooks in 1985 describes the part lactate plays in the delivery of oxidative and gluconeogenic substrates. Examples of cell-cell shuttles in humans include lactate exchange between Type II and Type I muscle fibres within an exercising muscle, between exercising skeletal muscle and the heart, and between tissues of net lactate release and gluconeogenesis. Examples of an intracellular lactate shuttle are the uptake of lactate by mitochondria, and lactate-pyruvate exchange in peroxisomes (Brooks, 2002). The movement of lactate between areas of production and removal, such as across the sarcolemma of skeletal muscle, is facilitated by the several isoforms of specialist lactate transporters called monocarboxylate transporter (MCT) proteins that are differentially expressed in cells and tissues (Juel and Halestrap, 1999; Pilegaard et al., 1999b). The lactate shuttle hypothesis describes how lactate formed in muscle cells with

high levels of glycolysis, can be moved to other sites where it becomes an energy source and a gluconeogenic precursor (Brooks, 1986). In addition to neighbouring cells, these sites can include anatomically distant sites such as cardiac muscle and other skeletal muscle groups (Brooks, 1999). The ability to clear lactate is strongly correlated to performance in endurance sports (Baldari et al., 2007; Jacobs et al., 2011).

It is the presence of these cell-cell and intracellular lactate shuttles that supports the idea of oxidative and glycolytic energy pathways being linked in a continuum of predominance rather than alternatives. The substrate used in one pathway may be the lactate produced by the other (Brooks, 2002). Western blots from muscle biopsies taken by Bonen et al. (1998) showed training effects on the expression of MCT1 in both muscle sarcolemma and mitochondria, although this was not true for MCT4 (Brooks, 2002). These changes in sarcolemmal MCT1 expression and mitochondrial proteins, brought about through training, revealed that during exercise, lactate clearance rate is increased. Brooks (2002) states that the mitochondrial lactate-pyruvate transporter seems to operate in union with mitochondrial lactate dehydrogenase which allows oxidation of lactate in cells which are actively respiring. It has been shown that lactate is not only formed but also oxidised within the same cells through arteriovenous difference measurements on working skeletal and cardiac muscle, and also through nuclear magnetic resonance spectral analysis (Brooks, 2002). To maintain the redox balance in the cytosol and mitochondria and allow high flux rates, lactate oxidation and glycolysis must occur within the cells.

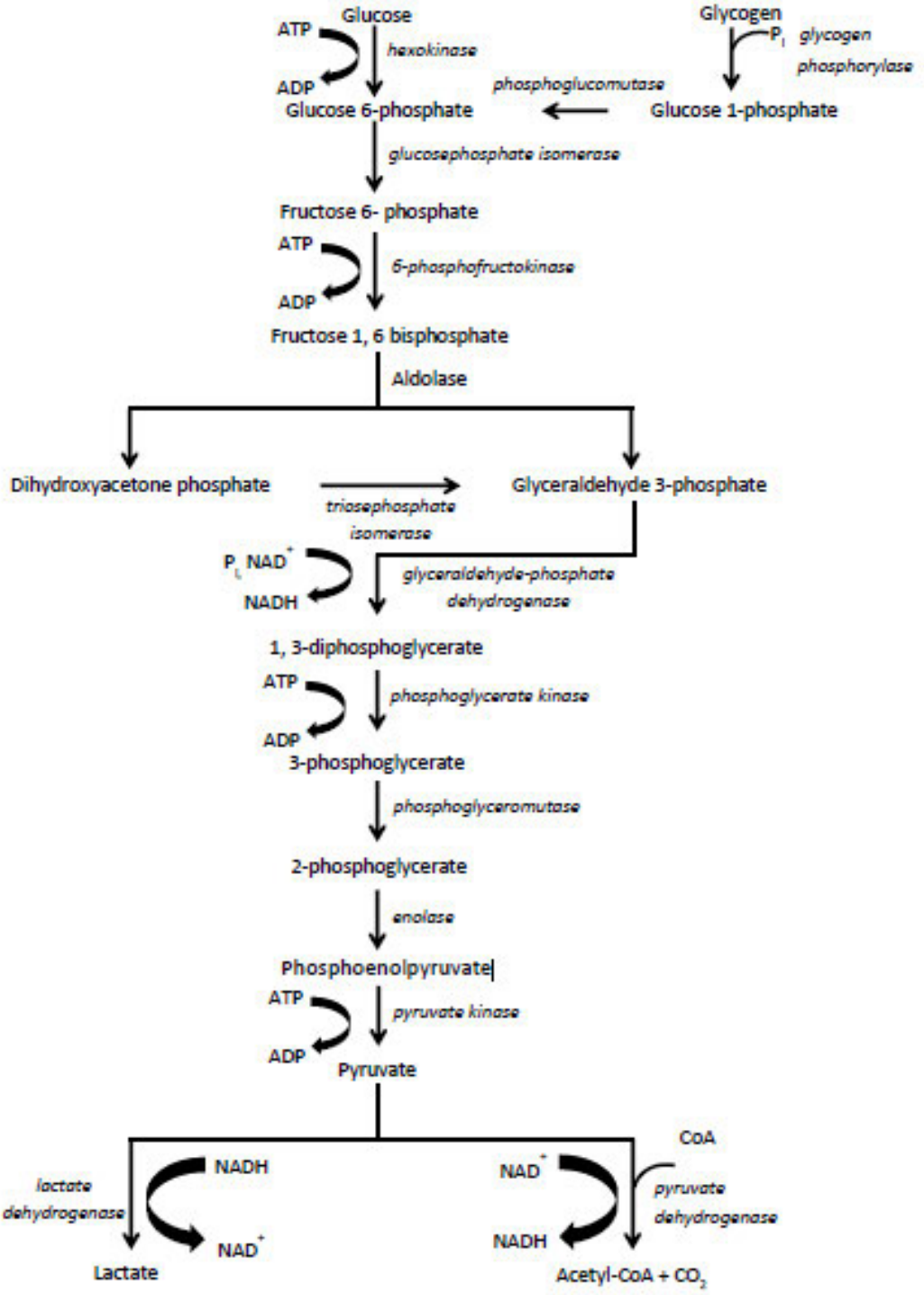


***Figure 2.4 The glycolytic pathway (Maughan and Gleeson, 2004)***

### 2.4.3 Oxidative phosphorylation

Commencing constant load submaximal physical activity, oxygen uptake ($\dot{V}O_2$) increases in a mono-exponential pattern which is thought to be a close approximation of $\dot{V}O_2$ response in the exercising limbs (Krustrup et al., 2009), and reaches a steady state within a few minutes when intensity is low to moderate (Christensen et al., 2010). Christensen and colleagues (2010) propose two main factors which determine the speed of these $\dot{V}O_2$ kinetics. The first of these is the activation of skeletal muscle respiration, triggered by an increase in concentrations of intramuscular ADP and inorganic phosphate ($P_i$) following hydrolysis of ATP. Pyruvate and fatty acids, formed through glycolysis and lipolysis respectively, enter the muscle cell mitochondria and form acetyl coenzyme A (acetyl-CoA) which in turn enters the tricarboxylic acid (TCA) cycle. It has been suggested that pyruvate dehydrogenase (PDH) plays an important role in this because it controls carbohydrate (CHO) and fat substrates entering the TCA cycle (Figure 2.6) (Gurd et al., 2005; Gurd et al., 2006). Through this cycle the two-carbon acetyl-CoA is converted to carbon dioxide ($CO_2$) and water, along with the production of energy. In addition to the production of a single molecule of ATP, hydrogen ions are donated to $NAD^+$ and flavin adenine dinucleotide (FAD) creating their reduced forms, NADH and $FADH_2$. A total of three molecules of NADH and two $FADH_2$ are formed and then enter the ETC for further processing. For each molecule of $FADH_2$ entering the ETC, a net gain of two ATP is made while for each molecule of NADH, there is a net gain of three ATP. The second element is the delivery of oxygen ($O_2$) to the skeletal muscle mitochondria which allows aerobic metabolism to occur, and once this is achieved, oxidative phosphorylation is the predominant source of fuel during steady-state exercise below the lactate threshold (Jones et al., 2010). ATP turnover is greater during high intensity compared to low intensity exercise (Howlet et al., 1998), with a greater reliance on oxidation of CHO and use of glycogen within the working muscle (van Loon et al., 2001). As exercise intensity increases, as seen in incremental time to exhaustion (TTE) tests, oxidative phosphorylation is unable to meet the demand for ATP leading to a greater reliance on anaerobic metabolism and an elevated rate of lactate production (Spriet, 2006). There are two main points of relevance in this

transition; lactate threshold (LT) and maximal lactate steady state (MLSS). LT is defined as the greatest exercise intensity which can be maintained during which $[La^-]b$ is elevated <1 $mmol \cdot l^{-1}$ from pre-exercise levels (Weltman et al., 1990), and occurs between 50 - 65% $\dot{V}O_{2max}$ (Wall et al., 2011). MLSS is the highest intensity at which oxidative phosphorylation is sufficient (Denadai and Higino, 2004). It is associated with a mean $[La^-]b$ of 4 $mmol \cdot l^{-1}$ (Billat et al., 2003), and a $\dot{V}O_2$ equivalent to 70 - 90% $\dot{V}O_{2max}$ (Jones and Vanhatalo, 2017). These intensities of exercise are an important marker when measuring physical capabilities of athletes and have been shown to correlate well with endurance performance (Denadai, Gomide and Greco, 2005).

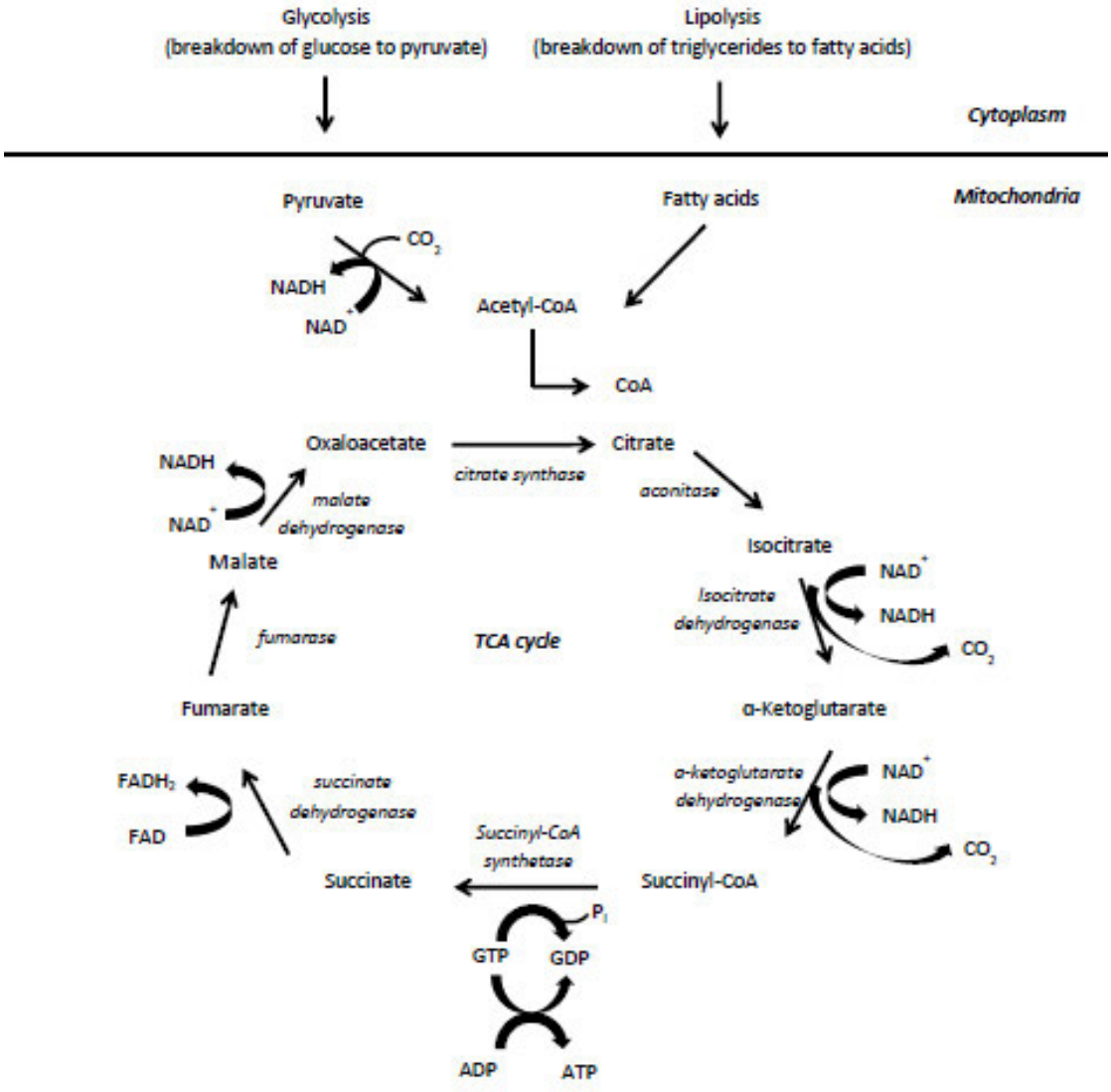


***Figure 2.5 An overview of oxidative phosphorylation (Powers and Howley, 2009)***

### 2.4.4 Energy production during maximal exercise

Invasion sports require multiple bouts of maximal effort exercise often separated by less time than is required to fully recover PCr stores (Bogdanis et al., 1996; Spencer et al., 2004). At the onset of maximal effort activity such as sprinting, PCr supplies ~50% of the total ATP production with anaerobic glycolysis supplying ~44% and the remaining ~6% contribution from aerobic metabolism (Gaitanos et al., 1993; Glaister, 2005). During this phase, maximum power achieved is typically 300 - 400% $\dot{V}O_{2max}$ which is far greater than can be met through aerobic metabolism alone (Spriet, 2006). As sprint duration increases, anaerobic glycolysis becomes more predominant, but the supramaximal rate at which energy is produced during a WAnT, for example, can only be maintained for a short duration (Parolin et al., 1999) with aerobic metabolism becoming increasingly prevalent (Figure 2.7). As pathway predominance transitions to aerobic metabolism, there is a concomitant reduction in power output (Bangsbo et al., 1990) particularly during the final 15s of a WAnT (Parolin et al., 1999), and overall aerobic contribution is ~28% (Serresse et al. 1988).

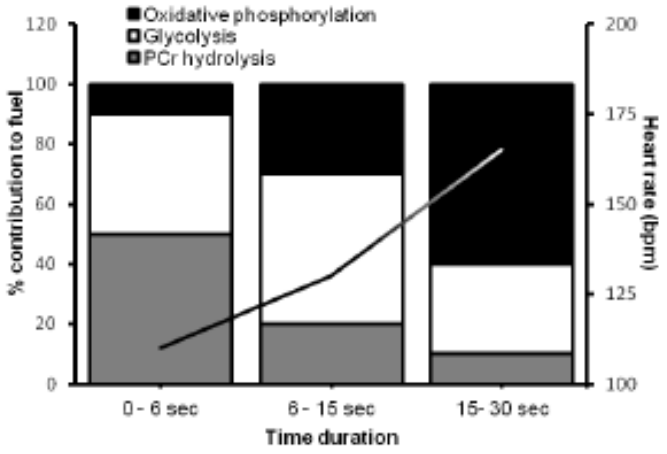


***Figure 2.6 Different fuel contributions to a single Wingate test (Parolin et al., 1999)***

If an effort is made to continue exercising at maximal intensity beyond 30s, the declining power output stabilises, and this point is termed critical power (CP) (Vanhatalo, Jones and Burnley, 2011). It is the maximum rate of work which can

be sustained through oxidative phosphorylation with stable values for muscle PCr concentration, pH, [$La^-$]b and $\dot{V}O_2$ (Jones and Vanhatalo, 2017). CP occurs at a higher absolute and relative intensity than LT and has been likened to MLSS (Jones and Vanhatalo, 2017) occurring between 70 - 80% $\dot{V}O_{2max}$ (Poole et al., 2016). During the fluctuating exercise intensities of invasion sports, the amount of work which can be undertaken above CP (W') is finite, and power output will return to CP as PCr and glycolytic pathways are exhausted. Although W' is constant, it will be utilised at different rates depending on the proximity of exercise intensity to CP (Jones and Vanhatalo, 2017). W' is depleted at intensities >CP and replenished at intensities <CP through resynthesis of muscle phosphocreatine concentrations (MacLaren and Morton, 2012). Training which develops $\dot{V}O_{2max}$ will also increase CP (Jones and Vanhatalo, 2017) and this is desirable for invasion sport performance as it allows athletes to perform at higher intensities without depleting energy substrates critical for the intermittent high intensity bouts.

### 2.4.5 Energy production during repeated sprints

The repetition of maximum effort sprinting leads to muscular fatigue, and a subsequent decline in power output (Mendez-Villanueva, Hamer and Bishop, 2008). There is a complex relationship between the disturbance locally within the muscle fibres during exercise and neural adjustments, with no clear single cause for performance decline during repeated sprint activity (Girard, Brocherie and Millet, 2015). It is known however, that aerobic energy metabolism provides a significant contribution to ATP production during repeated sprint exercise (Bogdanis et al., 1996; McKenna et al., 1997; Parolin et al., 1999; Trump et al., 1996). As the number of short (6 - 30s) high intensity bouts increase, the relative contributions of the anaerobic pathways decrease, and aerobic metabolism becomes the predominant source of fuel (Bogdanis et al., 1996; Bogdanis et al., 1998, Gaitanos et al., 1993; Parolin et al., 1999). As aerobic capacity increases, the potential to maintain repeated sprint performance is also increased (Gharbi et al., 2015). Comparing a group of invasion sport athletes to endurance-trained runners, Hamilton et al. (1991) demonstrated that the greater aerobic capacity of the endurance runners ($\dot{V}O_{2max}$: 60.8 ± 4.1ml·$kg^{-1}$·$min^{-1}$)

allowed them to maintain performance better than the invasion sport athletes ($\dot{V}O_{2max}$: 52.4 ± 4.9ml·kg$^{-1}$·min$^{-1}$). Throughout ten bouts of six second treadmill sprints with a 1:4 work to rest ratio, mean power output decline of the team players was 29.3 ± 8.1% while the endurance runners demonstrated a significantly smaller decline in performance of only 14.2 ± 11.1%. This idea is supported by the work of Bishop and Edge (2006), who found that athletes with a higher $\dot{V}O_{2max}$ (49.6 ± 4.8 vs. 36.4 ± 4.7ml·kg$^{-1}$·min$^{-1}$) showed a smaller decrement in sprint performance than their less aerobically fit counterparts. Likewise, using a similar protocol of ten bouts of six second cycle sprints with 1:3 work to rest ratio, Tomlin and Wenger (2002) found that while there was little difference in power for the first six sprints, the moderately-trained group with the higher $\dot{V}O_{2max}$ (47.6 ± 3.8ml·kg$^{-1}$·min$^{-1}$) maintained higher power output over the last four sprints compared to the group with lower $\dot{V}O_{2max}$ (34.4 ± 2.4ml·kg$^{-1}$·min$^{-1}$). Aziz, Chia and Teh (2000) also noted a significant negative correlation ($r = -0.346$ and $-0.323$; $p < 0.05$) between $\dot{V}O_{2max}$ and RSA in field hockey and soccer players. Given the requirement of invasion sport athletes to repeatedly perform multiple maximal effort sprints throughout match play (Drust, Atkinson and Reilly, 2007; Faiss, Girard and Millet, 2013; Lemos et al., 2017; Paul, Bradley and Nassis, 2015), this reinforces the importance of cardiorespiratory fitness in these types of athletes.

## 2.5 Supplementation to improve performance in invasion sport

The margins for winning and losing in invasion sports are small and the use of ergogenic aids to improve performance are of interest to athletes, coaches and sports scientists (Glaister et al., 2018). There are many commercially available sports drinks designed to meet the energy requirement of physical activity and traditionally, these drinks supply energy in the form of sugars such as glucose, fructose and sucrose (Coombes and Hamilton, 2000; Azevedo et al., 2007). There is a large body of research aiming to improve aerobic and anaerobic capacity, maximal sprint speed, and maintain both repeated sprint and intermittent sprint exercise performance through supplementation during

invasion sport play. Some of these focus on providing a supplement over a prolonged period of time, while others employ an acute dose prior to and/or during the exercise being undertaken. This research project will focus on an acute dose approach to improve performance for invasion sport athletes with particular focus on aerobic capacity, power, RSA, maximal speed and maintenance of sprint performance levels.

### 2.5.1 Endurance

Many researchers (Coombes and Hamilton, 2000; Ingle, Cooke and King, 2011; Phillips et al., 2012; Phillips, Sproule and Turner, 2011, Roberts et al., 2012; Russell, Benton and Kingsley, 2012) conclude that supplementation of glucose is beneficial to endurance exercise capacity in humans, and this is likely due to preservation of muscle glycogen (Nicholas et al., 1999; Russell, Benton and Kingsley, 2012; Tsintzas and Williams, 1998; Whitley et al., 1998). It is also well established that ingestion of glucose facilitates an increase in the athlete's capacity for intermittent high intensity exercise during simulation protocols of invasion sports such as rugby, soccer and field hockey (Davis et al., 1999; Davis, Welsh and Alerson, 2000; Davison et al., 2008; Foskett et al., 2008; Nicholas et al., 1995; Patterson and Gray, 2007; Welsh et al., 2002).
Stuart et al. (2005) reported that during prolonged exercise simulating the demands of rugby union, caffeine supplementation also lessened the decline in performance over the duration of testing. Similarly, during competitive and simulated match play, relative match intensity (87.5 ± 8.3 vs. 95.4 ± 12.7 $m \cdot min^{-1}$, $p < 0.05$) and pace at sprint velocity (4.6 ± 3.3 vs. 6.1 ± 3.4 $m \cdot min^{-1}$, $p < 0.05$) were significantly higher with caffeine supplementation compared to a placebo for female international rugby sevens players (Del Coso et al., 2013). Volume of high intensity running (303 ± 67 m and 358 ± 117m; $p = 0.05$) and sprinting (85 ± 41 m and 117 ± 55m; $p = 0.02$) was also significantly increased during simulated field hockey play, although total distance covered (6,035 ± 451m and 6,055 ± 499 m; $p = 0.87$) was unaltered (Del Coso et al., 2016). This was contradictory to the findings of Lara et al. (2014) who found that simulated soccer match play, caffeine supplementation significantly increased total distance covered (6,631 ± 1,618 vs 7,087 ± 1,501 m; $p < 0.05$). They also found

a significant increase in the number of individual sprints (16 ± 9 vs 21 ± 13; $p < 0.05$), and the distance covered sprinting (161 ± 99 vs 216 ± 103 m; $p < 0.05$). Additionally, throughout endurance performance testing specific to invasion sports (Nunes et al., 2021), performance has been significantly improved through supplementation of nitrate in the form of beetroot juice with 4.2% ($p < 0.05$) higher scores in the Yo-Yo IR1 test (Wylie et al., 2013). When perfuming 40-minutes of simulated basketball play however, López-Samanes et al. (2020) concluded beetroot juice had no effect on distance covered, running speeds, or the number of accelerations and decelerations performed by adolescent players.

### 2.5.2 Sprinting

Most studies have found that supplementation of CHO does not attenuate the decline in sprinting performance seen throughout invasion sport match play (Abbey and Rankin, 2009; Ali and Williams, 2009; Foskett et al., 2008; Morris et al., 2003; Roberts et al., 2010b). Phillips et al. (2010, 2012) concluded glucose supplementation had no effect on peak or average 15m sprint times during a modified Loughborough Intermittent Shuttle Test and Kingsley et al. (2014) found that carbohydrate gel compared to placebo provided no benefit to average sprint speed during a 90-minute simulated soccer protocol.
In contrast, ingestion of 3 or 6 mg·kg body mass$^{-1}$ caffeine has been shown to improve both maximal and average running speed in invasion sport athletes during repeated sprint testing although this difference was not replicated during simulated match play (Carr et al., 2008; Lara et al., 2014). Stuart et al. (2005) reported that during prolonged exercise simulating the demands of rugby union, maximal sprint speed was improved following caffeine supplementation. However, it should be noted that any attenuation of sprint decrement may only be significant in low habitual caffeine consumers (Evans et al., 2018), and some studies report any difference in performance as negligible (Paton, Hopkins, and Vollebregt, 2001) or non-existent (Brown, Brown and Foskett, 2013). Performing 12 x 20m repeated sprints at 30s intervals, 0.15 g·kg body mass$^{-1}$ L-arginine supplementation also provided no benefit compared to a placebo for total sprint time or sprint speed decrement in male soccer players (Birol et al., 2019). Buck and colleagues (2015) found no benefit for RSA through supplementation of

nitrate in invasion sport athletes as did Reynolds et al. (2020). Maximal sprint speed was also unaffected through the use of nitrate (López-Samanes et al., 2020). However, Buck et al., (2015) reported no benefit to RSA following supplementation of sodium phosphate.

In field hockey players, supplementation of 0.2 g·kg body mass$^{-1}$ sodium bicarbonate provided no ergogenic benefit during the Loughborough intermittent shuttle test (Macutkiewicz and Sunderland, 2018). Neither average sprint time nor the decline in sprint speed over time were different and the authors concluded there to be no benefit from supplementing sodium bicarbonate for invasion sport performance. Another study found the opposite of this concluding acute supplementation of the same dose of sodium bicarbonate resulted in significantly shorter running times (939 ± 26 vs. 914 ± 22 s, $p = 0.006$) during exercise testing designed to replicate the demands of a field hockey match (Durkalec-Michalski et al., 2020).

### 2.5.3 Power

Testing power output following repeated bouts of anaerobic exercise, Lee et al. (2011) concluded there was no difference between glucose treatment and control groups for peak power output although average power was significantly ($p = 0.03$) higher with glucose. Supplementation of glucose has no effect on appearance rates of lactate (Rotstein et al., 2007), despite claims that CHO will actively limit lactate accumulation (Singh, Chaudhary and Sandhu, 2011).

In off-pitch performance testing of invasion sport athletes, a single dose of caffeine compared to a placebo has been shown to significantly ($p \leq 0.05$) increase vertical jump performance in female rugby sevens players (Del Coso et al., 2013) and female soccer players (Lara et al., 2014), and Schneiker et al. (2006) found it to significantly improve both peak power and total work performed during intermittent sprint exercise. Additionally, when combined with 1 g·kg body mass$^{-1}$ taurine, Karayigit et al. (2021) reported 6 mg·kg body mass$^{-1}$ caffeine significantly improved both peak ($p = 0.03$) and mean power ($p = 0.01$) during Wingate tests performed by female invasion sport athletes. There was no ergogenic benefit to supplementing nitrate in the form of beetroot juice on vertical jump performance (López-Samanes et al., 2020).

Durkalec-Michalski et al. (2020) found no difference in anaerobic power determined through Wingate tests before and after the simulated match exercise through supplementation of sodium bicarbonate. However, in another study involving invasion sport athletes, Miller et al. (2016) found supplementation of 300 mg·kg body mass$^{-1}$ sodium bicarbonate provided a significant ($p < 0.05$) increase in total work performed during a 10 x 6s repeated sprint cycling protocol compared to a control or placebo. In an intermittent sprint test performed by female invasion sport athletes and designed to mimic the work patterns of invasion sports, 0.2 g·kg body mass$^{-1}$ sodium bicarbonate also provided a benefit to total work performed and the authors concluded it would be a useful ergogenic supplement for invasion sport athletes (Bishop and Claudius, 2005).

## 2.6 Lactate Supplementation

There have been relatively few studies investigating the use of lactate as an ergogenic aid and all sources identified through the review process are cited in Table 2.1. While findings are contradictory, there is some evidence indicating supplementation of lactate may indeed be beneficial to physical performance. During steady state exercise to exhaustion, Fahey et al. (1991) concluded compared to a control, supplementation of 216 mg·kg body mass$^{-1}$ sodium lactate in male cyclists (age: 31.2 ± 3.2 years, body mass: 73.5 ± 2.3kg) was beneficial to performance during cycling to exhaustion at 50% $\dot{V}O_{2max}$. They attributed this to lactate preserving blood glucose for the latter stages of the protocol. This may have been facilitated by lactate providing a readily available energy substrate for the working muscles (Hamann, Kelley and Gladden, 2001; Kelley et al., 2002) thus preserving blood glucose and muscle glycogen. Alternatively, it has been suggested supplemented lactate will stimulate gluconeogenesis within the liver thereby replenishing blood glucose levels (Brouns et al., 1995). Together, this indicates lactate supplementation during intermittent sprint exercise such as that seen during invasions sports may be beneficial as the maintained blood glucose and muscle glycogen could be utilised during high-intensity bouts in anaerobic glycolysis.

This effect was demonstrated by Azevedo et al. (2007) who found that the inclusion of a lactate-polymer in a 250ml drink two minutes before, and halfway through steady state, moderate intensity cycling (90mins at 62% $\dot{V}O_{2peak}$) followed by maximum effort, high intensity exercise to exhaustion (86% $\dot{V}O_{2peak}$), resulted in greater performance in six male cyclists (age: 29.7 ± 7.7 years, body mass: 76.3 ± 7.9kg). Duration of high intensity exercise to volitional fatigue was in fact, 25% longer with the lactate solution compared to a glucose-based sports drink. Carbon tracers measured in collected $CO_2$ showed that supplemented lactate was utilised more rapidly and more extensively than the carbohydrates during the exercise protocol. Conversely, Swensen et al. (1994) concluded that when male cyclists performed a single cycle to exhaustion at 70% $\dot{V}O_{2max}$ using a 7% glucose polymer solution either with or without the addition of 3.75g poly-lactate totalling 300 mg·kg body mass$^{-1}$ there was no difference in performance between the two conditions. They did not however include any high intensity element required by Azevedo and colleagues (2007). Additionally, Bryner et al. (1998) also concluded there was no benefit to the supplementation of lactate on peak power during a Wingate performed immediately following constant workload cycling to exhaustion at ~86% maximal heart rate. This may, however, have been due to the mode of prolonged exercise (Table 2.1). Exercise producing a respiratory exchange ratio (RER) of <1.0 indicates fat utilisation in addition to carbohydrate and suggests the intensity was not sufficiently high to increase anaerobic energy production associated with depletion of glycogen stores and diminished high-intensity performance (Nielsen et al., 2011).

During high intensity exercise to exhaustion, Morris et al. (2011) found a positive effect of supplementing lactate when investigating the effect of 120mg·kg body mass$^{-1}$ calcium lactate solution on cycling performance in 11 highly trained competitive cyclists (age: 22 ± 2 years, body mass: 77.1 ± 6.7kg) cyclists. They concluded that compared to water or a placebo solution, TTE was significantly improved ($p \leq 0.05$) by 17% with lactate supplementation (Table 2.1). Similarly, in 19 trained male distance runners performing a single bout of high-intensity treadmill running to exhaustion, van Montfoort et al. (2004)

reported a performance increase after consumption of 400 mg·kg body mass$^{-1}$ sodium lactate and 750ml of water compared to water alone although this was only an improvement of 1.7%. Investigating 40km cycling time trial (TT) performance in seven recreationally active males (age: 22.3 ± 3.3 years, body mass: 79.2 ± 6.37kg), Northgraves et al. (2013) also found a slightly faster performance when supplementing 1115mg (average 14.1 mg·kg body mass$^{-1}$) sodium lactate compared to 300 mg·kg body mass$^{-1}$ sodium bicarbonate or a placebo. In practical terms however, the average improvement was 30s for the lactate supplement which may have quite meaningful implications in competition (Table 2.1). In contrast, Peveler and Palmer (2012) reported no change to 20km cycling TT performance in competitive male cyclists (age: 29.71 ± 3.09 years, body mass: 81.79 ± 15.87kg) when supplementing 21.48 mg·kg body mass$^{-1}$ magnesium and calcium lactate (Table 2.1). Russ, Schifino and Leong (2019) also concluded there was no benefit from the same supplementation on aerobic capacity following a graded cycling to exhaustion in recreationally active men and women.

Unlike caffeine supplementation (Karayigit et al., 2021), there is no clear ergogenic benefit from supplementing either 150 or 300 mg·kg body mass$^{-1}$ calcium lactate in recreationally active individuals (age: 26.0 ± 4.5 years, body mass: 82 ± 11kg) performing 3 x 30s upper body Wingate tests separated by three minutes recovery (Painelli et al., 2014). The authors concluded that for short duration performance testing there is no benefit to be gained from lactate supplementation. Oliveira et al. (2017) also concluded 4 x 30s upper body Wingate performance was not enhanced through lactate supplementation. However, the study design provided a 4 x 125 mg·kg body mass$^{-1}$ dose for the five days prior to testing and therefore does not fall within the supplementation criteria for this research. Additionally, given that blood lactate levels are likely to return to baseline within two hours (Vrese, Koppenhoefer and Barth (1990), and no baseline blood lactate concentrations are reported in the study, it is unlikely blood lactate concentrations were elevated when the testing commenced as any lactate would have been either oxidised within various tissues or converted to glucose in the Cori cycle.

To date there is no research investigating the effect of lactate supplementation on intermittent sprint exercise or sport specific performance indices such as sprint speed, power, and RSA in invasion sport athletes.

***Table 2.2 Summary of studies examining the effects of supplementing lactate solution on physical performance indices***

| Study | N | $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$) | Supplement | Performance measure | Performance changes |
|---|---|---|---|---|---|
| Azevedo et al., 2007 | 6 M | 67.4 ± 3.2 | Lactate containing CytoMaxTM | TTE (86% $\dot{V}O_{2max}$) | TTE 25% ↑ (compared to sports drink) |
| Bryner et al., 1998 | 6 M, 1 F | - | Lactate (2%) | TTE | TTE → |
| Painelli et al., 2014 | 12 M | - | Calcium lactate (150-300 mg·kg body $mass^{-1}$) | Upper body WAnT PP, AP, TW | PP, AP, TW → |
| Fahey et al., 1991 | 5 M | 56.2 ± 2.2 | Sodium lactate (216 mg·kg body $mass^{-1}$) | Moderate intensity cycling | maintenance of blood glucose |
| Morris et al., 2011 | 9 M, 2 F | 60.5 ± 6.6 | Calcium lactate (120 mg·kg body $mass^{-1}$) | TTE (high intensity) | TTE ↑ 17% |
| Northgraves et al., 2013 | 7 M | - | Magnesium lactate and calcium lactate (14.1 mg·kg body $mass^{-1}$) | 40km TT | 1-3% ↑ |
| Oliveira et al., 2017 | 18 M | - | Calcium lactate (500 mg·kg body $mass^{-1}$) | WAnT TW | TW → |
| Peveler and Palmer, 2012 | 7M, 2F | 52.46 ± 11.8 | Magnesium lactate and calcium lactate (21.5 mg·kg body $mass^{-1}$) | 20km TT, AP | TT 0.75% ↓, AP 1.5% ↓ |
| Russ, Schifino and Leong, 2019 | 14 M, 4 F | 46-51 | Magnesium lactate and calcium lactate (16 mg·kg body $mass^{-1}$) | $\dot{V}O_{2peak}$, Power at OBLA | $\dot{V}O_{2peak}$ ↑ 1.9%, Power at OBLA ↑12.7% |
| Swensen et al., 1994 | 5 M | 67.4 | Poly lactate/ glucose mix (300 mg·kg body $mass^{-1}$) | TTE (moderate intensity) | TTE → (<0.1%) |
| van Montfoort et al., 2004 | 19 M | - | Sodium lactate (300 mg·kg body $mass^{-1}$) | TTE (high intensity) | TTE ↑ 1.7% |

## 2.7 Physical conditioning to improve performance in invasion sport

In order to cope with the physical demands of their sport, athletes should ideally utilise a training modality that replicates the metabolic stresses experienced during game play (Gabbett, 2010; Smart et al., 2014; Vaz et al., 2014). This includes the ability to repeatedly perform high-intensity activities that directly transfer to match play for invasion sports rugby union, football and hockey, and that may be critical to match success (Brocherie et al., 2015; Iaia, Rampinini and Bangsbo, 2009). High-intensity training (HIT) involves repeated bouts of exercise interspersed with longer periods of rest (Fox et al., 1973). It has been shown repeatedly to induce meaningful physiological and metabolic changes in skeletal muscle leading to improvement in aerobic capacity (Babraj et al., 2009; Burgomaster et al., 2005; Burgomaster et al., 2008; Helgerud, 2001; Seiler 2005; Seiler, 2013; Smilios et al., 2017; Smith, Coombes and Geraghty, 2003) and endurance performance improvements are comparable to those following traditional endurance training, even though training volume is significantly reduced (Astorino et al., 2012; Burgomaster et al., 2008; Gibala et al., 2012; Hazell et al., 2010; Holloway, Bliss and Hearon, 2017; Little et al., 2010). While the two modalities are comparable, it has also been suggested that the performance improvements attributable to HIT are mainly enhancement of skeletal muscle respiratory capacity (Jacobs et al., 2013), whereas the improvements more common with traditional endurance training were predominantly attributable to haematological adaptations (Montero et al., 2015). The metabolic stress experienced by the athlete depends on several factors including exercise intensity, duration and volume, as well as the mode, intensity and duration of the recovery periods (Buchheit and Laursen, 2013; Jones and Vanhatalo, 2017).

### 2.7.1 High intensity training

HIT falls broadly into two main categories; high intensity interval training (HIIT) and sprint interval training (SIT). For HIT to be effective, there must be sufficient metabolic stress to elicit adaptation, therefore exercise must be performed at more than 90% $\dot{V}O_{2max}$ (Millet et al., 2003).

HIIT can be defined as submaximal exercise at an intensity ≥ 80% of $HR_{max}$ or > 90% $\dot{V}O_{2max}$, and SIT as shorter, supramaximal 'all out' bouts (Gibala and Jones, 2013; Millet et al., 2003; Weston et al., 2014).

#### *2.7.1.1 High intensity interval training*

HIIT consists of longer duration work periods ranging from one to four minutes and requires an accumulation of five to ten minutes of exercise at the crucial intensity (Smilios et al., 2017). An example of this is the study conducted by Little et al. (2010) where they employed 8 – 12 repetitions of pedalling on a cycle ergometer for 60 seconds at an intensity equal to the maximum power output achieved during a ramp $\dot{V}O_{2peak}$ test (~355W). Performance levels increased significantly ($p < 0.05$) for both 2km (11%) and 30km (9%) time trials following the intervention. For optimal increases in aerobic capacity, as much of the training period as possible should be spent at this intensity to ensure HR and $\dot{V}O_2$ remain elevated between exercise bouts (Smilios et al., 2017). This can be achieved through manipulation of the training protocol by either inclusion of active recovery periods where a reduced exercise intensity is maintained (Yamagishi and Babraj, 2016), or through variations of the work to rest ratios (Smilios et al., 2017). Smilios et al. (2017) reported that a 4:2 work to rest ratio relied more heavily on anaerobic glycolysis than a 4:4 ratio as $[La^-]b$ at the beginning of the next bout of exercise was greater. This however seems flawed logic as the accumulated lactate will have had less time to dissipate in two compared to four minutes of recovery. They also reported no significant difference between 2 or 4 minutes of recovery regarding oxygen consumption in the final three minutes of the bouts which was similar to the findings of Seiler and Hetlelid (2005). HIIT also relies on the maintenance of a given predetermined intensity which does not reflect the maximal efforts required during invasion sport match play.

*2.7.1.2 Sprint interval training*

What may then be more sports specific, is SIT which is characterised by the 'all-out' or 'supramaximal' efforts at a pace ≥ to that which would elicit $\dot{V}O_{2peak}$ and lasting ≤ 30 seconds (MacInnis and Gibala, 2017). Examples of this type of training were reported by Gibala et al. (2006) and Jakeman, Adamson and Babraj (2012). In the first of these studies, the SIT protocol required four to six supramaximal bouts of 30s cycling at ~250% $\dot{V}O_{2peak}$ separated by 4 min recovery. Training improved performance in both 50 and 750 kJ cycling time trials in a similar way to the traditional endurance training group but with ~90% less training volume. Maximal activity of cytochrome c oxidase (COX), which signals increases in muscle oxidative capacity, were also similar between groups, as were muscle buffering capacity and glycogen content. Jakeman and colleagues reduced training volume even further using ten repeated supramaximal efforts of only six seconds per sprint equating to a total maximal effort training time of six minutes over the two-week study. There was a significant reduction in the time it took participants to complete a self-paced 10km time trial (TT) following the training which had a significant positive relationship with the change in OBLA (also called maximal lactate steady state - defined as the exercise intensity that raises $[La^-]b$ to 4 $mmol \cdot l^{-1}$) which improved by 17%. Time to exhaustion had improved 4% in the SIT group after the two weeks although this was not statistically significant.

With SIT protocols as short as six seconds bouts producing similar results to longer duration work, it could be assumed that the adaptations are occurring due to the metabolic demands of the early phase of the sprints (Jakeman, Adamson and Babraj, 2012) and in a recent study by Yamagishi and Babraj (2017), it was shown that a 15s SIT protocol was just as effective as 30s at improving $VO_{2peak}$, TTE, 10km cycling TT and more so at increasing anaerobic power output. Hazell et al. (2010) also found similar results for $\dot{V}O_{2max}$, 5km cycling TT, and power output when comparing 10s and 30s Wingate bouts interspersed with four minutes recovery.

### 1.1.1 Effect of recovery duration and mode during HIT

Kavaliauskas, Aspe and Babraj (2015) investigated the adaptation differences when work to rest ratios were altered during SIT. Participants performed six SIT sessions over two weeks, each consisting of six bouts of ten seconds supramaximal cycling. Rest periods were either 30, 80 or 120 seconds in duration equating to 1:3, 1:8 and 1:12 work to rest ratios respectively. They reported that a 1:3 work to rest ratio elicited greater performance gains in aerobically dependent tests such as 3km running TT and incremental TTE than the longer duration rests. The 1:3 group made significant improvements of 3.1 ± 4.0% and 6.4 ± 6.3% for the two performance measures. The 1:8 group also made a significant improvement in the TTE test with a group mean improvement of 4.4 ± 2.7%. In the peak power assessment however, the 1:3 group had very little change (0.3 ± 4.1%) whereas the 1:8 and 1:12 groups made significant power increases with 4.6 ± 4.2% and 5.3 ± 5.9% respectively. This suggests that, for team sports where it is desirable to develop both aerobic and anaerobic capacity, use of a SIT protocol utilising a 1:8 work to rest ratio may be beneficial to performance measures. Supporting this, Yamagishi and Babraj (2017) found that a SIT protocol using repeated 15s supramaximal bouts lead to significant aerobic adaptations, with increases in TTE, 10km cycling TT, and $\dot{V}O_{2peak}$ after nine weeks, as well as significant power output increases measured through a 3-minute maximal effort critical power test. When recovery periods are increased in relation to sprint duration (1:8 and 1:12), power development during a 30s Wingate test is significantly increased following training (Kavaliauskas, Aspe and Babraj, 2015) which may be due in part to increased resting muscle glycogen levels (Jakeman, Adamson and Babraj, 2012). Gibala et al. (2006) reported a 28% increase in muscle glycogen content following SIT (30s Wingate interspersed with low-intensity (30W) recovery) when using a W:R ratio of 1:8 although no power output tests were conducted. Replicating Gibala and colleagues' protocol however, Hazell et al. (2010) found significant increases in both peak power and average power of 9.5% and 12.1% respectively following the training. This process is reversible and following two weeks of inactivity, glycogen levels within the myofibrils can decrease by approximately 50% (Nielsen et al., 2010b).

#### 1.1.1.2 Recovery mode

When work:rest ratios (1:1 – 1:5) mean that recovery time is relatively short compared to the supramaximal bout, it has been shown that active recovery has a detrimental effect on repeated sprint performance with greater decline in the bouts that follow (Buchheit et al., 2009; Dupont et al., 2007; Spencer et al., 2006; Spencer et al., 2008). TTE is also reduced compared to passive recovery for recovery intensities ranging from 20–50% $\dot{V}O_{2max}$ (Dupont, Blondel and Berthoin, 2003; Dupont et al., 2004; Dupont et al., 2007; Spencer et al., 2006, 2008). The explanation for this decline offered by several authors (Buchheit et al., 2009; Dupont, Blondel and Berthoin, 2003; Dupont et al., 2004; Dupont et al., 2007; Spencer et al., 2006; Spencer et al., 2008) is lack of restoration of intramuscular PCr. With continued muscle activation during active recovery still requiring elevated energy and oxygen supply, less ATP is available to resynthesise PCr and less oxygen to replenish haemoglobin and myoglobin oxygen.

Contrary to this, relatively long recovery periods, with work:rest ratios of 1:8 - 1:12, appear to enhance overall sprint performance (Bogdanis et al., 1996b; Connolly Brennan and Lauzon, 2003; Spierer et al., 2004). Power output was better maintained with active recovery at 80W (Connolly et al., 2003), and with active recovery ranging from 28-40% $\dot{V}O_{2max}$, mean power output and total work were greater compared to passive recovery (Bogdanis et al., 1996b; Spierer et al., 2004). Unlike the relatively short rest periods which do not allow for sufficient PCr renewal, maintaining an elevated aerobic demand seems to be beneficial for aerobic power in subsequent bouts of supramaximal activity.

The continuing work being performed keeps HR elevated (Bogdanis et al., 1996b; Yamagishi and Babraj, 2016) which in turn, ensures more blood flow through the muscles, and therefore more oxygen availability for PCr restoration (Hastler, Hogan and Richardson, 1999) following the initial cardio-dynamic phase (Krustrup et al., 2009). Greater PCr resynthesise is highly correlated ($r = 0.84$) with maintenance of power output during the first third of a 30s sprint (Bogdanis et al., 1996a) and this may become increasingly relevant as number of sprints is increased (Tomlin and Wenger, 2001). However, during aerobic HIT, Smilios et al. (2017) state that if the recovery period is too long, there is a

decrease in HR and $\dot{V}O_2$ at the start of the next bout meaning there may be a reduction in the duration spent exercising at sufficiently high intensity to elicit the desired physiological adaptations.

### 2.7.2 Physiological and metabolic adaptations induced by HIT

#### *2.7.2.1 Mitochondrial biogenesis*

Skeletal muscle mitochondrial density regulates substrate metabolism during low or moderate-intensity exercise (Egan and Zierath, 2013) such as might be performed between high-intensity bouts during field sports like rugby, football and field hockey (Drust, Atkinson and Reilly, 2007; Faiss, Girard and Millet, 2015; Lemos et al., 2017; Paul, Bradley and Nassis, 2015). As mitochondrial content increases, skeletal muscle becomes more efficient at fat oxidation leading to a proportional decrease in carbohydrate utilisation (Egan and Zierath, 2013). Therefore, following training, glycogen stores are better preserved (Joyner and Coyle, 2008) and can be expended during the intermittent high-intensity bouts which rely more heavily on glycolysis (Casey et al., 1996; Howlett et al., 1998). Even a single HIT session will activate signalling pathways associated with mitochondrial biogenesis (Gibala et al., 2009, Little et al., 2011), with acute and transient elevation of mRNA expression after each session of HIT (Perry et al., 2010). When regularly and repeatedly activated, there will be a sustained increase in mitochondrial content and function (Christensen et al., 2016; Coffey and Hawley, 2007).

Upregulation of peroxisome proliferator-activated receptor gamma coactivator 1α (PGC-1α), a major regulator of mitochondrial biogenesis, is reported as the main driver for increasing skeletal muscle mitochondrial content following exercise (Adhihetty et al., 2003; Gibala et al., 2012; Laursen, 2010; Valero-Grinan, 2014). Both Gibala et al. (2009) and Little et al. (2011) noted elevated PGC-1α mRNA expression within skeletal muscle following a single HIT session consisting of 4 x 30s Wingate sprints separated by 4-minute recovery periods, and the increase seen by Little and colleagues (~66%) was greater than that elicited in their earlier work (~55%) (Little et al., 2010) on steady-state moderate-intensity cycling (65% $\dot{V}O_{2peak}$). Employing HIT is therefore a powerful

stimulus to elicit increases in skeletal muscle mitochondrial content (MacDougall et al., 1998) and this is because levels of PGC-1α gene expression are associated with the intensity of the exercise performed (Egan et al., 2010; Nordsborg et al., 2010). Egan et al. (2010) demonstrated that, following 36 minutes of high-intensity steady-state cycling (80 % $\dot{V}O_{2peak}$), expression of PGC-1α increased to a greater extent than a longer duration of moderate intensity exercise (70 minutes, 40 % $\dot{V}O_{2peak}$). Investigating HIIT, Nordsborg et al. (2010) also found a greater expression of PGC-1α through higher intensity work when intervals were performed at 70 and 85 % $\dot{V}O_{2max}$. When volume of work is matched, HIT can therefore be more effective, both in terms of training commitment and metabolically, than traditional steady-state endurance training (Di Donato et al., 2014; MacInnis and Gibala, 2017).

Adaptations are also related to intensity in terms of the magnitude of metabolic stress, with higher intensity exercise generating a greater metabolic signal than moderate-intensity exercise (Egan and Zierath, 2013). Skeletal muscle motor units are recruited in proportion to the intensity of the exercise. Employing a maximal effort protocol replicating the most demanding elements of invasion sport, will recruit more Type II fibres than low intensity training and may therefore lead to increased AMPK levels resulting in increased mitochondrial content. This is supported by Kavaliauskas, Steer and Babraj (2017) who reported a non-significant increase in $\dot{V}O_{2peak}$ after four weeks of SIT training using a reduced intensity during repeated Wingate tests meaning metabolic demand may not have been optimised to induce mitochondrial biogenesis and develop cardiorespiratory function.

ATP concentrations fell by ~40% following 4 x 30s bouts of SIT (Gibala et al., 2009) and phosphorylated AMPK and p38 MAPK levels were significantly elevated which further supports the idea that response is associated to intensity. However, the same authors reported CaMKII levels were not significantly elevated following the same protocol. Calcium-sensitive protein kinases require an increased calcium ($Ca^{2+}$) flux following prolonged muscle contraction to activate them (Adhihetty et al., 2003; Kang et al., 2009; Laursen, 2010) and it may be the four repetitions were not sufficient. That is not to say

however, that CaMKII levels cannot be elevated through SIT, it may just require a higher volume of exercise. Place et al. (2015) found that six repetitions of 30s Wingate tests elicits a ROS-dependent fragmentation of the ryanodine receptor (RyR) which may be linked to elevated intracellular calcium concentrations which is a signal for mitochondrial biogenesis and therefore likely to elevate levels of CaMKII.

The activity levels of mitochondrial enzymes can also be used to indicate an increase in mitochondrial biogenesis (MacInnis and Gibala, 2017). Elevated maximal activity of citrate synthase (CS) is one example of this. Perry et al. (2010) reported CS activity levels to increase consistently over seven sessions of HIT with CS maximal activity significantly increased from baseline after only three sessions. Care must be taken when designing training programmes however as increasing training volume from 2-3 sessions per week to 7-8 sessions has a detrimental effect on CS maximal activity (Hatle et al., 2014). Increased maximal activity of cytochrome c oxidase (COX) expression following low-volume HIT was also noted and was similar to the response from much higher volume steady-state moderate activity (Daussin et al., 2008). Measuring changes in either CS or COX, it was estimated that mitochondrial content increases after 6-7 sessions of HIT by approximately 25–35% (Burgomaster, Heigenhauser, and Gibala, 2006; Gibala et al, 2006; MacInnis et al., 2016; Talanian et al., 2006).

#### *2.7.2.2 Skeletal muscle remodelling following HIT*

Regular exercise elevates specific proteins involved in cellular glucose uptake including glucose transporter-4 (GLUT4) and hexokinase II (HKII) (Zierath, 2002). Following two weeks of HIT, Little et al. (2010) reported an increase of 17% in resting muscle glycogen levels and a 119% increase in protein content of the insulin-regulated glucose transporter, GLUT4. During supramaximal bouts, there is a greater rate of glycogenolysis and glycolysis in the initial 10s than from 10 - 20s and a greater decrement of ATP and PCr (Bogdanis et al., 1998). Even during 6s bouts, similar levels of skeletal muscle glycogen are utilised (Gaitanos et al.,1993). This elevated turnover elicits a super-compensatory response which leads to elevated resting skeletal muscle

glycogen levels post training (Burgomaster et al., 2005; Gibala et al., 2006; Rodas et al., 2000) with an increase of 17% in resting muscle glycogen levels following two weeks of HIIT (Little et al., 2010), and a 28% increase following SIT (Gibala et al., 2006).

Following 60 minutes cycling at 60% $\dot{V}O_{2max}$, Koval et al. (1998) reported HKII levels elevated by 114% in skeletal muscle. Increased GLUT4 allows greater transport of glucose into the muscle from circulating blood, and this can either be stored as glycogen for future use or converted to glucose 6 phosphate by HKII during glycolysis (Figure 2.5).

Skeletal muscle mitochondrial density regulates substrate metabolism during low or moderate intensity exercise (Egan and Zierath, 2013) such as might be performed between high intensity bouts during invasion sports like rugby, soccer and field hockey (Drust, Atkinson and Reilly, 2007; Faiss, Girard and Millet, 2013; Lemos et al., 2017; Paul, Bradley and Nassis, 2015). As mitochondrial content increases, skeletal muscle becomes more efficient at fat oxidation leading to a proportional decrease in CHO utilisation (Egan and Zierath, 2013; Talanian et al., 2006). Therefore, following training, glycogen stores are better preserved (Joyner and Coyle, 2008) and can be expended during the intermittent high intensity bouts which rely more heavily on glycolysis (Casey et al., 1996; Howlett et al., 1998).

### 2.7.3 Effect of HIT on Lactate Kinetics

HIT has also been shown to alter lactate kinetics (Best et al., 2013; Gharbi, 2008; Jakeman, Adamson and Babraj, 2012), and this change is associated with enhanced endurance capacity (Bergman et al., 1999). While endurance training will also increase the rate of lactate clearance (Bassett and Howley, 2000; Jones and Carter, 2012; Messonier et al., 2006), it has been demonstrated that HIT is more effective at this (Gharbi et al., 2010). Following training, at a given moderate intensity, lactate production will be reduced (Bergman et al., 1999). This may be due to the increased utilisation of fat and reduced CHO use (Egan and Zierath, 2013), or an increased use of pyruvate in oxidative metabolism (Burgomaster et al., 2008). The intensity at which metabolic markers (LT and MLSS) are evident will be increased as the lactate

produced is transported (Burgomaster et al., 2007) and utilised more efficiently (Bosquet et al., 2003; Holloway, Bliss and Hearon, 2017; van Hall et al., 2003). This change in lactate shuttle capacity is facilitated through increased levels of MCT1 and MCT4 (Pilegaard et al., 1999b) leading to greater exercise tolerance at high intensities (Holloway, Bliss and Hearon; 2017; Messonnier et al., 2001; Messonnier et al., 2002). Training has been shown to increase the expression of MCT1 in both muscle sarcolemma and mitochondria (Bergman et al., 1999), although Brooks (2002) states this is not also true for MCT4. However, Burgomaster et al. (2007) reported an increase in skeletal muscle of both MCT1 and MCT4 content after six weeks of SIT. Increases in MCT 1 and MCT4 content and activity were also reported by Pilegaard et al. (1999) following a HIT protocol and this was associated with improved aerobic and anaerobic capacity. These increases in MCT concentrations would elicit an increased uptake of lactate in skeletal muscle (Baker, McCullagh and Bonen, 1998) and allow an increased work intensity or time to reach defined $La^-$ thresholds (Hurley et al., 1984) which is in part due to use of the available lactate (Bosquet et al. 2003). These changes therefore may increase the usefulness of lactate as an ergogenic aid during invasion sport. The point at which $[La^-]b$ starts to noticeably accrue is primarily determined by mitochondrial volume density (Gibala, Bostad and McCarthy, 2019), and because of the training induced increase in this, $[La^-]b$ at a given intensity is reduced. This would permit an increased duration of activity at a greater percentage of $\dot{V}O_{2max}$ (Joyner and Coyle, 2008) which would also be beneficial to invasion sport athletes.

### 2.7.4 Effect of HIT on invasion sport performance

#### *2.7.4.1 Aerobic capacity*

During exercise, endurance performance is dependent on the cardiopulmonary system transporting oxygenated blood to the working skeletal muscles (Gibala, Bostad and McCarthy, 2019). A key determinant of this ability is the maximal rate at which oxygen can be delivered ($\dot{V}O_{2max}$) (Lundby and Robach, 2015). It also depends in part on the oxidative capacity of those muscles of which mitochondrial content is a critical component (Gibala, Bostad and McCarthy,

2019). Endurance performance can be enhanced through both traditional moderate intensity continuous training (MICT) and high intensity training (HIT) (Jacobs et al., 2013; Murias, Kowalchuk and Paterson, 2010). Adaptations affecting aerobic capacity can be defined as either central or peripheral in nature (Raleigh et al., 2018). These adaptations include: increased left ventricular mass and maximal cardiac output ($Q_{max}$) (Baggish et al., 2008; Montero, Diaz-Canestro and Lundby, 2015; Montero et al., 2015); an increase in total plasma volume and red blood cell volume (Bonne et al., 2014; Swaka et al., 2000); a decrease in vascular resistance (Weng et al., 2013); increased skeletal muscle capillarisation (Montero et al., 2015; Murias et al., 2011); increased mitochondrial content and function (Jacobs et al., 2013b; Jacobs and Lundby, 2013; Jørgensen et al., 2007; Little et al., 2010; Montero et al., 2015); improved oxygen extraction (Murias, Kowalchuk and Paterson, 2010); and more efficient blood flow distribution (Kalliokoski et al., 2001).

It is well documented that short duration high intensity training elicits a similar or greater increase in aerobic capacity to that induced by MICT (Astorino and Schubert, 2014; Babraj et al., 2009; Burgomaster et al., 2005, 2007, 2008; Cocks et al., 2013; Gibala, Bostad and McCarthy, 2019; Gist et al., 2014; Helgerud, 2001; Hood et al., 2011; Jacobs et al., 2013; Jakeman, Adamson and Babraj, 2012; Little et al., 2010; Milanović, Sporiš and Weston, 2015; Murias, Kowalchuk and Paterson, 2010; Scribbans et al., 2014; Seiler, 2005, 2013; Smilios et al., 2018; Smith, Coombes and Geraghty, 2003; Weng et al., 2013). An additional benefit of HIT is that these adaptations occur even though training volume and time requirement are significantly reduced (Astorino et al., 2012; Burgomaster et al., 2008; Gibala and Jones, 2013; Gibala et al., 2012; Gibala, Bostad and McCarthy, 2019; Gist et al., 2014; Hazell et al., 2010; Holloway, Bliss and Hearon, 2017; Little et al., 2010; Parker et al., 2017). A systematic review of 32 studies conducted by Weston et al. (2014) found previous research reported a mean improvement in $\dot{V}O_{2max}$ of 6.2% following HIT interventions lasting between two and ten weeks.

In contrast to MICT, adaptations are more likely to be peripheral for short term training (Jacobs et al., 2013; Raleigh et al., 2018) with central adaptations not seen without more prolonged training periods of six weeks or more (Astorino et

al., 2017; Gillen et al., 2016; Matsuo et al., 2014; Warburton et al., 2004). This is supported by Kavaliauskas, Steer and Babraj (2017) who reported a non-significant increase in $\dot{V}O_{2peak}$ after four weeks of HIIT but significantly increased performance measures. However, one study (Astorino et al., 2017) reported a modest increase in $Q_{max}$ following short duration HIT. Peripheral adaptations include: increased mitochondrial content (Burgomaster et al., 2005, 2006, 2008; Gibala et al., 2006; Gillen et al., 2016; Hood et al., 2011; Little et al., 2010, Ma et al., 2013; Perry et al., 2008; Talanian et al., 2006); increased mitochondrial function (Jacobs et al., 2013, Vincent et al., 2015, Granata et al., 2016); greater capillary density (Raleigh et al., 2018; Tan et al., 2018); increased rate of oxygen extraction (Jones, Hamilton and Cooper, 2012); and increased oxidative capacity of skeletal muscle (Burgomaster et al., 2008; Gillen et al., 2013, 2014; Jacobs et al., 2013; Ma et al., 2013; MacPherson et al., 2011; Perry et al., 2008; Talanian et al., 2006). The increase in skeletal muscle oxidative capacity is reflected in biochemical measurements of maximal protein activity/content of mitochondrial enzymes, and markers of mitochondrial respiration (Bishop et al., 2019; MacInnis and Gibala, 2017).

HIT has been shown to significantly improve endurance performance during both continuous and intermittent activities in invasion sport athletes (Dobbin et al., 2020; Elumalai et al., 2020; Kelly et al., 2018; Kelly et al., 2021; Taylor et al., 2015). Increased aerobic capacity is associated with greater work intensity, greater distance covered, and also more high intensity running and sprinting during matches (Castagna et al., 2010; Gabbet et al., 2013; Helgerud et al., 2001; Jennings et al., 2012; Mooney et al., 2011; Rebelo et al., 2014), and greater RSA (Aziz, Chia and Teh, 2000). HIIT has also been employed as a training modality to improve critical power (CP) and does so effectively within four weeks (Kavaliauskas, Steer and Babraj, 2017 Kendall et al., 2009). CP is an important indicator of potential work rate during invasion sport and increasing this is desirable for improving performance during matches (Jones and Vanhatalo, 2017)

#### *2.7.4.2 Anaerobic capacity*

Anaerobic capacity is a combination of the PCr and anaerobic glycolytic pathways. SIT has been shown to significantly improve several anaerobic performance indicators such as time to exhaustion during high intensity exercise (Kavaliauskas, Steer and Babraj, 2017); power output (Burgomaster et al., 2007; Jakeman, Adamson and Babraj, 2012; MacDougall et al., 1998); critical power (Kavaliauskas, Steer and Babraj, 2017); RSA (Edge et al., 2005; Viaño-Santasmarinas et al., 2018); sprint speed (Sperlich et al., 2011); and vertical jump height (Barnes et al., 2013; García-Pinillos et al., 2017).

RSA has been significantly improved through HIT (Bravo et al., 2007; Botoniss et al., 2019; Buchheit et al., 2010; Taylor et al., 2015; Tønnessen et al., 2011) and these adaptations occur within a short period of time (Viaño-Santasmarinas et al., 2018). Edge et al. (2005) found that in amateur female invasion sport athletes, cycling based HIIT improved RSA in terms of total work done by 13% after five weeks of three sessions per week. The adaptations leading to improved RSA are a combination of aerobic and anaerobic pathways. Enhanced aerobic capacity increases the rate of PCr resynthesis through elevated PCr shuttle activity (Figure 2.3) meaning levels are more fully restored at the beginning of each repetition which is associated with performance maintenance in the later sprints (Bogdanis et al., 1996a; Little and Williams, 2007). Power output in each repetition is also elevated aiding repeated sprint performance. Availability of skeletal muscle glycogen is important for the generation of peak power (Casey et al. 1996) and the increased resting glycogen levels following HIT are likely facilitating this increased power (Jakeman, Adamson and Babraj 2012). Burgomaster et al. (2005) also attributed a two-fold increase in anaerobic endurance capacity to this increase in skeletal muscle glycogen stores. Additionally, there is an increase in lactate dehydrogenase (LDH) activity in skeletal muscle following SIT (Linossier et al., 1997) and anaerobic glycolysis is sustained for longer by these increased concentrations (van Hall, 2000; Xu et al., 2016).

HIT has been shown to significantly improve WAnT peak power, average power, and fatigue index results (Kelly et al., 2021). Following 12 sessions of 4 - 6 x 15s SIT, Zelt et al., (2014) reported significant improvements in both peak

and average power during a WAnT. Anaerobic glycolysis plays a critical role in energy supply during short duration high intensity exercise (Beneke et al., 2002) so it is likely that SIT improves the rate of anaerobic glycolysis. There is an increase in phosphofructokinase (PFK) activity in skeletal muscle following SIT (Fournier et al., 1982; Linossier et al., 1997) which contributes to anaerobic energy production during invasion sports. Mohr et al. (2016) demonstrated in invasion sports, total sprint distance and the average duration of individual sprints during high intensity periods of match play was significantly correlated ($r = 0.46 - 0.48$; $p < 0.05$) to PFK activity levels. Likewise, Iaia et al. (2011) associated PFK levels with several sprint indicators including RSA.
These increases in power should translate to practical performance indices too since the ability to generate muscular power is strongly correlated to sprint speed in invasion sport athletes (Vescovi and Mcguigan, 2008). Improvements in maximal running speed have being reported following HIT (Elumalai et al., 2020; Thom, Kavaliauskas and Babraj, 2019; Sperlich et al., 2011; Taylor et al., 2015; Taylor et al., 2016; Tønnessen et al., 2011) although other studies reported no change in maximal running speed following HIT (Dobbin et al., 2020; Kelly et al., 2021). Ferrete et al. (2014) and Tønnessen et al. (2011) also concluded HIT can improve vertical jump performance although again both Kelly et al. (2021) and Dobbin et al. (2020) reported unchanged jumping ability following HIT.

Together, these findings indicate that SIT may prove to be a valuable training modality for invasion sport athletes whose performance is determined by both their aerobic and anaerobic capacities, as well as their ability to metabolise lactate. The increases in lactate kinetics may also enhance any ergogenic effect of supplementing lactate.

## 2.8 Conclusion

Invasion sports such as rugby union, soccer, and field hockey involve intermittent bouts of high intensity activity divided by longer periods of low to moderate activity recovery (Faiss, Girard and Millet, 2013; Lemos et al., 2017; Wehbe, Hartwig and Duncan, 2014). This activity pattern places a high demand on both the aerobic and anaerobic metabolic pathways (Osgnach et al., 2010; Paul, Bradley and Nassis, 2015; Stolen et al., 2005), with average match intensity of ~85% $HR_{max}$ leading to elevated blood lactate concentrations (Aslan et al., 2012; McLean, 1992; Kusnanik, Rahayu and Rattray, 2018). Maintenance of performance level during maximal effort bouts play a decisive role in match outcome (Bishop et al., 2006, 2011; Gabbett, 2010b; Girard, Brocherie and Millet, 2015; Rampinini et al., 2007) and these bouts are fuelled largely through anaerobic glycolysis (Nielsen et al., 2010). Glycogen levels are depleted over the duration of matches and result in diminished performance levels (Nielsen et al., 2010, 2011; Spencer et al., 2005).

Working skeletal muscle is the predominant site of lactate production (Bergman et al., 1999) and concentrations rise exponentially above exercise intensities of between 70% - 90% $\dot{V}O_{2max}$ (Jones and Vanhatalo, 2017). The ability to rapidly convert pyruvate produced in glycolysis to lactate, and transport this out of the working muscle may play an important role in maintenance of performance during invasion sports. Increasing both the lactate transport capacity and lactate metabolism potential, is likely to benefit invasion sport athletes. Studies (Table 2.1) have demonstrated that increasing the availability of lactate through ingestion of a lactate solution can benefit high intensity performance. This occurs principally in individuals with highly developed aerobic capacity, through provision of a metabolic substrate which is used more rapidly and fully than glucose (Azevedo et al., 2007). Provision of training which would help develop these aspects of fitness would be beneficial to invasion sport athletes. The metabolic stress experienced by the athlete depends on several factors including exercise intensity, duration and volume, as well as the mode, intensity and duration of the recovery periods (Buchheit and Laursen, 2013; Jones and Vanhatalo, 2017). Adaptations are also related to intensity in terms of the magnitude of metabolic stress, with higher intensity exercise generating a

greater metabolic signal than moderate intensity exercise (Egan and Zierath, 2013). Therefore, due to the supramaximal nature of SIT, it is likely to be a valuable training modality for invasion sport athletes whose performance is determined by both their aerobic and anaerobic capacities. Additionally, SIT reflects the repeated maximal efforts during invasion sports and training should ideally replicate the requirements of the sport (Hodun et al., 2016; McMahon and Kennedy, 2019).

With SIT protocols as short as six seconds bouts producing similar results to longer duration work, it could be assumed that the adaptations are occurring due to the metabolic demands of the early phase of the sprints (Jakeman, Adamson and Babraj, 2012), and in a study by Yamagishi and Babraj, (2017), it was shown that a 15s SIT protocol was just as effective as 30s at improving $\dot{V}O_{2peak}$, TTE, 10km cycling TT, and even more so at increasing anaerobic power output. Hazell et al. (2010) also found similar results for $\dot{V}O_{2max}$, 5km cycling TT, and power output when comparing 10s and 30s Wingate bouts interspersed with four minutes recovery. When volume of work is matched, SIT can therefore be more effective, both in terms of training commitment and metabolic demand, than MICT (Di Donato et al., 2014; MacInnis and Gibala, 2017).

# Chapter 3

# General Methods

## 3.1 Participants

Participants in all studies had at least three years playing experience in their respective invasion sport and were contacted via email, letter or verbal communication. Participants were excluded from studies if they had pre-existing musculoskeletal injuries which would affect performance or had undertaken sprint interval training (SIT) in the previous six months. All participants were required to complete a physical activity readiness questionnaire (PAR-Q) to ensure no contraindications for the protocol were present. They were fully informed of the requirements of the study both in writing, and again verbally before signing informed consent. In Study 2, where participants were under the age of 16, consent was sought from a parent or guardian as well as from the participant. Ethical approval was granted for all studies by Abertay University's School of Applied Sciences Ethics Committee and research was carried out in line with the Declaration of Helsinki (www.wma.net, 2013).

### 3.1.1 Study 1

Lactate absorption

Data was collected for 10 male participants to determine the length of time required for ingested lactate to be detected in the blood, and therefore be available for utilisation as an energy substrate. This was double the number of participants used by Fahey et al. (1991) but similar to sample size of Morris and colleagues (2011) who used 11 participants.

Ingested lactate can be processed by the body in several ways. A small percentage will pass through the digestive system and be excreted from the body unutilised (Duncan, Louis and Flint, 2004). Within the digestive tract, intestinal microflora can also synthesise butyrate from lactate, further reducing the quantity of lactate that will reach the blood (Bourriaud et al., 2005). Once in the blood, lactate may be converted to glucose by the liver in the process of gluconeogenesis (Brooks, 2002b; Yoon et al., 2001). Tissue with high oxidative metabolism and high concentrations of the H form of lactate dehydrogenase (LDH) such as cardiac and Type I skeletal muscle fibres, will convert lactate to pyruvate for further metabolism within the tricarboxylic acid (TCA) cycle (Gray,

Tompkins and Taylor, 2013). Lactate may also be utilised directly by skeletal muscle, cardiac muscle, and astrocyte cells of the brain (Brooks et al., 1999a; Brooks et al., 1999b; Brooks, 2002b; Dubouchaud et al., 2000; Lemire, Mailloux and Appanna, 2008). Due to the multifaceted and complex fate of ingested lactate, there is no simple model which can be used to calculate whole body lactate kinetics, so blood lactate concentration was reported as group mean values at each time point. Previous literature indicates no risk of lactic acidosis when lactate solution is consumed in quantities below that which would cause gastrointestinal efflux (de Vrese and Barth, 1991).

Performance testing

To assess the effectiveness of lactate as a supplemented energy source the Bath University rugby shuttle test (BURST) was used. As per Roberts et al. (2010a) initial investigations using this protocol, eight healthy and recreationally active male participants were recruited. The same activity level inclusion criteria as part 1 of the study was used but all participants were sub-elite rugby union players with varying degrees of current participation from zero to three times per week. The randomised controlled trial (RCT) is considered the gold standard (Hecksteden et al., 2018), and this randomised crossover trial can be considered as such (Hopewell et al., 2010). By participants acting as their own control and undertaking each of the intervention conditions, some potential confounders such as training status, nutritional intake and sleep patterns are lessened (Thiese, 2014), and fewer participants are required to achieve sufficient statistical power (Hecksteden et al., 2018).

### 3.1.2 Study 2

Participants were recruited from the Under-16 players of Dundee Football Club Academy. Whilst random allocation to treatment and control groups from within the squad would have been desirable to meet the RCT model, it is not always possible when working in a club environment (Campbell et al., 2012; Grimshaw et al., 2000; van Breuken and Candel, 2012). As an alternative, a cluster randomised control was used in which players in the treatment group were recruited from the 2015-16 squad while the control group was recruited from the

2016-17 squad. The benefit of this type of design is the avoidance of contamination between treatment and control group when they are training in the same environment, and it is desirable when exclusion of some participants from the potential benefits of the training intervention would be counterproductive (Hecksteden et al., 2018). A total of 16 players were recruited with eight in both the treatment and control groups. There are two professional soccer teams in the local area who each have a squad of approximately 15 players in this age group. Therefore, sample size represented ~25 - 30% of players of that age and standard in the area. This sample size is also similar to previously published high intensity training (HIT) studies (Abderrahman et al., 2012; Bayati et al., 2011; Berger et al., 2006; Warburton et al., 2004).

### 3.1.3 Study 3

Female athletes were recruited from a university women's hockey team. The change of participant sex should not alter the adaptations seen as it has been shown magnitude of change between male and female participant is similar following HIT for maximum oxygen uptake ($\dot{V}O_{2max}$), power output and endurance performance (Astorino et al., 2011; Scalzo et al., 2014). It has also been reported that reliability differences observed between sexes during power testing are negligible (Hopkins, Schabort and Hawley, 2001). However, Bagley et al. (2016) did find a significantly greater increase in $\dot{V}O_{2max}$ following 12 weeks of sprint interval training (SIT) for females compared to males although the female participants had particularly low starting $\dot{V}O_{2max}$ (33 vs 42 $ml \cdot kg^{-1} \cdot min^{-1}$) therefore greater increases would be expected. When training status is matched, females and males show a similar lactate production response to submaximal exercise (Davis et al., 2000), but females will accumulate lower absolute concentrations of lactate than males following short duration high intensity exercise (Brooks et al., 1990; Gratas-Delmarche et al., 1994; Jacobs et al., 1983). One explanation for this is the relationship of lactate production and the percentage of fast twitch muscle fibres (Gollnick et al., 1973). Phosphofructokinase (PFK) levels are also lower in females (Komi and Karlsson, 1978) which may result in blunted ability to produce energy through anaerobic glycolysis. This physiological difference may account for some of the

significant performance differences in terms of total work done and peak power evident between females and males during maximal exercise (Brooks et al., 1990; Gratas-Delmarche et al., 1994). When the ratio of lactate to power output is matched in terms of lean body mass, females and males are not different statistically (Gratas-Delmarche et al., 1994). This indicates that higher power output in males is not associated with the higher observed blood lactate concentration ([$La^-$]b) compared to females (Brooks et al., 1990). These differences are more likely related to training status than sex, with [$La^-$]b significantly higher in sprint trained females than untrained males following 400m running (Ohkuwa et al., 1988) suggesting sprint training enhances the potential for anaerobic glycolysis.

Eleven participants were recruited to provide similar sample sizes as previously published comparable studies (Jones, Hamilton and Cooper, 2015; Lo et al., 2011; Nybo et al., 2010; O'Donovan et al., 2005; Ziemann et al., 2011). Participants acted as their own controls allowing fewer total participants and helping minimise potential confounders (Heckteden et al., 2018; Thiese, 2014). Previous research has shown that significant performance increases in peak oxygen uptake ($\dot{V}O_{2peak}$) following three weeks of SIT, were not further enhanced after an additional three (Burgomaster et al., 2008) or six (Yamagishi and Babraj, 2017) weeks of training. This suggests that a three-week intervention is sufficient to assess effectiveness of SIT and changes in aerobic capacity. An additional benefit of reducing the training period is the reduced likelihood of participant dropout (Thiese, 2014).

## 3.2 Experimental procedures

### 3.2.1 Study 1

Lactate absorption

Blood lactate concentration was measured via fingertip blood samples (Lactate Pro 2, Arkray Inc., Kyoto, Japan). The Lactate Pro 2 portable analyser has been shown to be reliable and is considered suitable for use in sports science research (Badari et al., 2009; Pyne et al., 2000). It provides accurate measurement of blood lactate in only 15s, and readings from the Lactate Pro 2

correlate well (r = 0.913) with a more sophisticated laboratory blood gas analyser (Tanner, Fuller and Ross, 2010). Blood glucose was measured using a FreeStyle Lite portable analyser (Abbott Diabetes Care Inc., USA). This analyser has been shown to be accurate and reliable when compared to laboratory equipment (Freckman et al., 2012; Tack et al., 2012) and Baumstark et al. (2012) concluded that it was one of the more consistent systems between batches of test strips.

Previous research (Morris et al., 2011) used a tailored approach where each participant received 120mg·kg body weight$^{-1}$ but this is less practical when working in an applied sports setting. Therefore, a standardised solution was used containing 1% weight by volume calcium lactate. This was a smaller dose than Morris and colleagues used but oral consumption of lactate carries a risk of gastrointestinal distress (Swensen et al., 1994), so this was deemed sufficient if blood lactate concentration [La$^{-}$]b could be seen to rise significantly following ingestion. Like Morris et al. (2011), the entire dose had to be consumed within one minute to ensure accurate post consumption lactate readings.

Bath University rugby shuttle test

The Bath University rugby shuttle test (BURST) is an exercise protocol designed by Roberts et al. (2010a) to simulate the various physiological demands of rugby union match play. Based on previously conducted time motion analysis of rugby union, it aims to provide equivalent volumes of the varying exercise intensities, from standing still to maximal sprinting, along with the additional static exertions found in rugby union such as scrums, rucks, and mauls.

Roberts et al. (2008) reported that, for English premiership rugby matches, players covered 5581m in 80 minutes and Lacome et al. (2014) recorded mean distance to be greater (~7500m) during international level matches. Total distance covered during the 90-minute BURST protocol is 7078m which makes it applicable to what could be expected at elite level rugby union.

Approximately 80% and 20% of the BURST is spent in low and high intensity work respectively. While this is a lower percentage of low intensity work than has been seen in other rugby union studies (84% - Deutsch et al., 1998, 85% -

Duthie, Pyne and Hooper, 2012; and 88% - Deutsch, Kearney and Rehrer, 2007), it has been noted that low intensity work in invasion sports can range from ~70% (Bangsbo, Mohr and Krustrup, 2006) to ~97% (Gabbett, 2010a). With 9.9% of total time spent in static exertions such as rucking and mauling, similar to what is seen during match play (10% - Duthie, 2006; 11% - Duthie, Pyne and Hooper, 2005; 9.5% - Roberts et al., 2008), the additional high intensity work comes from high-speed running and sprinting.

Blood lactate is often used as an indicator of work intensity during training and match play (Beneke, Leithäuser, and Ochentel, 2011; Billat, 1996) with levels in international rugby union matches reaching concentrations of up to 9.8mmol·l$^{-1}$ (McLean, 1992). Mean blood lactate measured during the BURST was found to be 4.5 mmol·l$^{-1}$ (Roberts et al., 2012) which is less than the 6.6mmol·l$^{-1}$ mean value reported by Deutsch et al. (1998). It should be noted however that the sampling conducted by Deutsch occurred only once or twice per half during natural major breaks in play. These breaks tend to occur after phases of intense play such as following scoring and therefore may lead to more greatly elevated [La$^{-}$]b.

Mean heart rate (HR) during match play has been reported to be >85% of predicted maximum for ≥72% of the match in elite level Under-19 players, with back row forwards spending ~20% of the match >95% heart rate maximum ($HR_{max}$) (Deutsch et al., 1998). Doutreloux et al. (2002) measured a mean heart rate of 180 b·min$^{-1}$ in forwards, while Duthie, Pyne and Hooper (2003) reported mean match heart rate to be to 161 b·min$^{-1}$ for backs. Roberts et al. (2010a) recorded mean heart rate over the two BURST trials as 159 b·min$^{-1}$ which is slightly lower than that expected for forwards during match play.

Together, this information confirms that the BURST protocol is an acceptably valid way to simulate the physiological demands of rugby union match play and, with a coefficient of variation (CV) of only 1.1% and 1.3% for time taken to complete baseline performance tests and performance tests during BURST, it would appear to be reliable between trials.

Maintenance of performance was assessed during the performance test segments of the BURST via a 15m sprint test using an electronic timing system

(Brower Speed Trap II; Brower, Utah, USA). This distance is comparable to the mean sprint distance performed by forwards during match play (Deutsch et al., 1998; Eaton and George, 2006). These 15m maximal sprints occurred before the test commenced to ascertain baseline performance, then again at the end of each of the 16 exercise blocks of simulated match play. Roberts et al. (2010a) found a CV of 0.9% between equivalent sprints when the BURST was performed on different days showing good reproducibility in terms of this performance measure. These data were analysed using sprint time from each performance test, and total time spent sprinting was calculated for each block, half, and for the full test. Performance decrement was measured through percentage increase in time spent sprinting between blocks one and two, blocks three and four, and between first and second halves.

### 3.2.2 Study 2

Body composition

Participant body mass and body fat percentage were measured using bioelectrical impedance analysis (BIA) (SC-330ST Tanita Body Composition Analyser, Tanita Europe BV, Amsterdam, Netherlands), and height was measured using a stadiometer (SECA 213 stadiometer, United Kingdom). BIA shows excellent reliability for repeat measures (<0.2% variation) although it does tend to underestimate body fat by approximately 2% compared to air displacement plethysmography and dual-energy X-ray absorptiometry (de Castro, de Lima and Silva, 2018; Hurst et al., 2015). While it is important to be aware of this underestimation, the high level of reproducibility of body fat estimates means BIA is an accurate method to track changes in body composition over time (de Castro, de Lima and Silva, 2018).

Aerobic function during incremental tests

Maximum oxygen uptake ($\dot{V}O_{2max}$) is the greatest rate at which oxygen can be taken up and utilised by the body during intense exercise, and denotes the upper limit of aerobic function (Astorino et al., 2000; Bassett and Howley, 2000; Day et al., 2003; Gim and Choi, 2016; Yamamoto et al., 2014). The concept of

$\dot{V}O_{2max}$ was originally proposed by Hill and colleagues (1923, 1924) where they observed a $\dot{V}O_2$ plateau. They noticed that during incremental exercise to exhaustion, there is a point where oxygen uptake does not increase further despite increasing exercise intensity, and this criterion has been central to many protocols since. Protocols to test $\dot{V}O_{2max}$ were time consuming, with discontinuous tests such as the one used by Taylor, Buskirk and Henschel (1955) requiring the participant to attend up to five separate sessions to establish an exercise intensity sufficient to establish $\dot{V}O_{2max}$. These time-consuming tests have now largely been replaced by maximal effort ramp protocol tests which allow several parameters of aerobic fitness such as $\dot{V}O_2$ and lactate threshold to be measured in a single, time efficient test (Day et al., 2013; Rossiter, Kowalchuk and Whip, 2006). However, approximately half of these type of tests show no plateau (Howley, Basset and Welch, 1995), and the peak $\dot{V}O_2$ ($\dot{V}O_{2peak}$) observed is taken as the upper limit. It has been questioned whether the $\dot{V}O_{2peak}$ can been deemed to be at the upper aerobic capacity of the participant, even when they are apparently at maximum effort, if no plateau is observed (Rossiter, Kowalchuk and Whip, 2006; Wagner, 2000). Evidence suggests, however, that $\dot{V}O_{2peak}$ is most likely an accurate and valid representation of $\dot{V}O_{2max}$ (Day et al., 2003; Duncan, Howley and Johnson, 1997; Rossiter, Kowalchuk and Whip, 2006; Sousa et al., 2010), and repeating the test until a plateau is observed is unnecessary (Loftin et al., 2004). In addition to establishing a plateau, it can be deemed $\dot{V}O_{2max}$ has been reached if HR is within 10b·min$^{-1}$ of age adjusted maximum, respiratory exchange ratio (RER) is ≥ 1.15, or [La$^-$]b is > 8 mol·l$^{-1}$ (Astorino et al., 2000; Duncan, Howley and Johnson, 1997).

$\dot{V}O_{2peak}$ can be established by using the following sampling rates; breath by breath analysis where the single highest value is recorded (Sousa et al., 2010), averaging over a very short period of 5 - 20s (Day et al., 2003; Rossiter, Kowalchuk and Whip, 2006), and longer sampling intervals of 30 - 60s (Astorino et al., 2000; Yamamoto et al., 2014). Whilst these longer sampling periods help alleviate noise seen in breath-by-breath measurements (Day et al., 2003), they may miss the small, rapid changes in $\dot{V}O_2$ near exercise tolerance (Rossiter,

Kowalchuk and Whip, 2006). To achieve a balance between these two issues, Rossiter, Kowalchuk and Whip (2006) advise using an average value from the last 15s of the incremental tests although Dwyer (2004) suggest 20s may be more reliable. However, Astorino et al. (2000) show that 15s sampling provides the most accurate estimate of true $\dot{V}O_{2max}$ as this sampling time was most commonly associated with a $\dot{V}O_2$ plateau. The ramp protocol used in this study will therefore test $\dot{V}O_{2peak}$ using the highest 15s average from the breath-by-breath gas analysis.

$\dot{V}O_{2peak}$ was measured for the dual purpose of monitoring the effect of the training intervention on both cardiorespiratory function, and also repeated sprint ability (RSA). This is because aerobic energy metabolism provides a significant contribution during repeated sprint exercise (Bogdanis et al., 1996; McKenna et al., 1997; Parolin et al., 1999; Trump et al., 1996). The total time of the exhaustive ramp test was also recorded to the nearest second and this was deemed the participant's time to exhaustion (TTE) which is a valid measure of fatigue resistance (Jakeman, Adamson and Babraj, 2012; Stevens and Dascombe, 2015).

Wingate anaerobic test

Participants were required to perform a single 30s Wingate anaerobic test (WAnT) which is considered the gold standard for assessing lower body anaerobic power in sports science laboratories (Herbert et al., 2015). The WAnT was performed on a cycle ergometer (Monark Ergomedic 874E, Varberg, Sweden) against a resistance of 0.075 kg·kg body mass$^{-1}$. This resistance was selected as it is described by Ayalon et al. (1974, Cited in: Vandewalle, Pérès, and Monod, 1987) to be the optimum resistance, and it is also used as the resistance in many investigations employing Wingate type sprints (Burgomaster et al., 2005, 2006, 2008; Hazell et al., 2010; Jakeman, Adamson, Babraj, 2012; Kavaliauskas, Aspe, and Babraj, 2015; Lloyd Jones, Morris, and Jakeman, 2017; Yamagishi and Babraj, 2017; Zagatto, Beck and Gobatto, 2009).

The purpose of this test was to determine participants' maximal anaerobic power and anaerobic capacity. Anaerobic power is reported as peak power output (PP) and is achieved within the first 5 - 6s of the sprint (Attia et al., 2014; Herbert et al., 2015) with adenosine triphosphate (ATP) supply coming predominantly from phosphocreatine (PCr) for the first 15s (Serresse et al., 1988). Anaerobic capacity is denoted by the average power output (AP) over the 30s with energy supply being mainly anaerobic glycolytic from 15 - 30s (Serresse et al., 1988). Investigation into the overall energy contribution during a 30s WAnT originally reported relative contributions of 23%, 49% and 28% for phosphogenic, glycolytic, and oxidative pathways respectively (Serresse et al., 1988). More recently, Beneke et al. (2002) found contribution to be 31%, and 50%, and 19% for the same pathways, suggesting anaerobic glycolytic contribution was higher than previously thought.

Short-duration cycle ergometer sprints are a valid and reliable measure when assessing invasion sport athletes (Attia et al., 2014), and they can be used reliably by non-cycling sport athletes to effectively monitor change in performance over time (Wehbe et al., 2015b). Perez-Gomez et al. (2008) showed that 30m sprint performance was significantly corelated with WAnT peak power ($r = -0.36$, $P < 0.05$) and average power ($r = -0.34$, $P < 0.05$) in males, and peak power in females ($r = -0.66$, $P < 0.05$) suggesting it is a valid test for invasion sport athletes where sprints typically range between 10 to 20m (Jennings et al., 2010).

Vertical jump tests

A vertical jump (VJ) is commonly used to evaluate athletic performance in invasion sports, and to monitor changes in explosive power elicited by training programmes (Chamari et al., 2008; Driss and Vandewalle, 2013; Markovic et al., 2004; Peterson, Alvar and Rhea, 2006; Sattler et al., 2012; Vandewalle, Pérès and Monod, 1987). VJ is an important element in performance for many sports (Aragón-Vargs, 2000), and jump performance is related to level of performance in elite soccer (Arnason et al., 2004), rugby union (Cunningham et al., 2018), and field hockey (Reilly and Borrie, 1992). Contractile rate of force

development is the ability to rapidly generate muscular force (Aagaard et al., 2002) and is the primary contributor to counter movement jump (CMJ) performance (McLellan et al., 2011). High performance levels in invasion sports are reliant on rapid force development because time available to apply that force is generally very short (~50 - 250ms) (Anderson and Aagaard, 2006; Newton and Kraemer, 1994). Wisløff et al. (2004) investigated the relationship between VJ and sprint performance and found a strong correlation between them over 10m and 30m ($r = 0.72$, $p < 0.001$ and $r = 0.60$, $p < 0.01$).
CMJ has been shown to be a valid measure of explosive power in lower limbs, with high reliability (Cronbach's $\alpha = 0.97$ and 0.98) when assessed using a jump mat (Aragón-Vargs, 2000; Hopkins, Schabort and Hawley, 2001; Markovic et al., 2004). In this study jump height was recorded using an electronic jump testing system (JumpMat, FSL Electronics Ltd, Cookston, United Kingdom). The gold standard of assessing jump height is considered to be a 3-camera motion analysis system (Aragón-Vargas, 2000), but electronic contact mat systems show strong correlations (Pearson's $r = 0.97$), are valid (Castagna et al., 2013; Leard et al., 2007), reliable (Nuzzo, Anning, and Scharfenberg, 2011; Pueo et al., 2018), and sensitive to change (Pueo et al., 2017).

Repeated Sprint Ability

The ability to repeatedly produce high-intensity, maximal effort sprints throughout match play is an important factor determining success in invasion sports (Bishop et al., 2011; Faiss, Girard and Millet, 2013: Gabbett, 2010b; Girard, Brocherie and Millet, 2015; Spencer et al., 2004). RSA has significant correlation to WAnT for fatigue index ($r = 0.63$, $p < 0.01$) and is a good sports movement specific test for fatigue resistance (Zagatto, Beck and Gobatto, 2009). Repeated sprint tests are used as an accurate indicator of high-intensity work performed during match play (Barbero-Álvarez, Pedro and Nakamura, 2013; Rampinini et al., 2007), and have been shown to be a reliable and valid method in soccer players (Barbero-Álvarez, Pedro and Nakamura, 2013; Gabbett, 2010b; Impellizzeri et al., 2008; Wragg, Maxwell and Doust, 2000). A 6 x 20m RSA test was used as per Gabbett (2010b). The test involved 20m

maximal sprints repeated on a 15s cycle with 10m deceleration and 10m active recovery between sprints. Total sprint time, average sprint time and percentage decrement in performance were reported as per other research on RSA in youth soccer players (Bravo et al., 2007; Eniseler et al., 2017; Rampinini et al., 2007). Percentage decrement in RSA performance ($RSA_{dec}$) was calculated using the following formula:

$$RSA_{dec} = 100 - \left(100 \cdot \left(\frac{RSA_{mean}}{RSA_{best}}\right)\right)$$

where $RSA_{mean}$ is the average sprint time, and $RSA_{best}$ is the shortest sprint time. However, Gabbett (2010b) and Spencer et al. (2006) have demonstrated that measures of performance decrement during RSA tests can be unreliable and should be used with discretion as large changes are required to detect meaningful improvement. Both studies reported that total sprint time was more reliable and should be the main indicator of changes in RSA.

Sprint speed

Due to the range of distances covered at maximal effort during match play, both acceleration and maximal speed are important elements of soccer performance (Burgess and Gabbett, 2013; Little and Williams, 2005). Sprint speed was determined by a 20m sprint which has been shown to be the average distance covered per sprint during match play (Burgess, Naughton and Norton, 2006). A 10m split was used to separate acceleration (0 - 10m) and maximum speed (10 - 20m) (Burgess and Gabbett, 2013). There is clear validity of a 20m sprint test to measure running acceleration and speed, and Duthie et al. (2006a) demonstrated good levels of reliability between sprints in invasion sport athletes even when starting technique varied.

Blood lactate

Blood lactate concentration was measured via the same fingertip blood sampling technique as study 1. Blood lactate kinetics can be calculated using bi-exponential functions such as the ones used by Beneke et al. (2005) and

Messonnier et al. (2006) which determine the rate of change in [$La^-$]b over time. For this study, lactate kinetics were modelled using the three-pool model that has been previously used in adolescents (Beneke et al., 2005), and provided better goodness of fit ($r^2$ = 0.985 ± 0.017). The following bi-exponential function was used:

$$[La^-]b_t = \frac{A \cdot k_1}{k_2 - k_1} \cdot (e^{-k_1 \cdot t} - e^{-k_2 \cdot t}) + [La^-]b_0$$

where A is extravascular release of lactate from exercise metabolism, $k_1$ is the rate of lactate accumulation, $k_2$ is the rate of lactate clearance, t is time, and [$La^-$]$b_0$ is blood lactate concentration prior to the WAnT test. Maximum blood lactate concentration ([$La^-$]$b_{max}$), time to maximum blood lactate concentration (T[$La^-$]$b_{max}$), and the turn point (TP) where blood lactate concentration begins to decrease were described by mono-exponential functions:

$$[La^-]b_{max} = [La]b_0 + A \cdot \left(\frac{k_1}{k_2}\right)^{\frac{k_2}{k_2 - k_1}}$$

$$T[La^-]b_{max} = \frac{1}{k_1 - k_2} \cdot 1n\frac{k_1}{k_2}$$

$$TP = \frac{2}{k_1 - k_2} \cdot 1n\frac{k_1}{k_2} = 2T\Delta[La^-]b_{max}$$

### 3.2.3 Study 3

Wattbike Pro cycle ergometer

Participants performed the exhaustive incremental test on a Wattbike Pro cycle ergometer (Wattbike Ltd, Nottingham, United Kingdom). The Wattbike Pro provides pedalling resistance through both air and magnetically braked systems. Air braked resistance (level 1 - 10), and magnetically braked resistance (level 1 - 7) are automatically prescribed based on participant body mass. The Wattbike Pro is currently used in many elite sport training facilities

and research projects (Beard et al., 2019; Driller et al., 2013; Jones, Hamilton and Cooper, 2012). They are favoured by National Governing Bodies such as England and New Zealand Rugby, and professional sports teams competing in top national leagues such as Leicester City Football Club and Saracens (Rugby) Football Club (www.wattbike.com). The Victorian Institute of Sport in Australia also utilises these cycle ergometers to train Olympic athletes from several sports including field hockey (www.vis.org.au), making it a relevant choice for assessing performance markers in hockey. With this level of industry recognition, research conducted using this cycle ergometer may have greater practical impact with sports scientists and strength and conditioning coaches through being available in commercial fitness settings in addition to high performance sport and research facilities.

Several studies have confirmed the validity and reliability of this cycle ergometer as a means of testing power production related to sporting performance, and for tracking adaptations elicited through training (Driller et al., 2013, 2014; Herbert et al., 2015; Hopker et al., 2010; Wehbe et al., 2015b). The 30s WAnT performed on a Monark cycle ergometer has long been seen as the gold standard for assessing anaerobic power (Herbert et al., 2015). In their research, these authors found the Wattbike Pro 6s peak power test correlated significantly with both a traditional 30s WAnT ($r = 0.9$, $p < 0.001$) and a 6s modified version of the WAnT ($r = 0.95$, $p < 0.001$). They also found no difference in time to peak power over any test and concluded the 6s peak power test was a valid measure of peak power compared to the 30s WAnT.

Results from 30s sprint tests on the Wattbike Pro are highly reproducible in trained cyclists for peak power output, mean power output, and blood lactate concentrations with correlation coefficients of 0.97 (90%CI 0.94 - 0.99), 0.99 (90%CI 0.97 - 1.00), and 0.94 (90%CI 0.87 - 0.98) respectively (Driller et al., 2013). Wehbe et al. (2015b) also reported highly reliable results for peak power with professional invasion sport athletes. In three separate trials spaced seven days apart, athletes performed 2 x 6s peak power tests interspersed by one minute of passive recovery. Data showed mean CV, intra-class correlation coefficient and standard error (SE) for peak power was 3% (90%CI, 2.5 - 3.8),

0.96 (90%CI, 0.91 - 0.98) and 39W, which again supports the reliability of this cycle ergometer.

Additionally, Hopker and colleagues (2010) reported that the Wattbike Pro was acceptably accurate and reliable when compared to the highly validated SRM Powermeter during constant load cycling over a range of resistive forces with coefficients of variation of 2.6% (95%CI 1.8 - 5.12) and 1.1% (95%CI 0.7 - 2.0) respectively, in trained cyclists.

Assessing Power

Critical power (CP) represents the greatest production rate of ATP which can be sustained predominantly through oxidative phosphorylation before a progressively increasing contribution from anaerobic glycolysis leads to fatigue (Jones et al., 2010). This means that CP represents a key physiological boundary between heavy and severe intensity exercise and is closely related to maximal lactate steady state (MLSS) and a $\dot{V}O_2$ of 70 - 90% $\dot{V}O_{2max}$ (Jones and Vanhatalo, 2017; Vanhatalo, Doust and Burnley, 2008; Vanhatalo, Jones and Burnley, 2011). As exercise intensity increases beyond CP, an exponential increase in $[La^-]b$ is seen (Jones and Vanhatalo, 2017). CP can be calculated by the following formula, where TTE is the time to exhaustion, W' is the work capacity available above CP and P is the power output.

$$TTE = \frac{W'}{(P - CP)}$$

W' is finite but the rate it is depleted depends on how far in excess of CP exercise intensity is, but it is then restored if exercise intensity falls below CP (Jones and Vanhatalo, 2017). Theoretically, CP can be maintained indefinitely (Kendall et al., 2009) although, in practice, a work rate equal to CP cannot be sustained for more than approximately 30 minutes (Brickley, Doust and Williams, 2002). Morton and Billat (2004) were the first to modify the CP model for intermittent exercise such as invasion sports but since restoration of W' is nonlinear, this model was further refined by Skiba et al. (2015) to account for a variable rate of recovery.

Traditionally, CP was measured over four or more constant-power exercise to exhaustion tests performed on separate days, but this is a very time-consuming practice (Vanhatalo, Jones and Burnley, 2011). More recent evidence (Vanhatalo, Doust and Burnley, 2007, 2008) demonstrated that the constant power produced in the final 45s of a 3-minute, maximal effort test is equivalent to CP making this a valid and, due to its sensitivity to changes induced by training, reliable test to determine CP. The measures this 3-min test allows, provides an important prediction of exercise tolerance and serves as an indicator of effect of training intervention. With the intermittent nature of invasion sports such as soccer, rugby and hockey, CP is a relevant test as significant periods of play are spent in severe-intensity domain (Vanhatalo, Jones and Burnley, 2011). It is an additionally useful physiological test as it combines testing of power with $\dot{V}O_2$ and lactate response profiles (Jones et al., 2010).

Repeated Sprint Ability

Participants performed the repeated sprint ability test on a Wattbike Pro cycle ergometer. It has previously been established that repeated short duration maximal efforts using a cycle ergometer are a valid method to assess fatigue in female invasion sport athletes (McGawley and Bishop, 2006), and correlates to decreases in short sprint running performance during match play (Bishop et al., 2011). Participants performed 6 x 10s sprints with a work to rest ratio of 1:1 as this simulates the most intense periods experienced by athletes in this type of sport (McLean, 1992). The PP recorded during the first sprint was also used as a measure of maximal power output. Total work done was also calculated over the six sprints. This is considered the most reliable way of detecting worthwhile change in cycle-ergometer RSA performance for invasion sport athletes (Cushman, Bott and Highton, 2018).

Lactate Kinetics

Blood lactate kinetics were calculated using the same bi-exponential function as was used in study 2.

## 3.3 Training

Why cycle-based

Inclusion of a running-based element to invasion sport preseason training elicits a rightward shift in the blood lactate curve after five weeks and increases MLSS running speed (Best et al., 2013). However, increasing high-speed running through either SIT or SSG can significantly increase the risk of injury in athletes (Bowen et al., 2017; Jones, Hamilton and Cooper, 2015; Malone et al., 2016, 2018; Schreurs, Benjainse and Lemmink, 2017). Previous SIT interventions in athletes indicate a much-reduced risk of musculo-skeletal injury when using a cycle-based protocol (Willoughby et al., 2016). Therefore, a cycle-based approach may be more appropriate for improving performance and lactate kinetics in invasion sport athletes, although no previous studies have investigated lactate kinetics in adolescent soccer players or amateur female field hockey players in response to cycle-based SIT. An important element of training for running-based invasion sports is the transferability of performance indicators to the sport, in particular running performance. Cycle-based training has been shown to improve running based performance markers including repeated sprint ability (Hamlin et al., 2017), and exercise intensity in exhaustive running protocols (Castagna et al., 2009, 2010). In addition to this, and of particular importance to invasion sport athletes, cycle-based training reduces the injury risk associated with the high intensity running and acute joint loading associated with SSG (Bowen et al., 2016; Jones, Hamilton and Cooper, 2015; Schreurs, Benjainse and Lemmink, 2017). 74% of injuries in elite female field hockey players are non-contact with 14% due to overuse (Delfino Barboza et al., 2018). Kim, Cha and Park (2016) found that in elite female hockey players, the risk of lower limb injury increases with the volume of high intensity running performed and, in a study, investigating the effect of running based SIT, Willoughby and colleagues (2016) experienced a 21% participant attrition rate due to soft tissue injury. In an analysis of several SIT studies, Buchheit and Laursen (2013) concluded that, like SSG, running based SIT has a significant risk of injury associated with it, primarily through hamstring and lower limb joint injuries. Previous SIT interventions in athletes indicate a much-reduced risk of musculoskeletal injury when using a cycle-based protocol (Willoughby et al.,

2016). An important element of training for running-based invasion sports is the transferability of performance indicators to the sport, in particular, running performance. Cycle-based training has been shown to improve running-based performance markers including maximal speed (Thom, Kavaliauskas and Babraj, 2019), RSA (Hamlin et al., 2017), and exercise intensity in exhaustive running protocols (Jones, Hamilton and Cooper, 2015).

Study 2

Participants performed 6 x 10s maximal cycle sprints on Monark Ergomedic 874E cycle ergometer against a resistance of 0.075 kg·kg body mass$^{-1}$, separated by 80s passive recovery. Morton (1978) reported that 56% of activities during invasion sports lasted less than 10s, and 85% less than 15s, making 10s work bouts applicable. It has also been demonstrated that a short duration (6 - 10s) sprint interval training (SIT) protocol was effective at improving performance indices relevant to invasion sports such as $\dot{V}O_{2peak}$, TTE, and anaerobic power output (Gaitanos et al., 1993; Jakeman, Adamson and Babraj, 2012; Kavaliauskas, Aspe and Babraj, 2015). In a recent study by Yamagishi and Babraj (2017), it was shown that a 15s SIT protocol was just as effective as 30s at improving $\dot{V}O_{2peak}$, TTE, 10km cycling time trial (TT), and even more so at increasing anaerobic power output. Hazell et al. (2010) also found similar results for $\dot{V}O_{2max}$, 5km cycling TT, and power output when comparing 10s and 30s Wingate bouts interspersed with four minutes recovery. With SIT protocol bouts as short as six seconds producing similar results to longer duration work, it could be assumed that the adaptations are occurring due to the metabolic demands of the early phase of the sprints (Jakeman, Adamson and Babraj, 2012).

Previous research has shown that use of a SIT protocol utilising a 1:3 work to rest ratio elicits mainly aerobic adaptation whereas longer rests with 1:12 work to rest ratio develop anaerobic characteristics (Kavaliauskas, Aspe and Babraj, 2015). When work to rest ratios mean that recovery time is relatively short compared to the supramaximal bout (1:1 - 1:5), it has been shown that active recovery has a detrimental effect on repeated sprint performance, with greater decline in the bouts that follow (Buchheit et al., 2009; Dupont et al., 2007;

Spencer et al., 2006, 2008). TTE is also reduced compared to passive recovery for recovery intensities ranging from 20 – 50% $\dot{V}O_{2max}$ (Dupont, Blondel and Berthoin, 2003; Dupont et al., 2004, 2007; Spencer et al., 2006, 2008). The explanation for this decline offered by several authors (Buchheit et al., 2009; Dupont, Blondel and Berthoin, 2003; Dupont et al., 2004, 2007; Spencer et al., 2006, 2008) is lack of restoration of intramuscular PCr. With continued muscle activation during active recovery still requiring ATP, less is available to resynthesise PCr. Contrary to this, relatively long recovery periods, with work to rest ratios of 1:8 - 1:12, appear to enhance overall sprint performance (Bogdanis et al., 1996; Connolly Brennan and Lauzon, 2003; Spierer et al., 2004). Greater PCr resynthesis is highly correlated (r = 0.84) with maintenance of power output during a 10s sprint (Bogdanis et al., 1996), and this may become progressively more relevant as number of sprints increases (Tomlin and Wenger, 2001). However, during high intensity interval training (HIIT), Smilios et al. (2017) state that if the recovery period is too long, there is a decrease in HR and $\dot{V}O_2$ at the start of the next bout, meaning there may be a reduction in the duration spent exercising at sufficiently high intensity to elicit the desired physiological adaptations. Therefore, a 1:8 work to rest ratio which improves both aerobically (TTE) and anaerobically (PP) driven attributes (Kavaliauskas, Aspe and Babraj, 2015), with passive recovery (Buchheit et al., 2009; Dupont et al., 2007; Spencer et al., 2006, 2008), may be most beneficial to invasion sport athletes.

This protocol was performed twice per week with a minimum of 48 hours between training sessions to allow adaptation to occur as training on consecutive days results in sub-optimal performance change (Rodas et al., 2000).

Study 3

Participants performed 6 x 10s maximal cycle sprints on Wattbike Pro separated by 80s recovery utilising the same work to rest ratio as the previous study. Investigating the effect of SIT in elite level female field hockey players, Jones, Hamilton and Cooper (2015) set resistance at air brake level 3 and magnetic brake level 1 to determine power output. Participants were of similar

age and mass (20.6 ± 0.9 years; 65.0 ± 4.3kg) to this study (19.0 ± 1.0 years; 65.7 ± 10.5kg), but to provide a training overload (Hamlin et al., 2017) from test intensity, air brake resistance was increased to level 4 for all participants. Although this does not represent an equal relative intensity for all participants, a universal SIT protocol has been shown to be no less effective than a tailored approach based on characteristics such as ventilatory threshold (Astorino et al., 2018). In a study of amateur rugby union players, Hamlin et al. (2017) prescribed training resistance at air brake level 3 and magnetic brake level 3, increasing to air brake level 5 and magnetic brake level 3 to provide additional overload. With resistance prescribed automatically by the Wattbike software during power tests based on body mass, it would be expected these heavier (88.3 ± 14.1kg) male athletes would require a higher level of resistance to elicit physiological adaptations. Others (Wehbe et al., 2015a) have used much higher resistance for SIT (air brake level 10 and magnetic brake level 4) despite athletes having mean body mass of only 75.3 ± 7.8 kg. However, this additional resistance may have been due to the training status of the athletes who were Australian rules football players competing at an elite level. This protocol was performed twice per week over a three-week period with a minimum of 48 hours between training sessions to allow adaptation to occur as training on consecutive days results in sub-optimal performance change (Rodas et al., 2000).

This 1:8 work to rest ratio has been shown to develop both aerobic and anaerobic capacity (Kavaliauskas, Aspe and Babraj, 2015), and also replicates the 1:8 ratio seen during the repeated sprint phases in hockey matches (Spencer et al., 2004).

## 3.4 General statistical analysis

Statistical analysis was conducted using IBM® SPSS® Version 28.0 for Windows, while QtiPlot data analysis and scientific visualisation was used to graph the blood lactate data. Data cleaning removed values outwith two standard deviations from the mean and Little's MCAR test was then used to determine if missing data points, either before or after cleaning, were missing completely at random. Where Little's test is not statistically significant, meaning there is no pattern to the missing data, values were replaced using multiple imputation to allow repeated-measures analysis of variance (ANOVA). Assumptions of homogeneity of variance were tested using Mauchly's sphericity test and where this is violated, the Greenhouse-Geisser estimate value was used to adjust degrees of freedom, unless this estimate was > 0.75 in which case the less conservative Huynh-Feldt estimate would be used. To test differences between and within groups, repeated measures ANOVA was used. In study 1, this was a 1 x 3 (group x time) to determine effect of condition. In study 2 it was a 2 x 2 (group x time) to determine effect of group, time, and group by time interaction. In study 3 it was a 2 x 3 (condition x time) to determine effect of condition, time, and condition by time interaction. Partial eta squared ($\eta_P^2$), which indicates how much of the change in the dependent variable can be attributed to the independent variable, was calculated to show effect size for training or condition. Effect size was defined as follows; small = 0.01 - 0.05, medium = 0.06 - 0.13, and large ≥ 0.14 (Cohen, 1988). Add section describing confidence intervals to effect size estimates.

Pearson's correlations were used to assess the relationships between lactate kinetic parameters and performance measures. Statistical significance for group means difference was set a priori at $p \leq 0.05$.

# Chapter 4

# Study 1

## 4.1 Abstract

**Introduction:** In rugby union, sprint performance over the duration of match play is a critical element of success but there is a noticeable decline in this area as playing duration increases. Maintaining energy substrate availability is an important element of maintaining sprint performance and while many commercially available sports drinks exist, more recent evidence as show lactate is consumed preferentially to glucose.

**Methods:** Ten recreationally active participants were recruited for part one of the study to determine the rate at which orally consumed lactate solution appears in the blood. For part two of the study, seven amateur rugby union players underwent the BURST under three conditions. Sprint performance was measured through 17 x 15m maximal sprints over the course of the BURST.

**Results:** An increase in blood lactate concentration ($[La^-]b$) was evident within 10 minutes of ingestion and it was significantly ($p \leq 0.05$) elevated from 20 to 60 minutes post ingestion. There was no change in blood glucose concentration throughout the testing period. There was no significant ($p > 0.05$) difference in sprint time between conditions at any performance test, with total sprint time per block, per half, and across the full BURST protocol also showing no difference. There was no significant difference in decrement of sprint performance between the first and second ($p = 0.69$), or third and fourth ($p = 0.13$) exercise blocks, or between the first and second halves ($p = 0.59$). There was a significant difference in peak heart rate between condition in both the first (C = 179 ± 4, L = 172 ± 6, G = 179 ± 9; $p = 0.04$) and second (C= 178 ± 3 L = 170 ± 7, G = 176 ± 6; $p = 0.03$) halves of simulated match play. There was no significant difference in respiratory rate between conditions in either the first (C = 44 ± 8, L = 38 ± 6, G = 47 ± 11; $p = 0.16$) or second (C = 42 ± 17, L = 36 ± 6, G = 40 ± 19; $p = 0.68$) halves.

**Conclusions:** Supplementation of 1% Wt/vol calcium lactate solution did not enhance sprint speed or significantly reduce the drop in sprint performance seen throughout a match. There was a trend for the decrement in performance to be less in the lactate condition and therefore, the use of calcium lactate could be recommended prior to rugby matches as an ergogenic aid to sustain sprint performance.

## 4.2 Introduction

### 4.2.1 Demands of the sport

Rugby union is an intermittent high intensity sport that involves two 40-minute periods with a half time break of no more than 10 minutes (Quarrie and Wilson, 2000). It is a sport which requires strength, power, speed, and endurance (Coutts, Reaburn and Abt, 2003; Duthie, 2006; Holway and Garavaglia, 2009). Average total distance covered by players during a match varies according to player ability. At an international level, players can cover as much as 7500m (Lacome et al., 2014), whereas in top-level domestic matches the average is 5198m for Super 14 (now Super 18) teams, and 5866m for teams in the English Premiership (Roberts et al., 2008). Approximately 20% of total distance is covered through high intensity running (running speed > $5m \cdot s^{-1}$) with the remainder covered through low to moderate intensity walking or jogging (Austin, Gabbett and Jenkins, 2011a; Schoeman and Coetzee, 2014). As with other invasion sports, players experience a decline in activity as play duration progresses (Austin, Gabbett and Jenkins, 2011b; Harley et al., 2010; MacLeod, Bussell and Sunderland, 2007). It has been reported that distance covered is approximately 15% less in the final 10 minutes compared to the first 10 minutes of play (Roberts et al., 2008), and sprinting (running speed >$6m \cdot s^{-1}$) occurs 10 - 27% less frequently in the second half of a match (Tee, Lambert and Coopoo, 2017).

With rugby union being a complex game with different playing positions serving differing roles, players require physical attributes which are both common to all positions, and those particular to their own specific position (James, Mellalieu and Jones, 2005). Forwards tend to cover ~9% less total distance than backs (Roberts et al., 2008) but are involved in ~8% more high intensity static activity such as rucking and scrummaging (Deusch et al., 2007; Duthie et al., 2006). Approximately 12 - 16% of activity for forwards is high intensity work consisting of static exertion such as scrummaging and rucking, and dynamic activities like tackling and ball carrying (Deutsch et al., 1998; Deutsch, Kearney and Rehrer, 2007; Duthie, 2006; Duthie, Pyne and Hooper, 2005; Roberts et al., 2008).

Throughout a game, players are required to demonstrate the ability to effectively utilise anaerobic energy systems due to the intermittent high intensity work (Coutts, Reaburn and Abt, 2003; Deutsch et al., 1998; Deutsch, Kearney and Rehrer, 2007; Duthie, Pyne and Hooper, 2003). Static exertion accounts for approximately 10% of playing time totalling ~70% of the high intensity work performed by forwards (Duthie, 2006). Mean static exertion duration for forwards is 5.2s so is largely dependent on the phosphocreatine (PCr) and anaerobic glycolytic systems (Duthie, Pyne and Hooper, 2003; Roberts et al., 2008). When periods of sustained play do not allow PCr stores to replenish sufficiently, there is an increase in anaerobic glycolysis which results in a greater net release of lactate (Deutsch, Kearney and Rehrer, 2007). During match play, mean $[La^-]b$ ranges from 4.8 to 7.2 $mmol \cdot l^{-1}$ but is not significantly different between playing positions (Deutsch et al., 1998; McLean, 1992). Players also perform ~50 bouts of high intensity running (running speed > 5 $m \cdot s^{-1}$), approximately 20 of which are sprints (running speed > 6.7 $m \cdot s^{-1}$) (Roberts et al., 2008), and mean distance covered per sprint is 10 - 20m (Deutsch et al., 1998; Deutsch, Kearney and Rehrer, 2007; Duthie et al., 2006; Duthie, Pyne and Hooper, 2003; Vaz et al., 2014). The remaining 84 - 88% of match play consists of either complete rest or, as competitors move between plays, low intensity dynamic activity. However, mean match intensity is ~80% maximal oxygen uptake ($\dot{V}O_{2max}$) (Coutts, Reaburn and Abt, 2003; Cunniffe et al., 2009), with back row forwards spending about 20% of the match above 95% maximum heart rate ($HR_{max}$) (Deutsch et al., 1998). Duthie, Pyne and Hooper (2003) estimate mean match heart rate (HR) to be 161 $b \cdot min^{-1}$ with 72% of the game played above 85% $HR_{max}$. Maximal lactate steady state (MLSS) is associated with a mean $[La^-]b$ of 4 $mmol \cdot l^{-1}$ (Billat et al., 2003), and an oxygen uptake ($\dot{V}O_2$) equivalent to 70 - 90% of $\dot{V}O_{2max}$ (Jones and Vanhatalo, 2017; Poole et al., 2016) suggesting that most players will be at, or above, MLSS intensity throughout matches. This indicates that lactate metabolism may be an important determinant of rugby performance.

### 4.2.2 Energy supply during invasion sports

Energy supply during invasion sports comes primarily in the form of stored muscle glycogen and blood glucose (Baker et al., 2015), and is an important aspect of maintaining performance (Azevedo et al., 2007). Matches can last upwards of 90 minutes and performance is seen to drop in the latter stages (Stolen et al., 2005) due to depletion of muscle glycogen leading to fatigue (Mohr, Krustrup and Bangsbo, 2005). It is therefore important to ensure an adequate supply of energy for athletes to maintain performance throughout a match.

### 4.2.3 Lactate supplementation

It is now understood that lactate is an important energy substrate being used to fuel not only working skeletal muscles during exercise (Hamann, Kelley and Gladden, 2001; Kelley et al., 2002), but also the highly oxidative cardiac muscle where virtually all lactate taken up by the heart is oxidised (Stanley, 1991, cited in Gladden, 2004). Evidence suggests, through combined lactate clamp and tracer studies, that the body will preferentially oxidise lactate over both glucose and fructose to provide fuel for working muscles (Azevedo et al., 2007). Potentially, this means that if a supply of lactate were made available to the working muscles, they could maintain higher levels of force production for longer before fatiguing.

There are relatively few studies (Table 2.1) investigating the use of lactate as an ergogenic aid, but findings indicate it may indeed be beneficial to physical performance particularly during high intensity activity. Fahey et al. (1991) found that supplementing 7% poly-lactate solution (80% poly-lactate, 20% sodium lactate) during prolonged moderate intensity exercise (180mins at 50% $\dot{V}O_{2max}$) produced no difference in perceived exertion, blood lactate concentration $[La^-]b$, heart rate, or oxygen consumption compared to glucose polymer and water. They did however conclude that lactate may help maintain performance during prolonged exercise through maintenance of blood glucose and, if exercise intensity were to increase, provide a useful buffering effect against decreasing pH levels.

Azevedo et al. (2007) found that the inclusion of a lactate polymer in a 250ml drink two minutes before, and at 45-minutes during steady state, moderate intensity cycling (90mins at 62% $\dot{V}O_{2peak}$), which was then followed by high intensity exercise to exhaustion (86% $\dot{V}O_{2peak}$) resulted in greater performance during the high intensity exercise with 25% longer time to exhaustion (TTE) compared to a glucose-based sports drink. They attributed this to the supplemented lactate being used more rapidly and more extensively than the carbohydrates to fuel the working muscles as exercise intensity increased. Matson and Tran (1993) had previously established a link between increased levels of blood acidity from high intensity exercise, and increased performance following supplementation of bicarbonate and lactate, to act as a buffering agent. They concluded that for an ergogenic effect to be noticeable from lactate supplementation, exercise had to be sufficiently intense to create an accrual of lactate and hydrogen ions ($H^+$). In fact, the greater the degree of metabolic acidosis generated by the exercise, the greater the ergogenic effect. This hypothesis that for lactate to be a useful supplement, exercise must be of a sufficiently high intensity to create an increase in $[La^-]b$ is also apparent in subsequent research. Cairns (2006) concluded that elevated lactate levels may be beneficial, and even vital, for performance during high intensity activity lasting 1 - 10 minutes. Morris et al. (2011) also found a significantly greater performance (17%) in single bouts of high intensity cycling to exhaustion following supplementation of calcium lactate compared to a placebo or no treatment. They also reported significantly elevated levels of blood bicarbonate supporting Matson and Tran's conclusions that lactate may have a buffering effect. Similarly, during a single bout of high intensity treadmill running, van Montfoort et al. (2004) reported a performance increase after consumption of sodium lactate although this was only 1.7%, and Northgraves et al. (2013) also found a non-significant improvement in 40km cycling time trial (TT) performance when supplementing lactate compared to sodium bicarbonate or a placebo. Conversely, one study consisting of cycling to exhaustion followed by a Wingate test for the final 30s, Bryner et al. (1998) concluded there to be no benefit in supplementing either lactate or glucose on peak power output. However, there are two noticeable limitation of this study. Firstly, during the steady-state,

exhaustive exercise portion, intensity selected was intended to produce a respiratory exchange ratio (RER) of <1.0 equating to an exercise intensity of only ~70 - 75% $\dot{V}O_{2max}$. This RER value indicates fat utilisation and suggests the intensity was not sufficiently high to require additional fuel from CHO based sources meaning muscle glycogen may have been sufficiently preserved for utilisation during the Wingate. Exercise intensity was also well below the ≥85% $\dot{V}O_{2max}$ required to elicit an exponential increase in the appearance of blood lactate through glycolysis (Brooks et al., 1991; Donovan and Brooks, 1983; Gladden, 2000; Gollnick, Bayly and Hodgson, 1986; Stanley et al., 1986) and stimulate an ergogenic effect. Secondly, inter-participant variation was large with standard deviation from the mean power of ± 37% and 45% for lactate and glucose treatments respectively, therefore increasing the probability of a Type II error. Swensen et al. (1994), who also found no increased performance from lactate supplementation, prescribed a continuous exercise protocol equivalent to 70% $\dot{V}O_{2max}$ in highly trained cyclists which again may not have been of a sufficiently high intensity.

### 4.2.4 Aim and hypothesis

Given the indications that lactate supplementation may be useful for maintaining performance in high intensity sprint activities, this study will, in a rugby union specific context, investigate whether supplementation of a 1% calcium lactate solution maintains sprint performance throughout simulated match play more effectively than more traditionally consumed carbohydrate-based sports drinks or water. It was hypothesised that those participants in the non-treatment (flavoured water) control group (C) would experience a greater level of performance decrement through fatigue during the Bath University rugby shuttle test (BURST), than those in the lactate (L) and glucose (G) supplementation groups.

## 4.3 Methods

### 4.3.1 Study design

A randomised, single blind, repeated measures experimental design was utilised. Participants were randomly assigned one of six treatment patterns by drawing group numbers from a hat and blinding was achieved by masking flavour differences between treatments using a sugar-free orange squash to match the orange flavour of the Lucozade sport.

### 4.3.2 Participants

For Part 1 of the study, which sought to determine lactate absorption rates, 10 participants were recruited. All participants self-reported at least the American College of Sports Medicine and American Heart Association recommended 2.5 hours of moderate physical activity per week (Haskell et al., 2007), although they were not required to have any specific association with rugby union.
For Part 2 of the study, eight healthy and recreationally active male participants (age: 25 ± 5 years, weight: 84 ± 6kg, and height: 180 ± 4cm) were recruited with seven completing the study. The same activity level inclusion criteria as part 1 of the study was used but all participants were sub-elite rugby union players with at least three years playing experience, with current participation levels of up to three times per week training and playing. The study was approved by the institutional ethics committee and all testing was carried out in line with the Declaration of Helsinki (www.wma.net, 2013).

### 4.3.3 Procedures

Session 1: Lactate appearance and clearance
Participants arrived at the laboratory and following a 20-minute period of seated rest, baseline blood lactate and glucose concentrations were recorded. Blood lactate and glucose concentration was measured via fingertip blood samples (Lactate Pro 2, Arkray Inc., Kyoto, Japan; FreeStyle Lite (Abbott Diabetes Care Inc., USA). The skin was punctured using an Accu-check single use lancet

(Roche Diagnostics, UK) and pressure applied to the finger to draw the blood. The initial drop was discarded, and the second drop was taken for analysis. Participants were then asked to consume a calcium lactate solution (500ml containing 1% weight by volume calcium lactate) before blood lactate and glucose concentrations were re-tested every ten minutes for the next hour to trace rate of appearance and clearance from blood. Participants remained seated throughout the testing to prevent skeletal muscle activity affecting blood metabolite levels.

Sessions 2-5: BURST

Participants reported to an indoor facility to avoid any environmental variation between test days and groups. The protocol was an amended version of the BURST (Roberts et al., 2010a).

Participants were required to refrain from undertaking strenuous physical activity for a period of 48 hours before each testing day, maintain their normal diet, and to drink 4 x 500ml of water throughout the day before the trial to ensure euhydration. Participants were informed of these conditions and requirements in writing beforehand and again verbally during the study. Participants were connected to a bio-harness monitor (Xypher Technologies) to measure HR and respiratory rate (RR) throughout and began each session with the same 10-minute warm-up. This comprised three minutes of jogging round the perimeter of the gymnasium and dynamic stretching. Dynamic stretching involved six walking lunges per leg, six diagonal lunges per leg, ten squats and ten counter movement jumps, ten leg swings each leg, ten back slaps and 15s of fast arm circles. This was followed by one 315s period of the BURST minus the performance test. A 315s period involved the participants walking 20m, turning 180º and returning to the start point at cruising speed. They then turned to face the original direction and jogged a further 10m. At this point, participants performed either a simulated ruck comprising a 5m carry of a 20kg tackle bag in 3.5s, a simulated maul for which the two participants attempted to alternately either gain or retain possession of a rugby ball for 5s, or a two-person scrum consisting of 5s of static maximal exertion against the other participant. Participants then jogged backwards 10 m and repeated the cycle following a

stationary rest. A 315s period consisted of five exercise cycles with rucks in cycles 1 and 3, mauls in cycles 2 and 4, a scrum in cycle 5 and a performance test and 15m sprint (Figure 4.1). Participants were then given two minutes passive recovery before the baseline performance test. Session 2 served as a familiarisation session where no drink was consumed and was limited to Block 1 of the protocol.

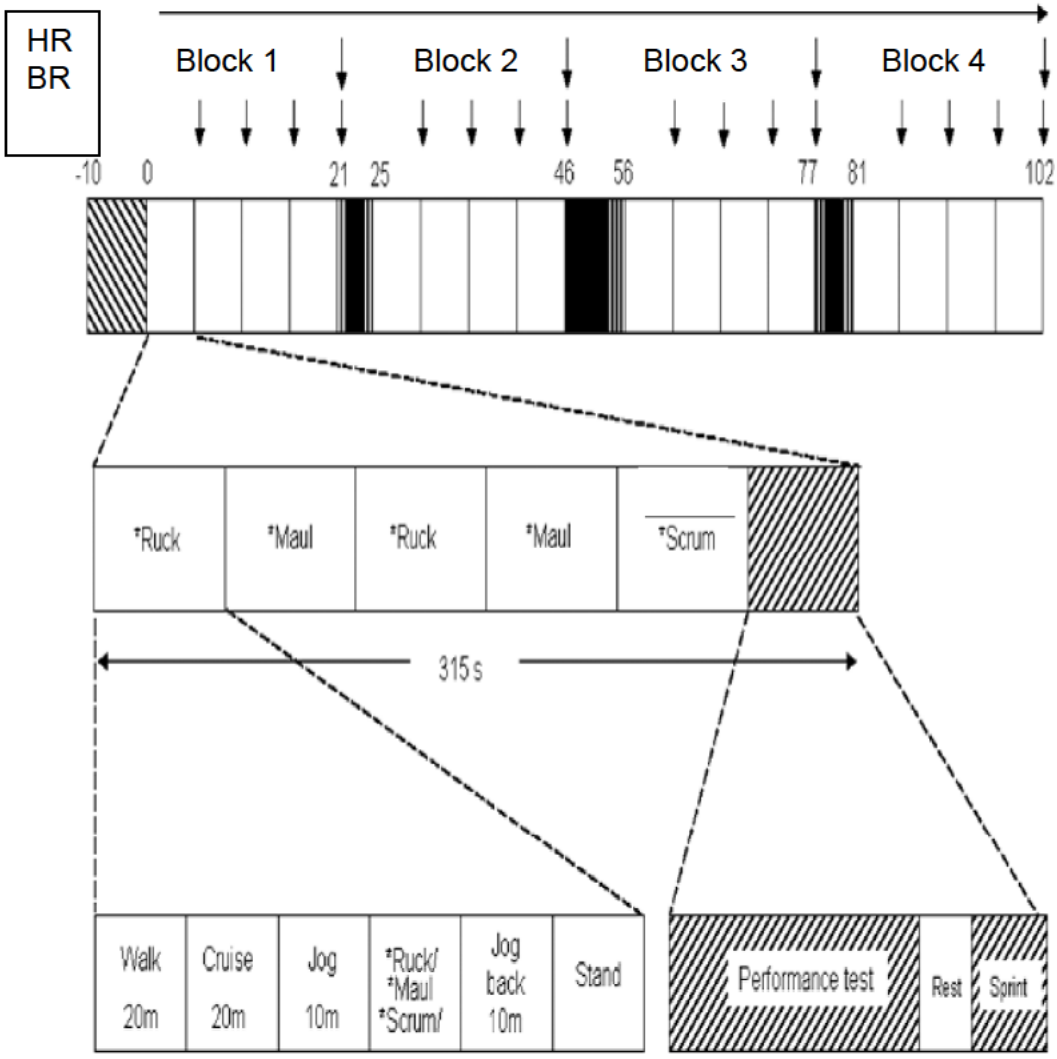


***Figure 4.1 A schematic representation of the exercise patterns in the Bath University rugby shuttle test (Roberts et al., 2010a).***

Performance Test

When instructed for the baseline test, and immediately following the fifth cycle thereafter, the participants walked to the start of the performance test (PT) (Figure 4.2). They began in a stationary position before picking up a tackle bag

and carrying it 9m, turning 180º, carrying it back, and placing it at the start point. The second participant would then repeat this procedure. Participants alternated between performing first and second to avoid discrepancies in the amount of overall rest they received. The first participant then picked up a rugby ball and carrying it in one hand, sprinted from marker 1 to marker 2. The participant then continued to sprint, making a sudden change of direction to sprint to the marker that they were directed to (3 or 4). Before passing marker 2, the participant did not know which direction change would be required. The participant then had 25s to move to the start of the 15m sprint and from a standing start, perform a single sprint between gates 1 and 2 (15m sprint time). Apart from the 25s of recovery, the PT and 15m sprint required maximum effort from the participant.

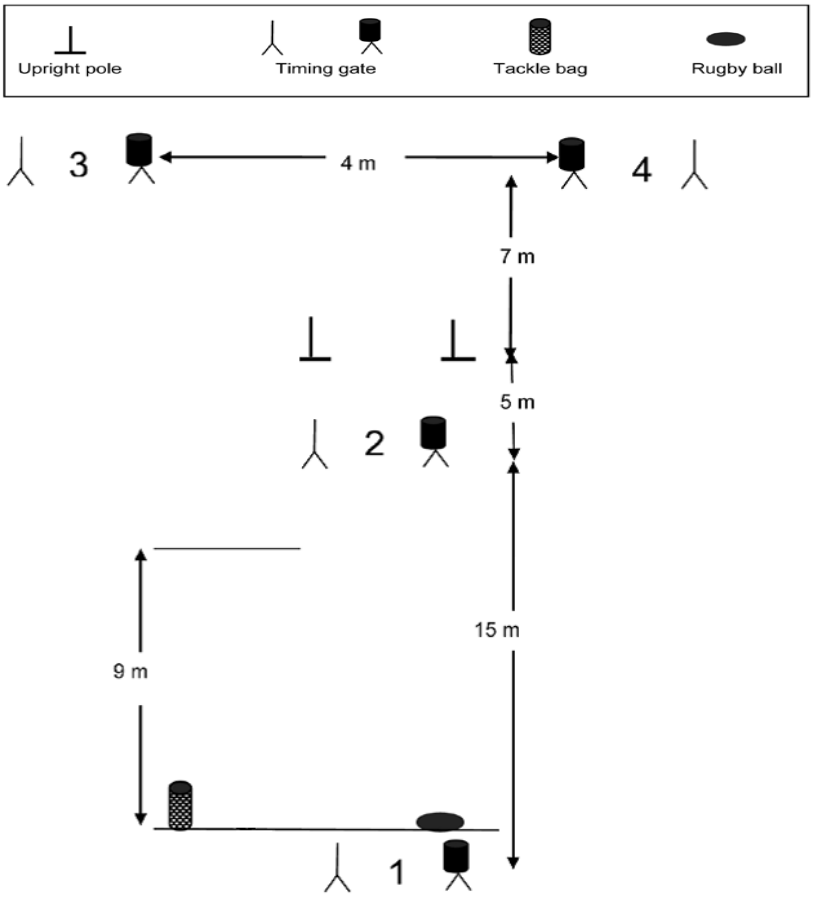


***Figure 4.2 A schematic representation of the Performance Test area (not to scale) (Roberts et al., 2010a).***

Following the baseline PT, the participants were required to consume a 500ml drink supplement which would either contain water, glucose (6% Wt/vol) or lactate (1% Wt/vol), depending on their supplement order group allocation. The BURST comprises 16 × 315s exercise periods grouped into 4 × 21min blocks (Figure 4.1). Blocks 1 and 3 were followed by a four-minute break, in which two minutes were spent standing followed by an equal period of walking. At the end of the second block there was a half time break as would be found in rugby union, which consisted of seven minutes and three minutes of sitting and walking respectively. Each participant consumed a second 500ml dosage of the pre-assigned drink at this point. Timing was maintained by computer-generated signals from a specifically recorded CD which ensured walking, jogging, and cruising were performed at mean speeds of 1.4, 3.0, and 4.2 $m \cdot s^{-1}$ respectively (Roberts et al., 2010a), and participants were verbally reminded of which task they were to perform at each stage.

Sprint performance was assessed through total sprint time for each exercise block, each half, and over the full BURST protocol. Decrement in performance was assessed using the percentage increase in total sprint time between blocks 1 and 2, blocks 3 and 4, and between the first and second half.

### 4.3.4 Data analysis

Statistical analysis of the data was performed using IBM® SPSS® Version 28.0 using a repeated measure analysis of variance (ANOVA). Statistical significance for group mean difference was set a priori at $p \leq 0.05$. Assumptions of homogeneity of variance were tested using Mauchly's sphericity test and where this was violated, the Greenhouse-Geisser value was to be used to adjust degrees of freedom unless this estimate was > 0.75 in which case the less conservative Huynh-Feldt estimate would be used. Partial eta squared ($\eta_P^2$) was calculated to determine effect size (ES) along with 95% confidence intervals (CI). Effect size was defined as small = 0.01 - 0.05, medium = 0.06 - 0.13, and large ≥ 0.14 (Cohen, 1988).

## 4.4 Results

### 4.4.1 Blood metabolites

Lactate

Mean [La$^-$]b at the pre-drink baseline test was 1.2 ± 0.5 mmol·l$^{-1}$. At the 10-minute post-drink test, mean [La$^-$]b had increased to 1.7 ± 0.7 mmol·l$^{-1}$ (Figure 4.3). At the 20-minute post-drink test, there was a significant increase in [La$^-$]b from baseline (Figure 4.3) with a mean value of 2.0 ± 0.7 mmol·l$^{-1}$. Peak blood lactate concentration occurred 30 minutes post consumption at a concentration of 2.3 ± 1.0 mmol·l$^{-1}$ (Figure 4.3). Blood lactate remained significantly higher than baseline at the 40-, 50-, and 60-minute post-drink tests with mean values of 2.0 ± 0.7 mmol·l$^{-1}$, 1.8 ± 0.6 mmol·l$^{-1}$, and 1.9 ± 0.4 mmol·l$^{-1}$ respectively (Figure 4.3).

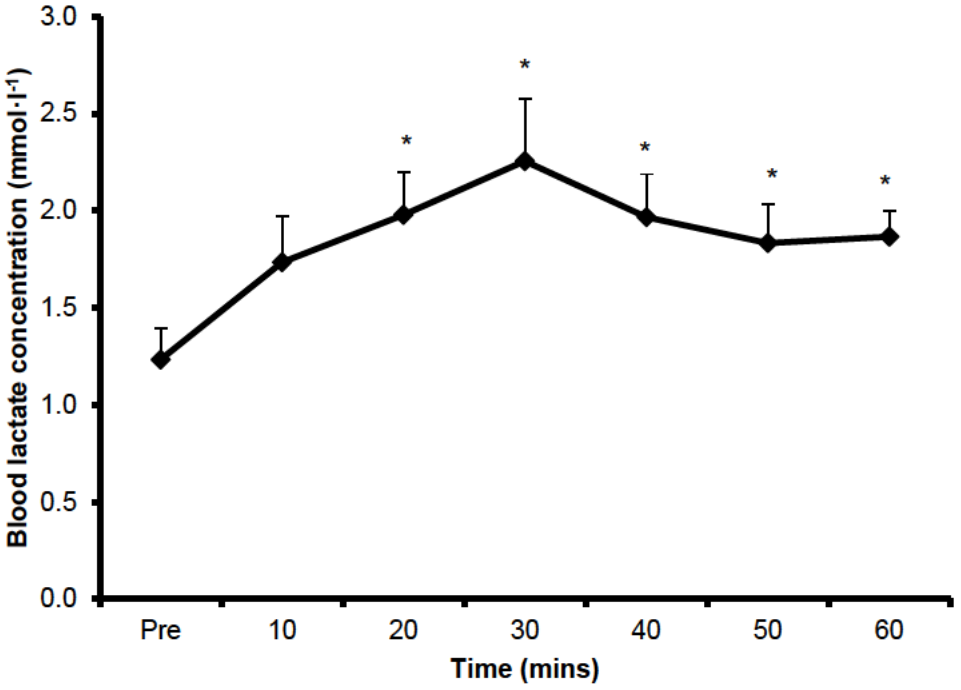


***Figure 4.3 Blood lactate concentration (mmol·l$^{-1}$) pre-exercise and at 10-minute intervals post consumption of 500ml 1% calcium lactate solution. * indicates significant difference from baseline ($p \leq 0.05$).***

Glucose

Mean blood glucose concentration was 4.8 ± 0.4 mmol·l$^{-1}$ at pre-consumption baseline test (Figure 4.4). At the 60-minute post-drink test, mean blood glucose level was 4.6 ± 0.4 mmol·l$^{-1}$ and there was no significant change at any other point throughout (Figure 4.4).

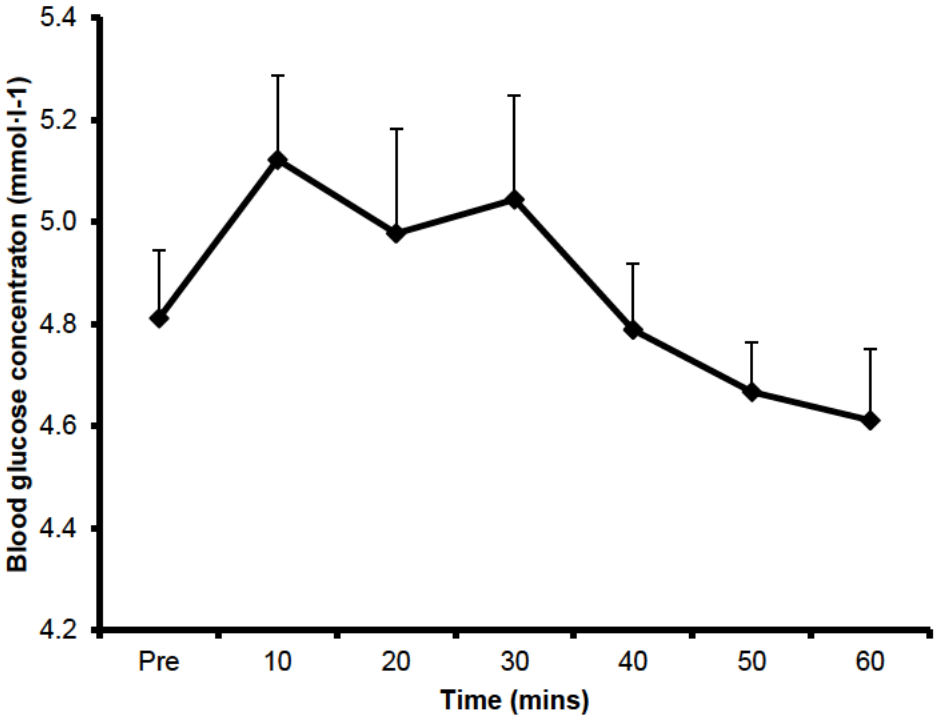


***Figure 4.4 Blood glucose concentration (mmol·l$^{-1}$) pre-exercise and at 10-minute intervals, post consumption of 500ml of 1% calcium lactate solution. * indicates significant difference from baseline ($p \leq 0.05$).***

### 4.4.2 Performance tests

***Table 4.1 15m sprint time (s) after each performance test.***

| Performance Test | Control | Lactate | Glucose | ANOVA p Value | ES ($\eta_P^2$) | 95% CI Range |
|---|---|---|---|---|---|---|
| Baseline | 2.57 ± 0.07 | 2.59 ± 0.12 | 2.64 ± 0.17 | 0.51 | 0.11 | 0 - 0.36 |
| 1 | 2.62 ± 0.12 | 2.66 ± 0.14 | 2.67 ± 0.15 | 0.64 | 0.07 | 0 - 0.31 |
| 2 | 2.66 ± 0.12 | 2.72 ± 0.12 | 2.69 ± 0.14 | 0.38 | 0.13 | 0 - 0.40 |
| 3 | 2.67 ± 0.12 | 2.73 ± 0.17 | 2.75 ± 0.16 | 0.53 | 0.10 | 0 - 0.36 |
| 4 | 2.67 ± 0.13 | 2.70 ± 0.14 | 2.78 ± 0.16 | 0.07 | 0.37 | 0 - 0.59 |
| 5 | 2.71 ± 0.12 | 2.71 ± 0.17 | 2.75 ± 0.15 | 0.77 | 0.04 | 0 - 0.26 |
| 6 | 2.70 ± 0.11 | 2.73 ± 0.10 | 2.74 ± 0.17 | 0.59 | 0.08 | 0 - 0.33 |
| 7 | 2.70 ± 0.09 | 2.73 ± 0.14 | 2.79 ± 0.17 | 0.32 | 0.17 | 0 - 0.43 |
| 8 | 2.70 ± 0.14 | 2.74 ± 0.10 | 2.81 ± 0.19 | 0.10 | 0.32 | 0 - 0.56 |
| 9 | 2.70 ± 0.12 | 2.76 ± 0.12 | 2.81 ± 0.18 | 0.23 | 0.22 | 0 - 0.48 |
| 10 | 2.73 ± 0.14 | 2.73 ± 0.13 | 2.80 ± 0.22 | 0.50 | 0.11 | 0 - 0.37 |
| 11 | 2.68 ± 0.12 | 2.72 ± 0.12 | 2.76 ± 0.19 | 0.22 | 0.22 | 0 - 0.48 |
| 12 | 2.69 ± 0.14 | 2.73 ± 0.12 | 2.76 ± 0.19 | 0.44 | 0.13 | 0 - 0.39 |
| 13 | 2.70 ± 0.12 | 2.72 ± 0.15 | 2.75 ± 0.21 | 0.58 | 0.06 | 0 - 0.29 |
| 14 | 2.74 ± 0.15 | 2.72 ± 0.11 | 2.75 ± 0.18 | 0.77 | 0.04 | 0 - 0.26 |
| 15 | 2.72 ± 0.13 | 2.71 ± 0.12 | 2.78 ± 0.21 | 0.40 | 0.12 | 0 - 0.38 |
| 16 | 2.71 ± 0.16 | 2.64 ± 0.08 | 2.72 ± 0.19 | 0.26 | 0.20 | 0 - 0.46 |

Values are mean ± SD.

Performance tests were not different between conditions at baseline tests nor at any of the subsequent 16 performance tests (Table 4.1). None of the groups demonstrated the slowest time in the final test and all final tests were quicker than the penultimate test. Total time spent sprinting was not different between conditions in each block, each half, or over the full BURST protocol (Table 4.2).

***Table 4.2 Total sprint time (s) for each block, each half, and full BURST protocol***

| | Time sprinting (s) | | | ANOVA p Value | ES ($\eta_P^2$) | 95% CI Range |
|---|---|---|---|---|---|---|
| | Control | Lactate | Glucose | | | |
| Block 1 | 10.61 ± 0.46 | 10.81 ± 0.45 | 10.90 ± 0.60 | 0.31 | 0.18 | 0 - 0.44 |
| Block 2 | 10.81 ± 0.44 | 10.90 ± 0.47 | 11.09 ± 0.67 | 0.34 | 0.16 | 0 - 0.43 |
| Block 3 | 10.81 ± 0.48 | 10.94 ± 0.47 | 11.13 ± 0.74 | 0.26 | 0.20 | 0 - 0.46 |
| Block 4 | 10.87 ± 0.54 | 10.79 ± 0.43 | 10.99 ± 0.76 | 0.44 | 0.11 | 0 - 0.37 |
| 1st half | 21.42 ± 0.90 | 21.71 ± 0.86 | 21.98 ± 1.24 | 0.29 | 0.19 | 0 - 0.45 |
| 2nd half | 21.68 ± 1.00 | 21.73 ± 0.88 | 22.12 ± 1.49 | 0.38 | 0.15 | 0 - 0.41 |
| Total | 43.10 ± 1.89 | 43.44 ± 1.71 | 44.10 ± 2.64 | 0.30 | 0.18 | 0 - 0.44 |

Values are mean ± SD.

There were no significant differences in performance decrement between conditions either within or between halves (Table 4.3). A trend was evident from the data showing drop in sprint performance was consistently greatest in the control group and least severe with lactate supplementation (Table 4.3).

**Table 4.3 Percentage decrement in sprint performance throughout BURST protocol comparing control, lactate and glucose conditions.**

| | Time sprinting (s) | | | ANOVA p Value | ES ($\eta_P^2$) | 95% CI Range |
|---|---|---|---|---|---|---|
| | Control | Lactate | Glucose | | | |
| Block 1 - 2 | 1.93 ± 0.79 | 0.90 ± 2.91 | 1.77 ± 2.35 | 0.69 | 0.06 | 0 - 0.29 |
| Block 3 -4 | 0.59 ± 1.81 | -1.38 ± 1.69 | -1.22 ± 1.15 | 0.13 | 0.26 | 0 - 0.53 |
| Half 1 - 2 | 1.18 ± 1.23 | 0.13 ± 1.46 | 0.61 ± 3.25 | 0.59 | 0.09 | 0 - 0.33 |

### 4.4.3 Heart rate

Block 1

There was no significant difference between the treatments for block 1 but a large effect size ($F(2,6) = 3.71$, $p = 0.09$, $\eta_P^2 = 0.55$ [95%CI: 0.00 to 0.74]). Mean peak 10s HR for C and G were 179 ± 5 b·min$^{-1}$ and 184 ± 7 b·min$^{-1}$ in block 1. The lowest in the block was 173 ± 6 b·min$^{-1}$ for L (Figure 4.5).

Block 2

There was no significant difference between the treatments for block 2 but a large effect size ($F(2,6) = 2.40$, $p = 0.17$, $\eta_P^2 = 0.45$ [95%CI: 0.00 to 0.67]). In C, mean peak 10s HR was highest in the block at 181 ± 6 b·min$^{-1}$ which was also the highest of the four blocks for this treatment (Figure 4.5). L dropped to 170 ± 5 b·min$^{-1}$ at the peak, which was the block lowest, and glucose dropped to 179 ± 8 b·min$^{-1}$ (Figure 4.5).

Block 3

There was no significant difference between the treatments for block 3 but a large effect size for treatment ($F(2,6) = 2.96$, $p = 0.13$, $\eta_P^2 = 0.50$ [95%CI: 0.00 to 0.70]). Mean peak 10s HR for block three for C was 179 ± 4 b·min$^{-1}$. The lowest peak in the block, and in all four blocks, was the lactate group which continued to drop from the previous block to 167 ± 6 b·min$^{-1}$. G peak 10s HR was 177 ± 8 b·min$^{-1}$ in this block and was the lowest peak HR for the glucose treatment (Figure 4.5).

Block 4

There was no significant difference between the treatments for block 4 but a large effect size ($F(2,6) = 1.30$, $p = 0.34$, $\eta_P^2 = 0.30$ [95%CI: 0.00 to 0.58]). In block four, peak 10s HR for C was 180 ± 7 b·min$^{-1}$. Block lowest was L at 172 ± 8 b·min$^{-1}$, while G 10s peak was 180 ± 5 b·min$^{-1}$ (Figure 4.5).

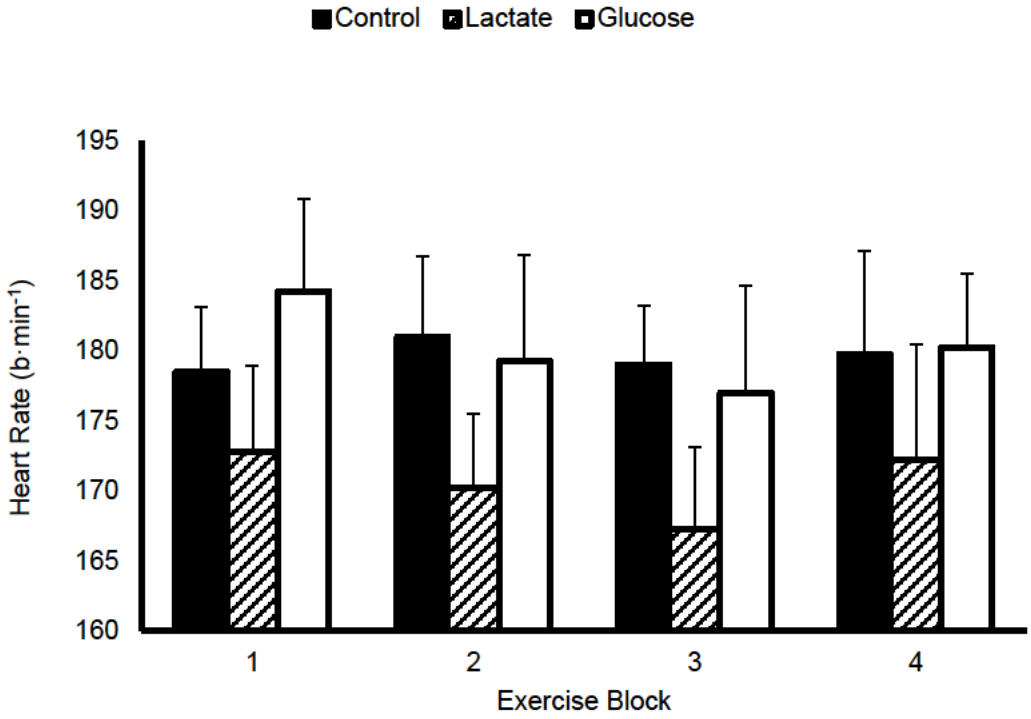


***Figure 4.5 Peak heart rate for each condition over the four exercise blocks.***

There was a significant main effect for condition on peak HR and a large effect size (Table 4.4). Lactate supplementation resulted in lower peak HR in both the first and second half.

***Table 4.4 Peak heart rate for each condition by half.***

| | Heart rate ($b \cdot min^{-1}$) | | | ANOVA p Value | Effect size ($\eta^2_P$) | 95% CI Range |
|---|---|---|---|---|---|---|
| | Control | Lactate | Glucose | | | |
| 1st half | 179 ± 4 | 172 ± 6 | 179 ± 9 | 0.04 | 0.43 | 0 - 0.64 |
| 2nd half | 178 ± 3 | 170 ± 7 | 176 ± 6 | 0.03 | 0.40 | 0 - 0.65 |

### 4.4.4 Respiratory rate

Block 1

There was no significant difference between treatments in block 1 for peak 10s RR ($F(2,6) = 1.50$, $p = 0.30$, $\eta_P^2 = 0.33$ [95%CI: 0.00 to 0.60]). Mean peak 10s RR for C was 45 ± 4 r·min$^{-1}$ in block 1. The lowest in the block was 39 ± 4 r·min$^{-1}$ for L, and G was highest at 50 ± 16 r·min$^{-1}$ which was also the highest block for the glucose treatment (Figure 4.6).

Block 2

Block 2 showed no significant difference in peak 10s RR between treatments ($F(2,6) = 1.06$, $p = 0.40$, $\eta_P^2 = 0.26$ [95%CI: 0.00 to 0.56]). In C, mean peak 10s RR was highest in the block at 46 ± 8 r·min$^{-1}$ which was also joint highest of the four blocks for this treatment (Figure 4.6). L peaked at 37 ± 7 r·min$^{-1}$ which was the block lowest, and G dropped to 40 ± 8 r·min$^{-1}$ (Figure 4.6).

Block 3

Differences in peak 10s RR in block three were not statistically significant but ES was large ($F(2,6) = 2.45$, $p = 0.22$, $\eta_P^2 = 0.45$ [95%CI: 0.00 to 0.67]). Mean peak 10s RR in block 3 for C was 49 ± 17 r·min$^{-1}$. The lowest peak in the block, and in all four blocks, was L which continued to drop from the previous block to 34 ± 4 r·min$^{-1}$. G mean peak 10s RR was 41 ± 6 r·min$^{-1}$ in this block and was the lowest peak RR for the glucose treatment (Figure 4.6).

Block 4

There was no significant difference between treatments for peak 10s RR in the fourth exercise block ($F(2,6) = 0.06$, $p = 0.83$, $\eta_P^2 = 0.02$ [95%CI: 0.00 to 0.16]). In block 4, peak 10s RR for C was 39 ± 13 r·min$^{-1}$ which was the lowest block for water (Figure 4.6). Block lowest was L at 38 ± 7 r·min$^{-1}$, and G 10s peak was 42 ± 25 r·min$^{-1}$. (Figure 4.6).

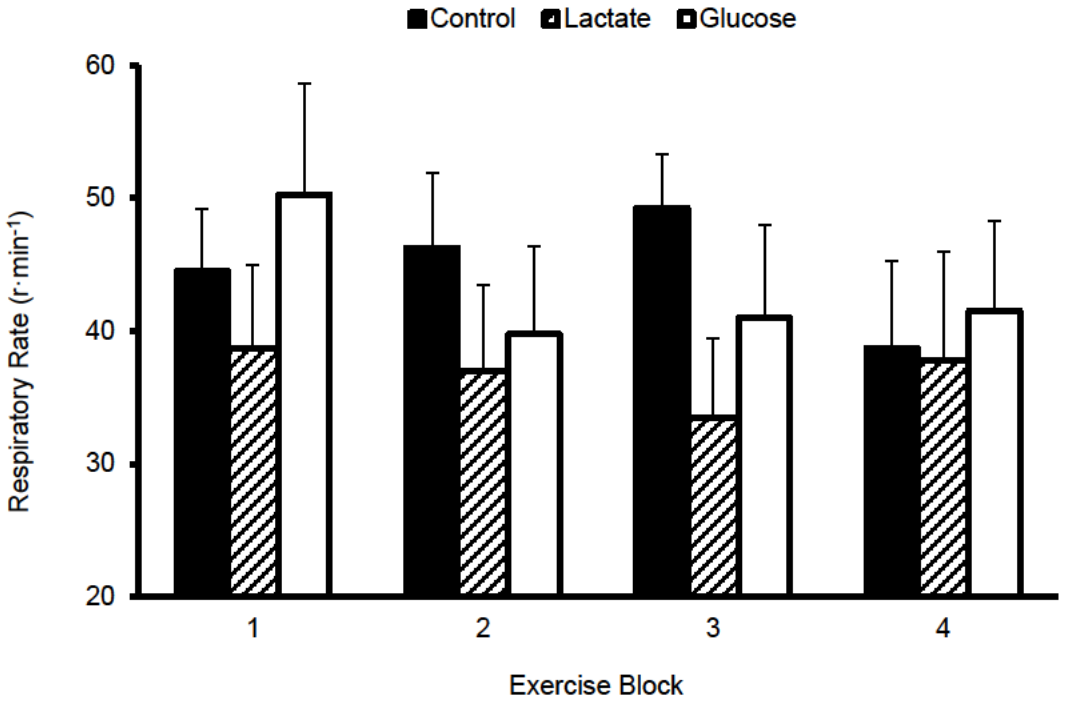


***Figure 4.6 Peak respiratory rate for each condition over the four exercise blocks.***

There was no significant main effect for condition on peak respiration rate and effect size was large in the first half but small in the second (Table 4.5).

***Table 4.5 Peak respiratory rate for each condition by half.***

| | Respiratory rate (r·min$^{-1}$) | | | | | |
|---|---|---|---|---|---|---|
| | Control | Lactate | Glucose | ANOVA p Value | Effect size ($\eta^2_P$) | 95% CI Range |
| 1st half | 44 ± 8 | 38 ± 6 | 47 ± 11 | 0.16 | 0.23 | 0 - 0.51 |
| 2nd half | 42 ± 17 | 36 ± 6 | 40 ± 19 | 0.68 | 0.05 | 0 - 0.30 |

## 4.5 Discussion

The major findings in the current study are that supplementation of 500ml of 1% weight by volume calcium lactate solution will elevate blood lactate concentration significantly at rest and appears to reduce the maximum heart rate and respiratory rate experienced during intermittent high intensity exercise. However, performance was not improved in terms of sprint speed or performance decrement over the duration of the BURST.

### 4.5.1 Lactate absorption rate

Orally consumed lactate is transported from the gastrointestinal tract into the blood by the $MCT_1$ related, splanchnic lactate transporter sMCT (Azevedo et al., 2007). This process occurs rapidly with peak $[La^-]b$ evident at the 30-minutes post drink test (Figure 4.3). This is similar to de Vrese, Koppenhoefer and Barth (1990), and de Vrese and Barth (1991) who reported peak $[La^-]b$ within 40 minutes following ingestion of a lactate solution. Peak values were also comparable to previous findings (de Vrese, Koppenhoefer and Barth, 1990) where peak values reported were 1.8 ± 0.13 $mmol \cdot l^{-1}$. With a mean value of 2.3 ± 1.0 $mmol \cdot l^{-1}$ (Figure 4.3), supplementation of this calcium lactate solution resulted in increased $[La^-]b$ comparable to that seen at an exercise intensity of 50 - 65% $\dot{V}O_{2max}$ (Wall et al., 2011), which is in excess of lactate threshold (LT) (Weltman et al., 1990). Glucose levels throughout the testing period remained relatively unchanged from baseline (Figure 4.4) suggesting there was little or none of the lactate being transformed to glucose through hepatic gluconeogenesis (Hostetler et al., 1969). It would appear that, once in the blood, this lactate was absorbed and oxidised by the various tissues within the body despite the participant being at rest which is consistent with the findings of previous research (Depocas, Minaire and Chatonnet, 1969; Donovan and Brooks, 1986; Gladden, 2004; Jacobs et al., 2013). Theoretically, if a player were to consume a lactate drink 10 minutes prior to the start of the game and again at the start of the half time period as they did in this protocol, they would experience peak absorbed lactate levels at the midpoint of each 40 minutes of playing time. However, gastric emptying speed is reduced during intense

exercise due to a reduction in splanchnic blood flow (Costill and Saltin, 1974). There has also been no previous research on gastric emptying speed following ingested lactate solution. Evidence suggests that when a CHO solution is consumed prior to exercise, gastric emptying rate is less in the first 15 minutes of intermittent shuttle running and small-sided soccer match play compared to the same period of walking (Leiper et al., 2001a, b). The reduction in gastric emptying rate was particularly noticeable when exercise intensity increased beyond 75% $\dot{V}O_{2max}$. Given that mean match intensity during rugby union is ~80% $\dot{V}O_{2max}$ then it seems likely that lactate absorption rate may also be reduced and therefore a longer period would be required prior to exercise. This could be verified using carbon-labelled lactate solution to differentiate between exogenous and endogenously produced lactate present in the blood, but this was beyond the scope of the current study.

At 30 minutes, and at all subsequent post drink tests, the standard deviation from the mean value was greater than at the pre-drink, 10 minutes and 20 minutes post drink tests. This could be due to the varying clearance rates of the individuals involved. It has been shown that individuals with higher levels of cardiovascular training will clear blood lactate more rapidly than their untrained counterparts (Aziz, Chia and Teh, 2000; Bishop and Edge, 2006; Donovan and Brooks, 1983; Hamilton et al., 1991; Mazzeo et al., 1986; Tomlin and Wenger, 2002). However, in this study no testing was conducted to establish $\dot{V}O_{2peak}$ and therefore a correlation could not be assessed.

### 4.5.2 15m Sprint

Performance in invasion sports is linked to the frequency and duration of high intensity actions with elite athletes covering greater distances and performing more frequent high intensity actions (Gabbett, 2010b; Girard, Brocherie and Millet, 2015; Lacome et al., 2014; Mohr et al., 2008; Rampinini et al., 2007; Roberts, et al., 2008). In rugby union, there is a decline in both total relative distance and sprint frequency (running speed >6m·s$^{-1}$) in the second half of a match (Stolen et al., 2005; Tee, Lambert and Coopoo, 2017) with this decline linked to glycogen depletion (Krustrup et al., 2006; Mohr, Krustrup and Bangsbo, 2005). Although differences were not significant it appears that

supplementation of calcium lactate appears to attenuate the decline in sprint performance throughout simulated rugby union match play compared to glucose and water alone. This was particularly evident in the latter stages of the second half (Table 4.3). A possible mechanism for this is that similar to CHO supplementation, lactate will act as an energy substrate (Morris et al., 2011). Lactate can be metabolised aerobically during low to moderate intensity activity, and thus muscle glycogen is preserved to be utilised anaerobically during high intensity bouts (Nicholas et al., 1999; Russell, Benton and Kingsley, 2012; Tsintzas and Williams, 1998; Whitley et al., 1998). It has previously been reported that CHO does not attenuate the decline in sprinting performance throughout invasion sports (Abbey and Rankin, 2009; Ali and Williams, 2009; Foskett et al., 2008) and the current study found the same albeit with a trend for glucose to be more beneficial than water alone (Table 4.3).

Although results non-significant for glucose compared to lactate supplementation (Table 4.3), it would appear sprint performance was maintained to a greater degree by lactate. This supports the idea that supplemented lactate is metabolised more rapidly and to a greater extent than supplemented CHO such as glucose (Azevedo et al., 2007). The same authors reported a 25% increase in exercise performance with lactate supplementation during high intensity exercise compared to carbohydrates alone. Although differences in this study were smaller, Schoeman and Coetzee (2014) reported that throughout 18 elite level rugby union matches, there was no significant difference in high intensity work performed between winning and losing teams suggesting that these small increases in performance may be important to match outcome.

As seen in Table 4.1, the final performance test saw an increase in sprint speed over the three conditions. This is known as the end spurt phenomenon (Tucker, 2009) and was comparable to the final sprint performance decrease shown by female soccer players performing 7 x 30s sprints (van den Tillaar, 2017). This demonstrates that even when instructed to complete the performance test with maximal effort, participants will maintain some kind of reserve until they know there will be no more physical exertion imminent (Ferraz et al., 2012). Billaut et al. (2011) found that when participants performed a pre-determined number of

cycle ergometer sprints, they increased power output in the final sprint, whereas participants who did not know the number of sprints they were to perform showed a steadier decline.

### 4.5.3 Heart rate

There have been contradictory conclusions about the effect of lactate supplementation on HR with researchers reporting increased (Northgraves et al., 2013), unaffected (Fahey et al., 1991), and reduced (Peveler and Palmer, 2012) peak heart rates. As exercise intensity increases, myocardial blood flow and myocardial $\dot{V}O_2$ increase and lactate becomes the preferred fuel for the highly oxidative cardiac muscle (Chatham, Gao and Forder, 1999; Gladden, 2004). As demonstrated in Figure 4.5, peak heart rate was lower in L and this may be due in part to the increased $[La^-]b$ providing more readily available fuel for the heart to work more efficiently in attaining the required cardiac output. Cardiac output (Q) is the product of heart rate and stroke volume (SV) and when lactate availability is reduced, SV and myocardial efficiency are reduced (Barbee, Kline and Watts, 2000), but when lactated Ringer's solution is given to patients, there is an increase of ~14% in left ventricular end-diastolic volume (Concha et al., 2009, Salinas et al., 2006). This suggests that lactate increases SV during intermittent high intensity exercise resulting in a lower peak HR. These findings were in contrast to those of Fahey et al. (1991) who detected no discernible difference in heart rate between lactate, glucose or sweetened water supplementation. This may however be due to the nature of the exercise being undertaken. Similar to Bryner et al. (1998), Fahey et al. (1991) used an exercise protocol involving steady state exercise, in this case 50% $\dot{V}O_{2max}$, which did not sufficiently exert the anaerobic energy systems to require additional energy resources, whereas this protocol measured peak 10s HR following maximal effort sprints resulting in an HR of up to $182 b \cdot min^{-1}$ which is associated with a much higher work intensity. They did however suggest that lactate helped maintain performance during prolonged exercise and would make a useful component in a sports drink.

### 4.5.4 Respiratory rate

Respiratory rate was, in this study, consistently lower in L than either C or G during the four blocks of exercise (Figure 4.6). As with HR, this was not the case with Fahey and colleagues' (1991) work. This was most likely down to exercise protocol, where the lower intensity exercise requirements of the previous studies did not place enough of a metabolic demand on participants to elicit a noticeable difference in RR. Peripheral chemoreceptors, located in the aortic arch, detect increases in $H^+$ concentrations and partial pressure of carbon dioxide ($PCO_2$) as well as decreases in partial pressure of oxygen ($PO_2$) associated with the onset of high intensity exercise (Powers and Howley, 2012). Based on these changes in the blood, RR is increased to maintain $PO_2$ and maintain supply of $O_2$ to the working muscles. If exercise is maintained at a low to moderate intensity below the lactate threshold, there will be no substantial increase in $H^+$ which would cause an increase in RR (Powers and Howley, 2012).

A possible explanation for the lower RR exhibited by L may be linked to the findings of Gurd and colleagues (2005, 2006). This research demonstrated that adaptation of pulmonary oxygen uptake during the transition from rest to moderate intensity exercise, is more rapid following a prior bout of exercise at an intensity great enough to elicit an accumulation of lactate in the circulating blood. They suggest that this is due to the delay in activation of aerobic metabolism, citing pyruvate dehydrogenase (PDH) as the point of metabolic inertia as it controls entry of CHO-derived substrates into the tricarboxylic acid (TCA) cycle. The bout of high intensity exercise prior to the moderate intensity exercise may have started this aerobic process catering for the secondary energy demands more readily and thus allowing more rapid pulmonary $O_2$ uptake adaptation. It may be that the increase in $[La^-]b$ attributable to the supplemented drink, acts in a similar way by providing a readily available substrate for oxidation in the mitochondria.

### 4.5.5 Limitations and further research

In this study, a 1% lactate solution elicited consistent although small differences in magnitude of performance decrement, and both peak HR and RR. The reason such a weak solution was used was to avoid the risk of gastrointestinal discomfort and upset. Other studies such as Bryner et al. (1998) have used 2% lactate solutions without reporting gastrointestinal distress and Swensen et al. (1994) suggest that severe gastrointestinal efflux will only occur once the lactate concentration is ≥ 2.5%. Future research could employ a stronger 2% solution which may help amplify the effect seen between the treatment groups.

Several factors affect an individual's ability to produce and metabolise lactate and if the study were to be repeated, recruitment criteria could be included to provide a more homogenous sample. Muscle metabolism and lactate kinetics change with both maturation (Armstrong, Barker and McManus, 2015) and aging (Massé-Biron et al., 1992), so recruitment of participants with similar age would be beneficial. The magnitude of lactate accumulation is reduced as endurance performance increases (Massé-Biron et al., 1992; Messonnier et al., 2013) and participants with similar $\dot{V}O_{2peak}$ and training status will exhibit similar responses (Messonnier et al., 2013). Introducing recruitment criteria to exclude participants outwith the designated cardiorespiratory fitness parameters would also help reduce the variation seen both at the baseline test and throughout the testing protocol. This could be partly achieved by recruiting participants from similar playing positions and clubs of the same standard as work rate and exercise tolerance, which is fitness dependent, increases as the standard of play increases (Austin, Gabbett and Jenkins, 2011b; Lacome et al., 2014; Mohr et al., 2008; Roberts et al., 2008).

The indication that supplementing lactate solution before undertaking strenuous physical activity leads to a reduced HR and RR compared to both water and glucose-based drinks requires further investigation. This may be especially true for sporting activities such as biathlon where studies show that reduced RR is positively correlated with shooting accuracy, and therefore increased performance (Groslambert et al., 1998).

While invasion sports are predominantly aerobically based, recorded blood lactate levels (McLean, 1992) suggest there is high utilisation of the anaerobic

glycolytic pathway during the high intensity periods of match play. Aslan et al. (2012) found that nearly a quarter of match play occurs at running speed greater than the onset of lactate accumulation, with a drop in lactate concentration between first and second halves of matches. Maintenance of high intensity efforts throughout a match may be linked to an athlete's ability to maintain lactate balance determined by lactate threshold (Edwards et al., 2003), and increased cardiovascular fitness has been linked to better repeated sprint performance (Bishop and Edge, 2006). Therefore, implementing training to improve both lactate kinetics and $\dot{V}O_{2peak}$ may be beneficial to invasion sport performance.

### 4.5.6 Conclusion

This study demonstrated that supplementation of a 1% calcium lactate solution resulted in lower peak HR and RR than either a glucose or flavoured water control during simulated rugby union match play. It also demonstrated that the decrement in sprint performance as exercise duration increases during simulated rugby union match play, is possibly attenuated through the supplementation of calcium lactate although this difference was not statistically significant and there was no difference in sprint speed over 15m. Therefore, based on these findings, the use of calcium lactate could be recommended prior to rugby matches as an ergogenic aid to sustain intermittent sprint performance but not enhance maximal running speed. At an elite level these small changes may enhance overall team performance in this decisive element of success criteria.

This study addressed the first aim of the thesis to determine whether supplementation of lactate could increase sprint speed or reduce the level of performance decrement in intermittent sprint performance throughout invasion sport match play. While the findings suggest there is some merit to using calcium lactate to attenuate the decline in intermittent sprint performance seen throughout invasion sport match play and that lactate metabolism may play an important role in maintaining sprint performance, it is possible this effect could be enhanced through physical training. Research such as Azevedo et al. (2007)

and Morris et al. (2011) which concluded there was an ergogenic benefit of lactate supplementation was conducted with participants exhibiting high levels of cardiorespiratory fitness ($\dot{V}O_{2peak} \geq 60$ ml·kg$^{-1}$·min$^{-1}$). Training which increases aerobic endurance capacity is accompanied by changes in lactate kinetics (Bergman et al., 1999) and a rightward shift in the blood lactate curve (Best et al., 2013; Jakeman, Adamson and Babraj, 2012) suggesting a change in the rate lactate is being metabolised. To address the second aim of the thesis and determine whether the rate at which lactate is transported and metabolised can be increased, it would be prudent to investigate whether a training paradigm such as sprint interval training can be used to enhance both the physical performance indicators important to invasion sport performance such as aerobic and anaerobic fitness, but also the rate of lactate metabolism which could allow greater benefit to be gleaned from supplemented lactate.

# Chapter 5

# Study 2

## 5.1 Abstract

**Introduction:** In youth soccer, 23% of the distance covered happens at speeds above maximal lactate steady state (MLSS) which suggests lactate kinetics may be important to soccer performance. This study sought to determine the effectiveness of sprint interval training (SIT) on improving match specific fitness indices and lactate kinetics in youth soccer players.

**Methods:** Thirteen elite soccer academy players (age 15 ± 0.5y) underwent baseline testing (Wingate anaerobic Test (WAnT) with blood lactate measurements, incremental V̇O2peak and time to exhaustion test, 0-10m and 10-20m sprint performance, repeated 20m sprint performance, and vertical jump performance) before being allocated to control or SIT group. The control group maintained training whilst the SIT group carried out twice-weekly all-out effort cycle sprints consisting of 6 x 10s sprint with 80s recovery.

**Results:** Training elicited significant improvements in $\dot{V}O_{2peak}$ (pre: 54.89 ± 3.09ml· $kg^{-1}$·$min^{-1}$ post: 60.81 ± 5.73ml· $kg^{-1}$·$min^{-1}$; p = 0.001), TTE (pre: 655 ± 54s post: 688 ± 55s; p=0.001), 10 - 20m sprint time (pre: 1.29 ± 0.04s post: 1.25 ± .04s; p=0.02), and peak power during WAnT (pre: 12.4 ± 1.3 $W.kg^{-1}$ post: 15.3 ± 0.7$W.kg^{-1}$; p=0.003) which were not seen in the control group. The changes in performance were significantly correlated to changes in lactate kinetics (time to exhaustion: r=0.77, p = 0.04).

**Conclusions:** Cycle-based SIT is an effective training paradigm for elite youth soccer players and the improvements in match specific fitness indices are associated with changes in lactate kinetics. These increased levels of lactate utilisation may facilitate a greater ergogenic benefit from supplemented lactate solution further enhancing its ability to mitigate the decline in sprint performance seen throughout invasion sport match play.

## 5.2 Introduction

### 5.2.1 Demands of the sport

Soccer is an invasion sport which consists of bouts of high intensity activity followed by longer periods of low to moderate intensity exercise (Aslan et al., 2012). Distance covered in elite youth matches has been reported to be approximately 8500mm (Buchheit et al., 2010a; Lovell et al., 2009; Saward et al., 2016). Percentage of playing time at different intensities varies depending on how the data is calculated. Typically high intensity activity, defined as running speed greater than ~4m·s$^{-1}$, accounts for approximately 20% of the time (Aslan et al., 2012; Buchheit et al., 2017; Castagna et al., 2010; Hill-Haas et al., 2010; Russell et al., 2011; Strøyer, Hansen and Klausen, 2004), with total sprint time being between 3 and 5% (Buchheit et al., 2010a; Rampinini et al., 2007). It has also been noted that high speed movements decline by ~20% in the second half of a match (Harley et al., 2010), suggesting an inability to maintain intensity across a match.

While soccer is generally considered an aerobically dominant sport (Drust, Atkinson and Reilly, 2007), game play taxes both the aerobic and anaerobic energy systems (Paul, Bradley and Nassis, 2015). Average heart rate (HR) during youth soccer matches is approximately 85% of maximum heart rate ($HR_{max}$) (Aşçi, 2016; Aslan et al., 2010; Capranica et al., 2001; Russell et al., 2011; Strøyer, Hansen and Klausen, 2004) which equates to ~70% maximal oxygen uptake ($\dot{V}O_{2max}$) (Bangsbo, Mohr and Krustrup, 2006). Maximal lactate steady state (MLSS) is associated with a mean blood lactate concentration ([La$^-$]b) of 4 mmol·l$^{-1}$ (Billat et al., 2003), and an oxygen uptake ($\dot{V}O_2$) equivalent to 70 - 90% $\dot{V}O_{2max}$ (Jones and Vanhatalo, 2017; Poole et al., 2016), suggesting that most players will be at, or above, MLSS intensity throughout matches. Recorded blood lactate during game play has been shown to be 4 - 8mmol·l$^{-1}$ in 17-year-old players (Aslan et al., 2012) and 1.4 - 8.1mmol·l$^{-1}$ in 11-year-old players (Capricanica et al., 2001). This shows a reliance on anaerobic glycolysis occurring during periods of high intensity activity but also substantial interindividual variation in blood lactate response. Aslan et al. (2012)

demonstrated that 23% of distance travelled during a game occurs at running speeds above MLSS. This suggests that a player's ability to metabolise lactate could be a key determinant of soccer performance. Indeed, blood lactate concentration is greater in elite soccer players compared to non-elite players due to the greater number of high intensity activities at the elite level (Mohr, Krustrup and Bangsbo, 2003). Blood lactate concentration is lower in the second half compared to the first half of matches, which coincides with a drop in HR, number of sprints, and high intensity activity (Aslan et al., 2012; Krustrup et al., 2006; Lovell et al., 2009; Mendez-Villanueva et al., 2012). It has been suggested that the distance a player covers at high intensity may reflect their ability to maintain lactate balance as assessed by lactate threshold (Edwards et al., 2003). Therefore, targeting lactate metabolism through training may be beneficial for soccer performance.

### 5.2.2 Relationship between indices of fitness and match performance

Soccer performance depends on a variety of physiological and physical capacities (Al Haddad et al., 2015), and small changes in these capacities can have a large effect on match performance and outcome (Comfort, Haigh and Matthews, 2012). To meet the demands of elite level soccer, it is important for youth players to have highly developed aerobic capacity (Reilly, Bangsbo and Franks, 2000; Reilly et al., 2000). It has been demonstrated that there is a strong, significant correlation between aerobic capacity and high intensity running during matches in young soccer players ($r = 0.77$, $p = 0.001$) (Castagna et al., 2009). The same is true in adult players and equates to ~2.8% more high intensity running and sprinting in those with greater aerobic capacity (Moir, Krustrup and Bangsbo, 2003). Due to the requirement to repeatedly perform high intensity actions during soccer, repeated sprint ability (RSA) is also an important aspect of soccer specific fitness. RSA is also positively associated with match play performance in terms of high intensity running actions (da Silva, Guglielmo and Bishop, 2010; Gibson et al., 2013; Rampinini et al., 2007). Maximal running speed is also an important element of match performance in young male soccer players (Little and Williams, 2005), with decisive moments such as winning possession of the ball and scoring being influenced by the

speed of the players (Reilly, Bangsbo and Franks, 2000). Increasing maximal running speed capability equates to greater running speeds during match play (Al Haddad et al., 2015). In 16 high level youth players (12.5 ± 1.3 years), Peñailillo et al. (2016) demonstrated that increased muscular strength was associated with increased maximum running speed. Vertical jump (VJ) performance is also related to level of performance in elite soccer (Arnason et al., 2004). These data would indicate that training which improved these performance indices, alongside enhancing whole body lactate kinetics, would be beneficial to the development of elite youth soccer players.

### 5.2.3 Training

#### *5.2.3.1 Small sided games*

Small sided games (SSG) are found extensively throughout invasion sport training regimes and are common in elite level youth soccer (Gabbett and Mulvey, 2008; Polglaze et al., 2015). It is generally agreed that having fewer players at this age allows more opportunities for each player to develop their technical and tactical ability (Capranica et al., 2001; Katis and Kellis, 2009; Randers et al., 2014), and manipulation of playing area and team size is useful for developing strategic elements of the sport in youth players (Silva et al., 2015). Physiological demands during SSG have been found to be similar or greater than match play in both youth and adult male players but intensity during SSG is largely dependent on format (Aşçi, 2016; Beenham et al., 2017; Casamichana, Castellano and Castagna, 2012).

There is, however, debate surrounding the effectiveness of SSG when it comes to developing physiological characteristics desirable for success given that sustained periods of >90% $HR_{max}$ are required during soccer training for aerobic adaptations to be maximised (Hoff and Helgerud, 2004). While some researchers have concluded that elite youth players exhibit a greater HR response during SSG than running based drills (Sassi et al., 2005), others have reported the opposite (Ade, Harley and Bradley, 2014), and it is likely that SSG format is an important element of its success as a training modality (Dellal et al., 2008). In elite level youth players, 12 weeks of SSG was found to be equally as

effective as running based high intensity interval training (HIIT) (4 x 4min at 90 - 95% $HR_{max}$ with 3mins recovery) for improving peak oxygen uptake ($\dot{V}O_{2peak}$) (Impellizzeri et al., 2006). However, when using the same protocol, Eniseler et al. (2017) found SSG did not improve aerobic capacity and HIIT was more effective in this area. Chaouachi et al. (2014) also reported SSG were not as effective as specific training on sprinting performance and agility in youth players following a six-week programme of either SSG or targeted strength and conditioning. However, some research would suggest that SSG was effective for improving performance indices for soccer. Investigating various performance measures in elite youth soccer players, Reilly and White (2005) reported 5 versus 5 SSG to be as effective as running based HIIT for improving countermovement jump, 10m sprint, anaerobic shuttles and multi-stage fitness test performance. Also comparing HIIT and SSG as a conditioning method, Jastrzebski et al. (2014) and Radziminski et al. (2013) both reported that $\dot{V}O_{2max}$ was improved only in SSG in Under-16 players. However, the HIIT protocol used in both studies resulted in session heart rates of less than 90% $HR_{max}$ leading the authors of the latter paper to acknowledge exercise intensity was not sufficient in the HIIT group to elicit an appropriate adaptation. A meta-analysis of HIIT and SSG in youth soccer concluded that SSG and HIIT were equally effective at increasing aerobic capacity, but SSG had no impact on sprint, jumping or RSA performance (Kunz et al., 2019).

While SSG can be an effective modality to develop physical attributes in young soccer players, it has several limitations (Hill-Haas et al., 2011). A major one of these, from a strength and conditioning perspective, is the questionability of SSG to replicate the highest intensity demands of match play. As previously stated, Casamichana, Castellano and Castagna (2012) reported higher overall work rate in SSG but time spent in the highest intensity category (> $5.8m \cdot s^{-1}$) was significantly higher ($p < 0.01$) during match play compared to SSG with average sprint number, duration and distance all greater. The number of repeated high intensity efforts, defined as ≥ 3 efforts at a speed > $3.6m \cdot s^{-1}$ with < 21s recovery between them, was also significantly higher ($p < 0.01$) during match play. One of the determinants of success in invasion sports is the ability

to perform maximum intensity efforts repeatedly (Gabbett, 2010a; Mohr et al., 2008; Portillo et al., 2014), and training modality should reflect this demand (Casamichana, Castellano and Castagna, 2012). A second major limitation of SSG is the inherent risk of injury that comes with this type of activity. Youth players are more susceptible to injury during training than adults (Pfirrmann et al., 2016), with joint sprains and muscle strains being the most common injury in Under-14 to Under-19 players (Brito et al., 2012; Le Gall et al., 2010; Renshaw and Goodwin, 2016). A strong link between high training loads and incidence of injury has been reported in youth soccer players with 72% of injuries being non-contact (Renshaw and Goodwin, 2016; Watson et al., 2017). One of the major factors in the increased risk of training injuries is exposing players to high volumes of high-speed running and sprinting (Malone et al., 2016, 2018). Sharper cutting angles and a greater number of direction changes are also risk factors when reducing playing area and can lead to increased injury during training (Schreurs, Benjainse and Lemmink, 2017). It is therefore advisable to attempt to reduce the incident of injury while still developing physiological conditioning desirable in youth athletes.

#### *5.2.3.2 High intensity training*

Physical conditioning for invasion sports must stress both the aerobic and anaerobic metabolic pathways in order to develop the aerobic capacity required for long duration matches, but also the ability to repeatedly produce high intensity actions (Casamichana, Castellano and Castagna, 2012; Gabbett, 2010b). In youth soccer players, high intensity training (HIT) is an effective training modality to improve both aerobic (Sperlich et al., 2011), and anaerobic (Bravo et al., 2007) components of fitness. The magnitude of physiological adaptation is strongly correlated to the intensity of exercise (Egan et al., 2010; Nordsborg et al., 2010), and the all-out bouts in sprint interval training (SIT) meet this requirement well. In addition to this, SIT replicates the repeated bouts of high intensity effort required during invasion sports (Hodun et al., 2016). Manipulation of rest periods between bouts of exercise during SIT affect the physiological adaptations elicited. Anaerobic adaptations are more pronounced with longer rest periods (1:12 work to rest) while aerobic characteristics develop

more when rest periods are shorter (1:3 work to rest) (Kavaliauskas, Aspe and Babraj, 2015). When phosphocreatine (PCr) is not fully replenished during a short recovery period, more demand is placed on aerobic metabolism to meet the energy requirements in subsequent exercise bouts (Gaitanos et al., 1993). When more time is allowed for PCr regeneration, anaerobic metabolism can remain predominant, and power output is maintained to a greater extent in subsequent sprints (Bogdanis et al., 1996; Connolly, Brennan and Lauzon, 2003; Spierer et al., 2004). Therefore, a work to rest ratio which develops both aerobic and anaerobic elements of fitness is desirable for invasion sport athletes, and this can be achieved utilising a 1:8 work to rest protocol (Kavaliauskas, Aspe and Babraj, 2015). SIT protocols using this ratio have elicited significant improvements in peak power output (PP), time to exhaustion (TTE), critical power (CP), and $\dot{V}O_{2peak}$ (Burgomaster et al., 2005; Kavaliauskas, Aspe and Babraj, 2015; Lloyd Jones, Morris and Jakeman, 2017; Yamagishi and Babraj, 2017). Time motion analysis of youth soccer matches showed that during periods of repeated high intensity efforts, individual sprint duration was ~3s (Buchheit et al., 2010b), making short duration efforts an appropriate training modality. Given that SIT protocols requiring participants to exercise in bouts as short as 6s can elicit similar adaptations to longer bout protocols, it would appear that metabolic demand during the early portion of activity may be the driver for adaptation (Jakeman, Adamson and Babraj, 2012). In a comparison of short duration sprints to longer 30s bouts, it was reported that both 10s (Yamagishi and Babraj, 2017) and 15s (Hazell et al., 2010) sprints were equally effective at improving fitness characteristics. In youth soccer players, short term (5 - 7 weeks) training programmes involving either SIT, HIIT or high-volume endurance training, have been shown to improve aerobic capacity with SIT and HIIT protocols requiring significantly less time commitment per week (Buchan et al., 2012; Faude et al., 2013; Sperlich et al., 2011). These high intensity programmes also reported improved anaerobic elements such as sprint performance and explosive power (Buchan et al., 2012; Sperlich et al., 2011).

Running-based intermittent training (90 - 100% maximal aerobic speed, 2:1 work to rest ratio, 35 - 75 minutes duration), and continuous running training (60

- 70% maximal aerobic speed, 35 - 75 minutes duration) have both been shown to improve lactate kinetics (Gharbi et al., 2008). Intermittent training resulted in greater rate of blood lactate appearance and rate of blood lactate removal, compared to continuous training (Gharbi et al., 2008). In adults, cycle-based HIIT at 80% $\dot{V}O_{2max}$ (4 minute intervals) resulted in improved clearance of blood lactate after a Wingate anaerobic test (WAnT) (Zelt et al., 2014). In triathletes, short duration cycle-based SIT (10 x 6s sprints with 60s passive recovery) has been shown to result in a rightward shift in the blood lactate curve during incremental exercise (Jakeman, Adamson and Babraj, 2012) suggesting changes in either the rate of production or clearance. Given that the lactate kinetic response is similar between adolescents and adults post WAnT (Beneke et al., 2005), then a similar adaptation to lactate metabolism and kinetics may be expected following SIT in adolescent athletes.

### 5.2.4 Aim and hypothesis

The aim of the current study was to determine the effects of a six week, twice-weekly cycle-based SIT protocol (12 sessions in total) on lactate kinetics and performance characteristics in adolescent male soccer players. It was hypothesised that cycle-based SIT would lead to increased rate of lactate utilisation and improve fitness components related to soccer performance.

## 5.3 Methods

### 5.3.1 Study design

A two-group pre-post research test design was employed in this study. To assess responses to cycle-based SIT, the participants were tested pre and post training. All baseline performance tests were conducted within a seven-day period and, following training, post intervention testing commenced within 72 hours of the final training session and was completed within seven days. Post-training tests were performed in the same order as baseline testing. For a given participant, each training and testing protocol was performed within two hours of the same time of the day. The participants were also asked to refrain from

vigorous training for 24 hours before each test and to maintain their normal training and diet routine throughout the study period.

### 5.3.2 Participants

Sixteen elite male youth soccer players were recruited for this study (age: 15 ± 0.5 years) and cluster randomised control was used. Players in the treatment group were recruited from the 2015-16 squad while the control group was recruited from the 2016-17 squad. One participant dropped out of the training group due to injury sustained during a match, and two participants dropped out of the control group after being released by the club, leaving seven in the training group and six in the control group (Table 5.1). The control group were asked to maintain their normal activities whilst the training group took part in twice weekly cycle-based SIT in addition to their regular training programme. All participants were informed of the study verbally and in writing before giving informed consent. The study had ethical approval from Abertay University Ethics Committee and was conducted in accordance with the declaration of Helsinki (www.wma.net, 2013).

***Table 5.1 Participant characteristics.***

| | Control (Pre) | Control (post) | SIT (pre) | SIT (post) |
|---|---|---|---|---|
| Age (y) | 15.0 ± 0.6 | 15.0 ± 0.5 | 15.0 ± 0.6 | 15.0± 0.5 |
| Height (cm) | 175 ± 5 | 175 ± 5 | 180 ± 8 | 180 ± 8 |
| Mass (kg) | 62.1 ± 5.3 | 62.9 ± 5.4 | 70.7 ± 6.5 | 71.6 ± 6.7 |
| Fat Free Mass (kg) | 54.7 ± 4.5 | 56.2 ± 4.6 | 60.6 ± 4.1 | 62.6 ± 4.8 |
| Body Fat (%) | 11.9 ± 2.0 | 10.8 ± 1.0 | 14.0 ± 2.3 | 12.3 ± 1.7 |
| Haematocrit (%) | 44.67 ± 1.37 | 41.83 ± 2.40 | 45.14 ± 3.39 | 43.87 ± 1.46 |
| Haemoglobin ($g \cdot dl^{-1}$) | 13.48 ± 0.75 | 13.58 ± 0.63 | 14.75 ± 0.52 | 14.30 ± 0.75 |

### 5.3.3 Procedures

Over the initial two visits, participants reported to the laboratory at a time suitable for them after a four hour fast. Height was measured (SECA 217 stadiometer, United Kingdom) prior to stepping onto a calibrated bioelectrical impedance meter (BIA) (SC-330ST Tanita Body Composition Analyser, Tanita

Europe BV, Amsterdam, Netherlands), where body fat mass and lean body mass were recorded to the nearest 0.1kg (Table 5.1). Producing a spot of fingertip blood in the same manner as for lactate testing, 10µl of capillary blood was collected using a Dr Lange micropipetter, mixed with the appropriate cuvette and allowed to rest for five minutes before being analysed using a photometer (Dr Lange LP2 miniphotometer, Sint-Denijs-Westrem, Belgium). Familiarisation trials were also performed for the WAnT and the incremental test to exhaustion to become oriented with all testing procedures and training devices. Field based tests were part of club requirements, so all participants were already familiar with these tests.

A sample was taken prior to the test commencing to establish a baseline, immediately following the 30s WAnT, and again at one, three, and five minutes post exercise as described by Zagatto, Beck and Gobatto (2009) since peak $[La^-]b$ is achieved within the first five minutes following intense exercise (Gleeson et al., 1998). Further samples were taken at 10, 15, and 20 minutes post exercise to establish $k_2$.

Haemoglobin and haematocrit

Producing a spot of fingertip blood in the same manner as for lactate testing, 10 µl of capillary blood was collected using a Dr Lange micropipetter, mixed with the appropriate cuvette and allowed to rest for five minutes before being analysed using a photometer (Dr Lange LP2 miniphotometer, Sint-Denijs-Westrem, Belgium).

Wingate anaerobic test

The WAnT was carried out on a cycle ergometer (Monark Ergomedic 891E Bike Ergometer, Vansbro, Sweden) to measure peak power (PP) and average power (AP). Participants were given a “3-2-1-Go” countdown and instructed to sprint ‘all-out’ for 30 seconds against a resistance of 0.075 kg·kg body mass$^{-1}$. The applied load was automatically released once the cadence reached 110

revolutions per minute ($r \cdot min^{-1}$). Verbal encouragement was supplied to participants throughout the 30s maximal intensity sprint.
Blood lactate concentration was determined via fingertip blood samples using a Lactate Pro 2 (Arkray Inc., Kyoto, Japan). Blood lactate concentration was measured prior to the WAnT and then 0, 1, 3, 5, 10, 15, 20, 25 and 30 minutes after the WAnT. Blood was sampled and blood lactate kinetics were modelled as described in Chapter 3.

$\dot{V}O_{2peak}$ and time to exhaustion
Participants performed an incremental test to exhaustion to determine their $\dot{V}O_{2peak}$ on a cycle ergometer (Monark Ergomedic 894E, Varberg, Sweden). Prior to starting the test, participants were connected to a breath by breath oxygen analyser (Metalyzer®3B gas analyser, Cortex, Leipzig, Germany). The test started at an initial power output of 70W, with an additional 35W increase every minute until volitional exhaustion or the participants could not maintain 70 $r \cdot min^{-1}$ despite strong verbal encouragement. Exercise duration at exhaustion was recorded to the nearest second and defined as TTE. $\dot{V}O_{2peak}$ was taken as the highest 15 second average over the incremental test.

20m sprint
Timing gates (Brower Speed Trap II; Brower, Utah, USA) were set up at 0, 10 and 20m at a height of 0.9m. Participants began from a crouched start 0.5m behind the first gate before sprinting as fast as possible through the three gates. Times for 0 - 10m and 10 - 20m were recorded and a four-minute recovery was given prior to repeating the sprint. The average value of three attempts is reported.

Repeated sprint ability
Participants completed six repetitions of a 20m maximal sprint. This was repeated on a 15s cycle with 10m deceleration and 10m active recovery between sprints. Timing gates (Brower Speed Trap II; Brower, Utah, USA) were set up at 0 and 20m at a height of 0.9m and the participants began each sprint

from a crouched start 0.5m behind the first gate and sprinted as fast as possible through the light gates.

Vertical jump

An electronic jump testing system (JumpMat, FSL Electronics Ltd, Cookston, United Kingdom) was used to measure countermovement jump (CMJ) height. Participants were required to keep their hands on their hips throughout each jump to negate the effect of arm swing technique on jump performance. A CMJ was performed three times with a one-minute rest between efforts and average height was used for analysis.

Sprint interval training

The training group performed 6 x 10s sprints against a resistance of 0.075 kg·kg body mass$^{-1}$, with 80s recovery between each sprint (1:8 work to rest ratio). Resistance was applied to the ergometer (Monark Ergomedic 894E, Varberg, Sweden) once the participant reached 110 r·min$^{-1}$. Training sessions were carried out twice per week over six weeks (12 sessions in total). Strong verbal encouragement was provided throughout training sessions to help ensure maximal effort in each sprint.

### 5.3.4 Data analysis

All data are presented as mean ± standard deviation (SD). The bi-exponential model was fitted using QtiPlot to generate values for A, $k_1$ and $k_2$. Statistical analysis of the remaining data was performed using IBM® SPSS® Version 28.0 (IBM Corp., Armonk, N.Y., USA) and Microsoft Excel (Microsoft Corporation, Redmond, Washington, USA). A 2 x 2 repeated measures analysis of variance (ANOVA) was performed to determine effect of group, time, and group by time interaction. Assumptions of homogeneity of variance were tested using Mauchly's sphericity test and where this was violated, the Greenhouse-Geisser value was to be used to adjust degrees of freedom unless this estimate was > 0.75, in which case the less conservative Huynh-Feldt estimate would be used. Partial eta squared ($\eta_p^2$) was calculated to determine effect size (ES) along with

95% confidence intervals (CI). Effect size was defined as; small = 0.01 - 0.05, medium = 0.06 - 0.13, and large ≥ 0.14 (Cohen, 1988).
Pearson's correlations were used to assess the relationships between lactate kinetic parameters and performance measures. Statistical significance for group means difference was set a priori at $p \leq 0.05$.

## 5.4 Results

### 5.4.1 Haematology

There was a significant main effect for group with a large effect size ($F(1,5) = 16.95$, $p = 0.01$, $\eta_P^2 = 0.77$ [95%CI: 0.10 to 0.88]). No main effect was apparent for time with a small effect size ($F(1,5) = 0.22$, $p = 0.66$, $\eta_P^2 = 0.04$ [95%CI: 0.00 to 0.45]).), and there was no group x time interaction although effect size was large ($F(1,5) = 0.92$, $p = 0.38$, $\eta_P^2 = 0.16$ [95%CI: 0.00 to 0.56]).) (Table 5.1).

### 5.4.2 Power

WAnT peak power
There was a significant main effect for time and group x time interaction with large effect sizes ($F(1,5) = 9.85$, $p = 0.03$, $\eta_P^2 = 0.66$ [95%CI: 0.00 to 0.83]).; $F(1,5) = 10.19$, $p = 0.02$, $\eta_P^2 = 0.67$ [95%CI: 0.00 to 0.83]).). No main effect for group was evident although effect size was large ($F(1,5) = 4.78$, $p = 0.08$, $\eta_P^2 = 0.49$ [95%CI: 0.00 to 0.74]) (Table 5.2).

WAnT average power
There was a significant main effect for group and group x time interaction with large effect sizes ($F(1,5) = 7.43$, $p = 0.04$, $\eta_P^2 = 0.60$ [95%CI: 0.00 to 0.79]; $F(1,5) = 14.80$, $p = 0.01$, $\eta_P^2 = 0.75$ [95%CI: 0.07 to 0.87]). No main effect for time was evident although effect size was large ($F(1,5) = 1.51$, $p = 0.27$, $\eta_P^2 = 0.23$ [95%CI: 0.00 to 0.60]) (Table 5.2).

Vertical jump

There was no main effect for group, time, or group x time interaction for CMJ (F (1,5) = 4.49, p = 0.09, $\eta_P^2$ = 0.470 [95%CI: 0.00 to 0.73]; F (1,5) = 0.46, p = 0.53, $\eta_P^2$ = 0.08 [95%CI: 0.00 to 0.50]; F (1,5) = 1.48, p = 0.29, $\eta_P^2$ = 0.23 [95%CI: 0.00 to 0.60]) (Table 5.2).

### 5.4.3 Endurance performance

$\dot{V}O_{2peak}$

There was no main effect for group or time, and no group x time interaction (F (1,5) = 0.28, p = 0.62, $\eta_P^2$ = 0.05 [95%CI: 0.00 to 0.46]; F (1,5) = 3.62, p = 0.12, $\eta_P^2$ = 0.42 [95%CI: 0.00 to 0.71]; F (1,5) = 1.47, p = 0.28, $\eta_P^2$ = 0.23 [95%CI: 0.00 to 0.60]) (Table 5.2) for peak oxygen uptake during incremental cycling to exhaustion. ES was small for group but large for time, and group x time interaction.

Time to exhaustion

There was no main effect for group or time (F (1,5) = 5.17, p = 0.07, $\eta_P^2$ = 0.51 [95%CI: 0.00 to 0.75]; F (1,5) = 0.07, p = 0.80, $\eta_P^2$ = 0.01 [95%CI: 0.00 to 0.38]). ES size for group was large but small for time. A main group x time effect was evident with a large effect size (F (1,5) = 24.34, p = 0.004, $\eta_P^2$ = 0.83 [95%CI: 0.20 to 0.91]) (Table 5.2).

### 5.4.4 Sprint performance

Acceleration

For acceleration there was a main effect for time with a large ES (F (1,5) = 10.65, p = 0.02, $\eta_P^2$ = 0.68 [95%CI: 0.01 to 0.84]). There was no main effect for group, and no group x time interaction (F (1,5) = 2.61, p = 0.17, $\eta_P^2$ = 0.34 [95%CI: 0.00 to 0.67]; F (1,5) = 0.06, p = 0.82, $\eta_P^2$ = 0.01 [95%CI: 0.00 to 0.37]). ES for group was large, but negligible for time and group x time interaction (Table 5.2).

Maximum speed

Maximum speed showed no main effect for group (F (1,5) = 2.60, p = 0.17, $\eta_P^2$ = 0.34 [95%CI: 0.00 to 0.67]) or time (F (1,5) = 0.51, p = 0.83, $\eta_P^2$ = 0.01 [95%CI: 0.00 to 0.51]). There was a group x time interaction (F (1,5) = 9.17, p = 0.03, $\eta_P^2$ = 0.65 [95%CI: 0.00 to 0.82]). ES for group, and group x time was large, but negligible for time (Table 5.2).

RSA

There was no main effect for group or time, and no group x time interaction (F (1,5) = 5.77, p = 0.06, $\eta_P^2$ = 0.54 [95%CI: 0.00 to 0.76]; F (1,5) = 1.05, p = 0.35, $\eta_P^2$ = 0.17 [95%CI: 0.00 to 0.57]; F (1,5) = 0.16, p = 0.70, $\eta_P^2$ = 0.03 [95%CI: 0.00 to 0.43]; Table 5.2) for best sprint time. For average 20m sprint time over 6 sprints, there was no main effect for group or time and no group x time interaction (F (1,5) = 2.74, p = 0.16, $\eta_P^2$ = 0.35 [95%CI: 0.00 to 0.67]; F (1,5) = 1.15, p = 0.33, $\eta_P^2$ = 0.19 [95%CI: 0.00 to 0.58]; F (1,5) = 2.55, p = 0.17, $\eta_P^2$ = 0.34 [95%CI: 0.00 to 0.66]; Table 5.2). Performance decrement showed no main effect for group (F (1,5) = 0.27, p = 0.63, $\eta_P^2$ = 0.05 [95%CI: 0.00 to 0.46]). There was a main effect for time and a group x time interaction (F (1,5) = 6.65, p = 0.05, $\eta_P^2$ = 0.57 [95%CI: 0.00 to 0.78]; F (1,5) = 10.75, p = 0.02, $\eta_P^2$ = 0.68 [95%CI: 0.01 to 0.84]; Table 5.2).

***Table 5.2 Changes in performance for control and sprint training groups. Values shown are group mean ± SD.***

| | Control group (n = 6) | | | | Sprint training group (n = 7) | | | |
|---|---|---|---|---|---|---|---|---|
| Variable | Pre-test | Post-test | p value | ES ± 95% CI | Pre-test | Post-test | p value | ES ± 95% CI |
| $\dot{V}O_{2peak}$ (ml·kg$^{-1}$·min$^{-1}$) | 53.39 ± 7.51 | 59.41 ± 8.10 | 1.00 | <0.01 ± 0.30 | 54.89 ± 3.09 | 60.81 ± 5.73 | 0.001* | 1.29 ± 1.38 |
| TTE (s) | 596 ± 63 | 562 ± 85 | 0.05* | -0.46 ± 1.70 | 655 ± 54 | 688 ± 55 | 0.001* | -0.62 ± 0.54 |
| 0 -10m (s) | 1.81 ± 0.09 | 1.86 ± 0.10 | 0.13 | 0.53 ± 0.00 | 1.76 ± 0.10 | 1.78 ± 0.05 | 0.56 | 0.25 ± 0.03 |
| 10 -20m (s) | 1.32 ± 0.07 | 1.35 ± 0.08 | 0.08 | -1.00 ± 0.00 | 1.29 ± 0.04 | 1.25 ± 0.04 | 0.02* | 0.16 ± 0.02 |
| $RSA_{best}$ (s) | 3.24 ± 0.15 | 3.24 ± 0.15 | 0.91 | 0.00 ± 0.00 | 3.13 ± 0.06 | 3.13 ± 0.08 | 0.76 | 0.00 ± 0.01 |
| $RSA_{mean}$ (s) | 3.34 ± 0.10 | 3.42 ± 0.17 | 0.21 | 0.57 ± 0.04 | 3.27 ± 0.08 | 3.26 ± 0.1 | 0.63 | -0.11 ± 0.01 |
| $RSA_{total}$ (s) | 20.05 ± 0.67 | 20.54 ± 1.04 | 0.25 | 0.56 ± 0.21 | 19.10 ± 1.25 | 19.55 ± 0.59 | 0.44 | 0.46 ± 0.35 |
| $RSA_{dec}$ (%) | -3.04 ± 0.46 | -5.58 ± 3.53 | 0.25 | -0.87 ± 1.02 | -4.36 ± 2.02 | -4.20 ± 1.71 | 0.85 | 0.09 ± 0.16 |
| PP (W·kg$^{-1}$) | 13.15 ± 1.33 | 13.19 ± 1.49 | 0.97 | -0.60 ± 0.05 | 12.38 ± 1.25 | 15.34 ± 0.66* | 0.003* | 2.96 ± 0.31 |
| AP (W·kg$^{-1}$) | 8.77 ± 0.28 | 8.61 ± 0.54 | 0.37 | -0.35 ± 0.15 | 9.07 ± 0.37 | 9.52 ± 0.32 | 0.06 | 1.30 ± 0.03 |
| CMJ (cm) | 31.83 ± 2.93 | 33.50 ± 2.59 | 0.92 | 0.03 ± 0.14 | 37.86 ± 5.46 | 38.00 ± 5.20 | 0.27 | 0.60 ± 0.19 |

***Abbreviations: $\dot{V}O_{2peak}$ is the highest rate of oxygen uptake recorded, TTE is time to exhaustion, 0-10m is sprint time over first 10m, 10 -20m sprint is sprint time over second 10m, $RSA_{best}$ indicates best sprint time, $RSA_{mean}$ indicates average sprint time, $RSA_{dec}$ is the percentage of performance decrement, PP is peak power, AP is average power, and CMJ is countermovement jump height. * indicates a significant ($p \leq 0.05$) change from Pre-test.***

### 5.4.5 Lactate kinetics

There was no main effect for group or time, and no group x time interaction (F (1,5) = 3.68, p = 0.10, $\eta_P^2$ = 0.38 [95%CI: 0.00 to 0.71]; F (1,5) = 0.68, p = 0.44, $\eta_P^2$ = 0.10 [95%CI: 0.00 to 0.53]; F (1,5) = 3.76, p = 0.10, $\eta_P^2$ = 0.39 [95%CI: 0.00 to 0.71]) for extravascular release of lactate from exercise metabolism (A) (Table 5.3). ES was large for group and group x time interaction, but medium for time.

For the rate of lactate accumulation ($k_1$) there was a main effect for group (F (1,5) = 11.03, p = 0.02, $\eta_P^2$ = 0.65 [95%CI: 0.01 to 0.84]). There was no main effect for time, and no group x time interaction (F (1,5) = 0.05, p = 0.83, $\eta_P^2$ = 0.01 [95%CI: 0.00 to 0.36]; F (1,5) = 0.05, p = 0.83, $\eta_P^2$ = 0.01 [95%CI: 0.00 to 0.36]). ES for group was large, but negligible for time and group x time interaction (Table 5.3).

Rate of lactate clearance ($k_2$) showed no main effect for group or time and ES was small for each (F (1,5) = 0.26, p = 0.63, $\eta_P^2$ = 0.04 [95%CI: 0.00 to 0.46]; F (1,5) = 0.29, p = 0.61, $\eta_P^2$ = 0.05 [95%CI: 0.00 to 0.47]; Table 5.3). There was a significant group x time interaction with a large ES (F (1,5) = 62.69, p < 0.001, $\eta_P^2$ = 0.91 [95%CI: 0.52 to 0.96]) (Table 5.3).

There was no main effect for time, and no group x time interaction on maximum blood lactate concentration ($[La^-]b_{max}$) (F (1,5) = 1.63, p = 0.25, $\eta_P^2$ = 0.21 [95%CI: 0.00 to 0.61]; F (1,5) = 1.40, p = 0.28, $\eta_P^2$ = 0.19 [95%CI: 0.00 to 0.60]), and ES for both was large (Table 5.3). A main effect for group was evident with a large effect size (F (1,5) = 8.56, p = 0.03, $\eta_P^2$ = 0.59 [95%CI: 0.00 to 0.81]) (Table 5.3)

There was no main effect for group or time, and no group x time interaction (F (1,5) = 4.09, p = 0.09, $\eta_P^2$ = 0.41 [95%CI: 0.00 to 0.72]; F (1,5) = 0.66, p = 0.45, $\eta_P^2$ = 0.10 [95%CI: 0.00 to 0.53]; F (1,5) = 2.76, p = 0.15, $\eta_P^2$ = 0.32 [95%CI: 0.00

to 0.67]; Table 5.3) for time to maximum blood lactate concentration (T[$La^-$]$b_{max}$). ES was large for group and group x time interaction was large, but medium for time (Table 5.3).

There was no main effect for group or time, and no group x time interaction (F (1,5) = 4.09, p = 0.09, $\eta_P^2$ = 0.41 [95%CI: 0.00 to 0.72]; F (1,5) = 0.66, p = 0.45, $\eta_P^2$ = 0.10 [95%CI: 0.00 to 0.53]; F (1,5) = 2.76, p = 0.15, $\eta_P^2$ = 0.32 [95%CI: 0.00 to 0.67]; Table 5.3) for turn point (TP). ES was large for group and group x time interaction was large but medium for time (Table 5.3).

***Table 5.3 Lactate kinetic variables for control and sprint training groups following 30s WAnT. Values shown are group mean ± SD.***

| Variable | Control group (n = 6) | | | | Sprint training group (n = 7) | | | |
|---|---|---|---|---|---|---|---|---|
| | Pre-test | Post-test | p value | Standardised differences, ES ± 95% CI | Pre-test | Post-test | p value | Standardised differences, ES ± 95% CI |
| A ($mmol \cdot l^{-1}$) | 15.85 ± 2.17 | 13.89 ± 3.19 | 0.23 | -0.72 ± 0.58 | 15.42 ± 3.82 | 18.73 ± 3.10 | 0.13 | 0.95 ± 0.38 |
| $k_1$ ($mmol \cdot min^{-1}$) | 0.71 ± 0.36 | 0.70 ± 0.22 | 0.97 | -0.03 ± 0.08 | 0.81 ± 0.33 | 0.86 ± 0.29 | 0.78 | 0.16 ± 0.02 |
| $k_2$ ($mmol \cdot min^{-1}$) | 0.11 ± 0.03 | 0.08 ± 0.03 | 0.03* | -1.00 ± 0.00 | 0.08 ± 0.03 | 0.12 ± 0.04 | 0.001* | 1.13 ± 0.01 |
| $[La^-]b_{max}$ ($mmol \cdot l^{-1}$) | 11.92 ± 0.84 | 11.79 ± 3.06 | 0.93 | -0.06 ± 1.26 | 13.16 ± 3.42 | 15.39 ± 1.67 | 0.17 | 0.83 ± 0.92 |
| $T[La^-]b_{max}$ (min) | 3.51 ± 1.26 | 3.81 ± 1.16 | 0.39 | 0.25 ± 0.06 | 3.48 ± 1.06 | 2.79 ± 0.53 | 0.20 | -0.82 ± 0.28 |
| TP (min) | 7.02 ± 2.53 | 7.61 ± 2.32 | 0.39 | 0.24 ± 0.12 | 6.96 ± 2.11 | 5.59 ± 1.06 | 0.39 | -0.82 ± 0.55 |

***Abbreviations: A is extravascular release of lactate, $k_1$* is rate of lactate accumulation, $k_2$ is rate of lactate clearance, $[La^-]b_{max}$ is maximal blood lactate concentration, $T[La^-]b_{max}$ is time to maximal blood lactate concentration, and TP is turn point. * *indicates a significant ($p \leq 0.05$) change from Pre-test.***

***Table 5.4 Correlation between training outcome and lactate kinetic parameter. * indicates p ≤ 0.05 training variable to lactate kinetic response.***

| | PP ($W \cdot kg^{-1}$) | AP ($W \cdot kg^{-1}$) | CMJ (cm) | $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$) | TTE (s) | 0 - 10 m sprint (s) | 10 - 20m sprint (s) | 20m sprint (s) | RSA total time (s) |
|---|---|---|---|---|---|---|---|---|---|
| A (mmol·l-1) | 0.01 | 0.31 | 0.19 | -0.03 | 0.56 | -0.66 | -0.29 | -0.53 | -0.60 |
| k1 (min-1) | -0.38 | -0.74 | -0.04 | -0.45 | -0.64 | 0.57 | 0.02 | 0.35 | 0.41 |
| k2 (min-1) | -0.16 | 0.50 | 0.20 | 0.33 | 0.77* | -0.54 | -0.71 | -0.66 | -0.42 |
| [La-]bmax (mmol·l-1) | 0.13 | -0.28 | 0.18 | -0.44 | -0.21 | -0.28 | -0.01 | -0.18 | -0.39 |
| T[La-]bmax (min) | 0.35 | 0.40 | -0.04 | 0.26 | 0.28 | -0.23 | 0.41 | 0.06 | -0.14 |
| TP (min) | 0.35 | 0.40 | -0.04 | 0.26 | 0.28 | -0.23 | 0.41 | 0.06 | -0.14 |

In SG there was a significant (p = 0.04) positive correlation between $k_2$ and TTE following six weeks of SIT, which was not seen in CG (Table 5.4).

## 5.5 Discussion

The major findings in the current study are a six week, twice-weekly cycle-based SIT intervention is an effective training modality to improve performance indicators and alter lactate kinetics in youth soccer players. There was a significant (p ≤ 0.05) improvement in endurance performance ($\dot{V}O_{2peak}$ 10.8%, TTE 5.1%), anaerobic power (PP 23.9%), maximum running speed (10 - 20m 3.7%), and lactate exchange rate ($k_2$ 48.5%). Additionally, these changes in lactate kinetics are correlated with endurance performance in adolescent soccer players.

Utilising high intensity intervals has been shown to be more time effective than longer protocols such as SSG and moderate intensity continuous training (MICT) in youth athletes (15.5 ± 2.2 years), leaving more training time for technical and tactical development (Engel et al., 2018). In this study, training duration was 18 minutes per session which is shorter than that previously

employed by Faude et al. (2013) and Sperlich et al. (2011) who utilised running-based protocols lasting 29 - 33 minutes. Therefore, it appears that performing cycle-based SIT training twice-weekly, in addition to the regular soccer training sessions, can help players develop aerobic and anaerobic capacity. It also effectively increases the rate at which endogenously produced lactate is cleared from the blood through utilisation in other tissues within the body.

### 5.5.1 Endurance performance

Aerobic capacity has been identified as a key element in the development of elite soccer players (Ostojic, 2004), and increased maximal oxygen uptake is associated with greater distance covered during match play (Helgerud et al., 2001) and at a greater running speed (Rampinini et al., 2007; Strøyer, Hansen and Klausen, 2004). Prior to training, maximal oxygen uptake in this study was similar to that previously reported in youth soccer players (Meckel, Machnai and Eliakim, 2009; Russell et al., 2011). Following SIT, $\dot{V}O_{2peak}$ increased in the training group significantly to 60.8 ± 5.7 ml· $kg^{-1}$·$min^{-1}$ (Table 5.2) which is comparable to values reported in elite level youth players (14.0 ± 0.2 years; 63.7 ± 8.5ml· $kg^{-1}$·$min^{-1}$) by Strøyer, Hansen and Klausen (2004). Increase in $\dot{V}O_{2peak}$ was greater than that reported in prepubertal soccer players following high intensity and strength training (Ferrete et al., 2014) and post pubertal elite players following SSG training (Jastrzebski et al., 2014; Radziminski et al., 2013). It was, however, comparable to level of change in elite youth soccer players following eight weeks of HIIT (4 x 4min at 90 - 95% $HR_{max}$) (Helgerud et al., 2001), despite vastly reduced time commitment (120s sprinting per week versus 32 min sprinting per week). Indeed, a recent study by Yamagishi and Babraj (2017) has shown that sprint duration does not affect endurance adaptation to sprint exercise. Additionally, Egan et al. (2010) reported that intensity of exercise was a determining factor in magnitude of change while Massicote and McNab (1974) reported that in adolescents, only those training at the highest intensities significantly increased maximal oxygen uptake. Traditional endurance training increases endurance performance by increasing maximal cardiac output and therefore oxygen delivery to the working skeletal muscles (Gibala, Bostad and McCarthy, 2019). Following 6 weeks MICT there

was a significant increase in peak cardiac output and haemoglobin mass (Montero et al., 2015), with performance changes being attributed to both these factors. In this study however, haemoglobin and haematocrit were unaltered following training, suggesting that changes were unlikely due to increased oxygen carrying capacity of the blood. It is possible that cardiac output was increased, and this has been evident following HIIT and SIT previously (Astorino et al., 2017; Trilk et al., 2011). High intensity training has also been shown to improve oxidative capacity of skeletal muscle (Burgomaster et al., 2008; Ma et al., 2013; Talanian et al., 2006). This manifests as increased capillary density within the muscles allowing greater oxygen delivery to the working muscles (Cocks et al., 2013, Raleigh et al., 2018; Tan et al., 2018). Increases in peroxisome proliferator-activated receptor γ coactivator 1α (PGC-1α), have also been reported following high intensity training (Adhihetty et al., 2003; Gibala et al., 2012). PGC-1α has been reported to be a main driver for increasing mitochondrial content in skeletal muscle following exercise (Laursen, 2010; Valero-Grinan, 2014; Uguccioni, D'souza and Hood, 2010) indicating this type of training is effective for increasing mitochondrial content and density. This increase in mitochondrial content and density is accompanied by increased maximal activity of citrate synthase (CS) and cytochrome c oxidase (COX) following SIT (Burgomaster et al., 2006; Gibala et al., 2006) allowing elevated activity in the tricarboxylic acid (TCA) cycle and electron transport chain (ETC), which enhances aerobic capacity through increased aerobic energy production. Given that sprint duration does not appear to affect training adaptations (Yamagishi and Babraj, 2017), this suggests that the mitochondrial adaptation must be driven by similar events in the skeletal muscle during training. Philp et al. (2012) have speculated about the importance of glycogen depletion for mitochondrial biogenesis and training adaptations in skeletal muscle. Given that the glycogen depletion is the same between sprints of different duration (Gaitanos, 1993), then this seems a likely driver for endurance adaptations to high intensity training. However, more research is needed to determine the importance of glycogen depletion to adaptation. Muscle glycogen depletion has been linked to the reduction in performance throughout soccer matches (Krustrup et al., 2006), and players with a lower aerobic capacity have a greater

decline during game play (Rampinini, 2008). The increase in aerobic capacity demonstrated by increased TTE following SIT may also be due to increased glycogen stores. Exercise intensity at exhaustion (~100% $\dot{V}O_{2peak}$) is beyond MLSS (70 - 90% $\dot{V}O_{2max}$) suggesting energy production in the latter stages of incremental exercise is predominantly supplied through anaerobic glycolysis and increased intramuscular stores would facilitate this for longer. However, elevated intramuscular lactate levels inhibit glycolysis (Bangsbo et al., 1992; Choi et al., 2002) meaning that if improvements in performance were due to increased glycolysis, lactate utilisation or removal must also be increased. In the present study, there was a significant positive correlation between $k_2$ and TTE suggesting increased time at high intensity may have been facilitated by an increased rate of lactate clearance from the working muscles.

### 5.5.2 Sprint performance and repeated sprint ability

Acceleration and maximum speed

Acceleration (0 - 10m) and maximum running speed (10 - 20m) (Table 5.2) are similar to previously reported data for elite male Under-16 soccer players (Buchheit et al., 2010; Gioldasis, Bekris and Gissis, 2014; Harley et al., 2010; Mendez-Villanueva et al., 2012; Nikolaïdis et al., 2018). In this study, the effect of the protocol on acceleration was unclear which agrees with Taylor et al. (2015), whose meta-analysis of SIT protocols of 2 - 12 weeks utilising sprints ≤10s, reported only a small effect on 0 - 10m running speed (d = -0.42 ± 0.24). Bravo et al. (2007) likewise found no change in 0 - 10m time following 7 weeks of either HIIT at 90 - 95% $HR_{max}$ or repeated 40m shuttle sprints in amateur youth soccer players (17.3 ± 0.6 years). However, Taylor et al. (2016) reported that two weeks (6 sessions) of running-based SIT improved 0 - 10m times in adult (24 ± 4 years) soccer players by 6.6 ± 7.4%. Acceleration in invasion sport athletes is linked to several kinematic factors such as stride length, torso lean and degree of knee lift (Hewit, Cronin and Hume, 2013), none of which were addressed in this study. Developing acceleration requires a specific training approach (Zafeiridis et al., 2005) and in professional soccer players, while there is significant correlation between acceleration and maximum speed ($p = 0.0005$), they are unique attributes and will not change uniformly (Little and

Williams, 2005). It could be the high volume of sprint accelerations (n = 49) required in the repeated sprint group of Taylor and colleagues' study had a technique enhancing effect.

Maximum running speed improved significantly ($p = 0.02$) following SIT which reflects improvements seen in other studies. 10 - 20m time was reduced by 3.7% which was slightly less than the 4.5% decrease seen in youth soccer players reported by Sperlich et al. (2011). This difference may have been due to the players in this study being faster at the initial test point ($6.56 m \cdot s^{-1}$ versus $5.73 m \cdot s^{-1}$), and therefore expected improvement would also be less. A worthwhile increase in sprint performance in invasion sports is 0.8% (Paton, Hopkins and Vollebregt, 2001) so the improvement seen would still be beneficial to match performance. Increased maximum running speed is attributable to increased ground reaction forces rather than more rapid limb movement (Weyand et al., 2000), and the increased maximum speed evident in this study indicates that cycle-based SIT effectively increases force production capacity transferable to running activities. In contrast to the current study, a seven-week intervention using repeated sprint training at 90% or 100% of maximal sprint velocity, did not improve single sprint or repeated sprint performance in adolescent soccer players (Haugen et al., 2015).

Repeated sprint ability

Following SIT there was a trend for RSA to improve. While fastest sprint remained unchanged, average sprint time improved 0.3%, and performance decrement was 0.16% less following training (Table 5.2). Following SIT, there was no improvement in the fastest sprint in SG (Table 5.2). Prior to training, the fastest 20m sprint during the repeated sprint tests was not different to the 20m sprint time ($p = 0.06$). However, following training, the improvement seen in 20m sprint time (Table 5.2) was not reflected in the fastest sprint during repeated sprint test leading to a significantly ($p < 0.001$) longer time. This lack of improved fastest time was also noted by Eniseler et al. (2017) who reported best sprint to be slower during repeated sprint testing following training. It is possible this lack of change is in part due to the use of pacing strategies by participants, which has been shown to impact RSA when the number of sprints

is known (Billaut et al., 2010). Following high intensity training, youth soccer players have shown an improvement in average sprint time of approximately 2.5% (Bravo et al., 2007; Buchheit et al., 2010; Tønnessen et al., 2011), which is larger than in the current study (Table 5.2). The lack of maximal effort in each repetition despite strong verbal encouragement may also have affected average time. Percentage decrement during repeated sprints was similar to previously reported values for youth soccer players, and improvement following cycle-based SIT matched improvements after six weeks of running-based HIIT (Eniseler et al., 2017). This improvement did not however match other results which showed no reduction in performance decrement following repeated sprint training (Bravo et al., 2007).

Possible adaptations leading to increased RSA performance include increased glycogen stores (Ørtenblad et al., 2000) and increased aerobic capacity (Bogdanis et al., 1996). Following six SIT sessions over three weeks, Hamlin et al. (2017) found a reduction in fatigue during 8 x 20m repeated running sprints in adults. Similar results were seen in youth soccer players (16.6 ± 0.6 years) where 12 sessions of resisted sprint training over seven weeks resulted in a reduction in mean sprint time during (Borges et al., (2016). It has been reported that RSA is primarily fuelled through PCr-mediated ATP during the work phase (Glaister, 2005; Spencer et al., 2005), and regeneration of PCr is associated with performance maintenance in the latter sprints (Bogdanis et al., 1996; Little and Williams, 2007). In both aforementioned studies, 20s rest was provided between sprints compared to 15s in this study, and the additional recovery time may have increased the level of PCr regeneration allowing increased RSA. As with da Silva, Guglielmo and Bishop (2010) and Gibson et al. (2013), this study found a strong correlation between aerobic capacity and RSA ($r = -0.64$, $p = 0.12$).

### 5.5.3 Power

WAnT

Prior to SIT, PP and AP (12.38 ± 1.25W·kg$^{-1}$, 9.07 ± 0.37W·kg$^{-1}$) were similar to WAnT data for both non-elite adolescent players aged 14 - 16 years (Nikolaïdis, 2011; Nikolaïdis et al., 2018) and elite players aged 16 - 18 years (Meckel, Machnai and Eliakim, 2009). Following SIT, participants significantly ($p = 0.003$) improved peak power by 2.96 W·kg$^{-1}$, and the improvement in average power of 0.45 W·kg$^{-1}$ was approaching significance ($p = 0.057$). Significant improvements in PP and AP after 12 sessions of 4 - 6 x 15s SIT have been previously reported in young healthy males (Zelt et al., 2014). Nikolaïdis (2011) suggested that PP during WAnT is determined primarily through PCr degradation and AP is a reflection of anaerobic glycolysis. However, this may be an oversimplification of the energetics during short duration maximal effort bouts, with glycolysis playing a key role in energy production in sprints as short as five seconds (Beneke et al., 2002). Therefore, it may be that improvements in PP and AP are linked to an increase in energy production through anaerobic glycolysis. This was demonstrated by Linossier et al. (1997) who found that following a seven-week short interval duration SIT intervention, phosphofructokinase (PFK) and lactate dehydrogenase (LDH) activity was elevated in skeletal muscle of moderately active young adults. Increased PP may also be due to neuromuscular adaptations with two weeks of sprint cycling resulting in greater neuromuscular activation (Martinez-Valdes et al., 2017). Alternatively, increased PP following SIT may, in part, be due to increased resting muscle glycogen levels (Jakeman, Adamson and Babraj, 2012). Cycle-based HIT (4 - 6 x 30s) has been shown to increase total skeletal muscle glycogen concentration (Burgomaster et al., 2005), although no studies have been carried out looking at skeletal muscle adaptation to SIT specifically in children or adolescents. However, given that glycolytic enzyme activity in adolescents is similar to young adults (Berg, Kim and Keul, 1986) and the improvements in performance recorded are similar to those seen in adults, then it seems reasonable to assume similar skeletal muscle adaptation in adolescent soccer players.

Counter-movement jump

CMJ performance was similar to that reported previously in adolescent soccer players (Buchheit et al., 2010; Papaevangelou et al., 2012). Six weeks of SIT had no effect on CMJ performance despite a significant increase in anaerobic power capacity which has been linked to jumping ability in youth soccer players (Nikolaïdis et al., 2018). These findings, that high intensity training does not enhance jumping performance, are in agreement with Taylor et al. (2015) who concluded that repeated sprint training had only a small effect on vertical jump performance. Bravo et al. (2007) and Sperlich et al. (2011) both reported increases in sprint performance but no change in CMJ for youth soccer players following seven weeks of SIT or five weeks of HIIT. Sperlich et al. (2011) suggested that in order to enhance jumping performance, additional power specific training may be required. One study which did report an increase in CMJ following high intensity training which included jumping movements, was Ferrete et al. (2014). They noted an increase of 6.72% in jump height for prepubertal soccer players. This difference may also have been due to the longer duration (26 weeks) allowing greater opportunity for adaptations to manifest. It may also have been the inclusion of jumping activities in the training protocol which, like sprint starts, may need specific training to adapt, with technique playing an important role in jump height achieved (Hara et al., 2006).

### 5.5.4 Lactate kinetics

Following six weeks of cycle-based SIT, there was a large, meaningful change in lactate kinetics after supramaximal exercise in adolescent soccer players (Table 5.3). It was likely that there was an increase in extravascular release of lactate (A) and rate of lactate appearance ($k_1$). Rate of lactate clearance ($k_2$) increased significantly and correlated with increased aerobic capacity. Maximum blood lactate concentration ($[La^-]b_{max}$) was likely increased, although only by a small magnitude. Both time to blood lactate concentration maximum ($T[La^-]b_{max}$) and blood lactate turn point (TP) were slightly decreased following training. To date, no other study has investigated lactate kinetics in adolescent athletes in response to SIT. Endurance training has been shown to increase

rate of lactate exchange (Bergman et al., 1999), and in adults undertaking six weeks of two-minute duration intervals at 90 - 100% maximal aerobic speed (Gharbi, 2008), adaptations were similar to this study. As with changes in aerobic capacity, this suggests that it is not the duration of the interval that is the main driver for changes in whole body lactate kinetics. Increased concentrations of monocarboxylate transporter (MCT) 1 and 4 have been associated with changes in lactate kinetics (Juel et al., 2004; Thomas et al., 2005). Following eight weeks of high intensity single leg exercise, Pilegaard and colleagues (1999b) reported significant increase in MCT1 (76%) and smaller increases in MCT4 (32%). These elevated levels of MCTs allow greater rates of lactate exchange ($k_1$ and $k_2$) which are associated with increased exercise duration at high intensities (Messonnier et al., 2001, 2002). In this study, there was a significant positive correlation between $k_2$ and TTE ($r = 0.77$, $p = 0.04$) but not $k_1$ ($r = -0.64$, $p = 0.12$). Following 24 session of MICT, Messonier et al. (2006) also reported increases in TTE which were precipitated by an increase in $k_2$, but not $k_1$. In this study, there was also a significant negative correlation between $T[La^-]b_{max}$ and $k_1$ ($r = -0.85$, $p = 0.02$). While Bassett et al. (1991) reported no association between training status and $k_2$, they did report that in trained participants, $T[La^-]b_{max}$ was reduced compared to untrained individuals indicating faster efflux of lactate from muscles ($k_1$). The correlation between endurance adaptation and rate of lactate exchange suggests that endurance adaptation is related to changes in the ability to limit lactate accumulation, and instead process it for energy production. In adults, this improvement in lactate metabolism has been shown to result in a rightward shift in the blood lactate curve during incremental exercise and is associated with greater capillarisation of the skeletal muscle (Juel et al., 2004; Messonnier, 2002). In this study, CG $[La^-]b_{max}$ was unaltered following the control period (pre 11.92 ± 0.84mmol·l$^{-1}$; post 11.79 ± 3.06mmol·l$^{-1}$), whereas $[La^-]b_{max}$ in SG increased 16.9% (pre 13.16 ± 3.42mmol·l$^{-1}$; post 15.39 ± 1.67mmol·l$^{-1}$). Improvements in 20m sprint performance were associated with increases in both A and $[La^-]b_{max}$ which suggests that sprint speed is associated with the ability of skeletal muscle to generate ATP through anaerobic glycolysis. $[La^-]b_{max}$ following exercise has been shown to be higher in trained versus untrained adults (Juel et al., 2004),

and increased sprint performance is accompanied by an increase in the post-exercise muscle lactate concentration (Nevill et al., 2017). Sprint performance has also been moderately correlated to post-exercise peak lactate concentration in highly trained youth soccer players (Mujika et al., 2009). In young adults, bioenergetics during a WAnT demonstrate that approximately 50% of the total energy turnover comes from anaerobic glycolysis, and the proportion of anaerobic glycolysis utilised explains >80% of the variation in PP and AP production (Beneke et al., 2002). A relationship between maximal and average power produced during a WAnT and subsequent lactate concentration, has previously been identified in adolescents (Falgairette et al., 1991). This suggests that the ability to generate adenosine triphosphate (ATP) from anaerobic glycolysis is an important determinant of power production during a WAnT. In the current study, however, correlations between A and $[La^{-}]b_{max}$, and PP and AP were small. This may be due to the differences in participants fitness and training status. The adolescents in Falgairette's research (14 - 15 years) had appreciably less developed aerobic capacity and power production capability ($49 \pm 6ml \cdot kg^{-1} \cdot min^{-1}$; $10.8 \pm 1.4W \cdot kg^{-1}$) than those in the present study which may have affected the relationship between lactate kinetic factors and power.

### 5.5.5 Conclusion

In conclusion, this study demonstrates that for improving whole body lactate kinetics and performance indices in youth soccer players, cycle-based SIT is both time efficient and effective. It produces adaptations in a short training period by adding only 18 minutes per week to regular training schedules. Adaptations in maximal running speed, anaerobic power and endurance would appear to be determined primarily by the athlete's ability to produce ATP through anaerobic glycolysis, with endurance performance strongly influenced by the rate of lactate exchange and extent of lactate utilisation. Increased maximal speed may allow increased soccer performance through a greater number of positive outcomes from crucial match moments requiring high speed running (Little and Williams, 2005; Reilly, Bangsbo and Franks, 2000). Total

distance covered and distance covered through high intensity running, which both increase with enhanced aerobic capacity, have also been associated with match performance (Castagna et al., 2009; Al Haddad et al., 2015). The increases in both $\dot{V}O_{2peak}$ and TTE induced through SIT may therefore enhance match performance through increased high intensity work and distance covered (Mendez-Villanueva et al., 2012). The improvements in performance are linked to changes in lactate kinetics, and therefore it seems a necessity that soccer specific training should seek to enhance lactate kinetics to improve player performance. Failure to structure training to enhance lactate metabolism may lead to increased risk of injury due to fatigue during the later stages of a game through poor kinetic chain control (Lehnert et al., 2017). These adaptations are key for adolescent player development and the low impact nature of cycling minimises the risk of overuse injuries (Willoughby et al., 2016).

This study addressed the second aim of the thesis which was to determine whether the rate at which lactate was transported and utilised could be increased following high-intensity exercise through the use of SIT. To optimise training benefits, training stimulus should replicate the demands of match play (Cummins et al. 2013; Gabbett, 2010b; Harper, Carling and Kiely 2019; Smart et al., 2014; Vaz et al., 2014) and the results confirmed sport specific performance indices such as $\dot{V}O_{2peak}$, maximal running velocity and anaerobic power were enhanced through cycle-based SIT. The results also confirmed that cycle-based SIT will enhance the rate of lactate kinetics and this increase correlates to the increases in aerobic capacity. The study helps to further explain the relationship between lactate production and consumption following high-intensity exercise similar to that which may be experienced during invasion sports. With the first study of this thesis indicating lactate supplementation may help attenuate the decline in intermittent sprint performance seen during invasion sport match play to a small degree, and this study showing lactate metabolism is enhanced through cycle-based SIT, further investigation is required for the third aim of the thesis to determine if any ergogenic benefit of lactate supplementation can be enhanced following a period of SIT which increases the rate of lactate metabolism.

# Chapter 6

# Study 3

## 6.1 Abstract

**Introduction:** Invasion sports such as field hockey require players to perform multiple repetitions of maximal effort sprints, with the volume and intensity of these playing a critical role in determining level of match performance. The drop in sprint performance over the duration of match play is linked to reduced substrate availability leading to fatigue, and there a various ergogenic aids used to mitigate this. One such supplement is lactate which has been shown to possibly maintain high-intensity exercise performance with the effect being amplified as cardiorespiratory fitness increases.

**Methods:** Eleven amateur female field hockey players underwent baseline testing for CP, $\dot{V}O_{2peak}$, TTE, PP and RSA both with and without supplemented calcium lactate solution. This was followed by a control period where normal training and match play were maintained. Testing was then repeated before and after carrying out twice-weekly all-out effort cycle sprints consisting of 6 × 10 s sprint with 80 s recovery.

**Results:** There were no differences in performance between either condition at the baseline testing or following the control period ($p > 0.05$).

There was no change in any performance indicator following three-week control period ($p > 0.05$). Training elicited significant improvements in CP (Pre 2: 210 ± 2W; Post: 222 ± 25W, $p = 0.01$), $\dot{V}O_{2peak}$ (Pre 2: 40.27 ± 6.04 ml· $kg^{-1}$·$min^{-1}$; Post: 44.52 ± 4.11 ml· $kg^{-1}$·$min^{-1}$, $p = 0.02$), and TTE (Pre 2: 613 ± 99s; Post: 677 ± 118s, $p = 0.01$). There were no changes in either power at LT (Pre 2: 118 ± 21W; Post: 118 ± 18W, $p = 1.00$), or $[La^-]b$ at LT (Pre 2: 3.15 ± 0.64 mmol·$l^{-1}$; Post: 2.91 ± 0.47 mmol·$l^{-1}$, $p = 0.17$).

With the exception of TTE ($p = 0.003$) which saw a significant detrimental effect of lactate supplementation, there were no differences between condition during post-training testing ($p > 0.05$).

**Conclusions:** It is evident performing cycling-based SIT training twice-weekly in addition to the regular field hockey training sessions can help players develop aerobic and anaerobic capacity in a short period of time. It would appear supplementation of a 2% calcium lactate solution offers no ergogenic benefit for short-duration performance tests and may in fact have a detrimental effect on endurance capacity.

## 6.2 Introduction

### 6.2.1 Demands of the Sport

For female athletes, work rate during field hockey is similar to that seen in soccer and rugby union (Hodun et al., 2016) and, like those sports, field hockey is for the most part, low to moderate intensity. Walking and jogging account for ~85 - 88% of playing time with the remaining 12 - 15% consisting of high intensity running and sprinting greater than ~4m·s$^{-1}$ (Harry and Booyen, 2020; McGuiness et al., 2019a). It is these intermittent periods of high intensity work both in terms of volume and duration, that are the discriminating factor between elite level performance and that of sub-elite invasion sport athletes (Gabbett, 2010a; Mohr et al., 2008; Portillo et al., 2014).

There are very few studies (McGuinness et al., 2019a; McMahon and Kennedy 2019; Morencos et al., 2019; Sell and Ledesm, 2016) reporting the physiological demands of female field hockey since the International Hockey Federation amended match format from two, 35-minute halves to four, 15-minute quarters in 2015. Comparing data from elite female players collected using a global positioning system (GPS), McMahon and Kennedy (2019) analysed differences between 12 matches played in 2014 under the old format, and 13 matches in 2015 played in the new format. They concluded that, in general, match intensity has increased for elite female field hockey players, specifically through increased relative distance covered, increased high-speed running, and decreased volume of low-speed running. Prior to the change in match format, elite female players spent on average 55 minutes in play, and in that time covered approximately 6000m (Macutkiewicz and Sunderland, 2011; Vescovi and Frayne, 2015). Mean heart rate (HR) throughout match play was ~168 b·min$^{-1}$ with the majority of match play eliciting HR of more than 75% maximum heart rate ($HR_{max}$) (Sunderland et al., 2006), and peak HR reaching 192 ± 5 b·min$^{-1}$ (Vescovi, 2016). Following the change in format, time in play is ~15 minutes less but players cover about 5250m which equates to a higher relative work rate (McGuinness et al., 2019a; Morencos et al., 2019). Mean and peak HR are also reported as being higher in the new format with values of 174.1 ± 13.4 b·min$^{-1}$ and 198 ± 4 b·min$^{-1}$ respectively (McGuinness et al., 2019a; Sell

and Ledesma, 2016). In terms of high intensity play, McGuinness et al. (2019a) reported 11.6% of match play as high intensity (running speed >4.44m·s$^{-1}$), whereas previously reported data for high intensity activity profiles in elite women's hockey ranged from 2% (running speed >5.0 m·s$^{-1}$) (Gabbett, 2010a), to 6.4% (running speed >4.19m·s$^{-1}$) (Macutkiewicz and Sunderland, 2011), and 11.8% (running speed >4.44 m·s$^{-1}$ (Vescovi and Frayne, 2015). Morencos et al. (2019) found that players performed, on average, 64 sprints (running speed >5.83m·s$^{-1}$), while in the old format of play Vescovi and colleagues (2014, 2015) reported number of sprints as 7 - 9 sprints (running speed >5.56m·s$^{-1}$). It had also previously been reported that there was a significant ($p < 0.01$) drop in high intensity running between halves (MacLeod, Bussell and Sunderland, 2007), but both McGuinness et al. (2019a) and Morencos et al. (2019) reported increased high intensity activity in the final quarter relative to total distance covered under the new format.

Both Sell and Ledesma (2016) and McGuinness and colleagues (2019a), reported mean HR throughout match play as ≥85% $HR_{max}$ which equates to ~70% maximal oxygen uptake ($\dot{V}O_{2max}$) (Bangsbo, Mohr and Krustrup, 2006). Maximal lactate steady state (MLSS) is associated with a mean blood lactate concentration ([$La^-$]b) of 4 mmol·l$^{-1}$ (Billat et al., 2003) and an oxygen uptake ($\dot{V}O_2$) equivalent to 70 - 90% $\dot{V}O_{2max}$ (Jones and Vanhatalo, 2017), suggesting match play is likely of an intensity ≥ MLSS. Heart rate in non-elite players prior to format change has been shown to be similar to that of elite players (mean value of 171 ± 6 b·min$^{-1}$ (Kusnanik, Rahayu and Rattray, 2018) versus 172 ± 8 b·min$^{-1}$ (Vescovi and Klas, 2018). In the amateur female players, this HR was accompanied by [La-]b of 5.2 ± 3.1 mmmol·l$^{-1}$ showing that the mean intensity is greater than MLSS and athletes are relying on anaerobic glycolysis during periods of high intensity activity. With elite players exhibiting higher mean HR following format change, it is likely amateur players are also experiencing [La-]b greater than 4 mmol·l$^{-1}$. This indicates that a field hockey player's ability to metabolise lactate may be an important element in sporting performance. These data, along with the conclusions of McMahon and Kennedy (2019), suggest that the new, four quarter format has led to faster paced, more metabolically demanding match play requiring players to demonstrate increased

levels of aerobic and anaerobic conditioning. For strength and conditioning programmes to be effective and efficient for female field hockey players, it is important that coaches tailor them to the demands of female field hockey (Hodun et al., 2016; McMahon and Kennedy, 2019), and with these recent data evidencing a change in match demands, training must be adapted to accommodate this. For training to be successful, it must address both the aerobic capacity of the athlete, which aids with recovery between high intensity bouts, and the anaerobic system which is predominant during sprinting (Gabbett, 2010a). Elferink-Gemser et al. (2006) reported elite level female youth players demonstrated higher aerobic capacity than non-elite females, and $\dot{V}O_{2max}$ correlates strongly ($r = 0.713$, $p < 0.01$) with exercise intensity at a heart rate approximately equivalent to mean HR during match play (Sharma and Kailashiya, 2017).

### 6.2.2 Training

Small sided games

Across most invasion sports, it is now common practice to use small sided games (SSG) as a conditioning tool for players (Gabbett and Mulvey, 2008; Polglaze et al., 2015) but results have been mixed. While some studies have found that SSG are effective at developing or maintaining the fitness characteristics desirable in invasion sports such as soccer (Reilly and White, 2005), others have found it a less reliable modality of training (Gabbett and Mulvey, 2008). While SSG may be an effective tool for training skills and situational awareness in field hockey (Gabbett, 2010a; Timmerman, Savelsbergh and Farrow, 2019), the major limitation of this training paradigm is the inability to overload the specific, outcome deciding activities, such as high-intensity running, sprinting and accelerations (Casamichana, Castellano and Castagna, 2012; Gabbett and Mulvey, 2008; Gabbett, Jenkins and Abernethy, 2012; Tyndel, 2018). This was true for both elite male (Johnston et al., 2004) and elite female (Gabbett, 2010a) field hockey players where SSG proved an insufficient stimulus. In a study of elite female players, Gabbett (2010a) analysed GPS data from 19 SSG-based training sessions and 32 Australian

Hockey league matches. Low to moderate intensity running (0 - 5m·s$^{-1}$) was higher in SSG than match play (≈99% versus ≈97%), high intensity running (>5m·s$^{-1}$) volume was significantly lower (≈1% versus ≈3%), and there was no evidence of maximal speed work (>7m·s$^{-1}$) during SSG. Gabbett concluded that SSG did not reflect either the volume of high intensity work or the repeated high intensity bouts seen in match play. Given that these matches were played before the format change, which has led to increased intensity of play (McMahon and Kennedy, 2019), it is probable that SSG will be insufficient to meet the training demands of the modern game structure. A second main issue with the use of SSG is the variability in work rate and physiological responses seen between players (Tyndel, 2018). Although no studies for field hockey have investigating this issue, Dellal et al. (2011), Ade, Harley and Bradley (2014), and Davies et al. (2013), have all reported issues for invasion sport athletes. These include high levels of variation between players for repeated sprints and agility (Ade, Harley and Bradley, 2014; Davies et al., 2013), and Dellal et al., (2011) reported inter-subject coefficient of variation (CV) was 100% greater (CV = 11.8% versus 5.9%) for heart rate response when comparing SSG and intermittent running. Additionally, the reduced playing areas used in SSG can lead to sharper cutting angles and more frequent change of direction, which poses a greater risk of joint injuries to athletes (Schreurs, Benjainse and Lemmink, 2017).

High intensity training

To meet the requirement for both a highly developed aerobic capacity and the ability to repeatedly perform high intensity bouts of exercise, Gabbett (2010b) states that a training modality which also stresses the anaerobic pathways is required. Adaptations to sprint interval training (SIT) vary depending on the duration of the recovery period in relation to the exercise bout. Shorter rest periods (1:3 work to rest) elicit greater aerobic adaptation and longer rests (1:12 work to rest) develop anaerobic characteristics more (Kavaliauskas, Aspe and Babraj, 2015). Short rests periods do not allow complete phosphocreatine (PCr) regeneration placing a greater demand on aerobic metabolism in the later sprint repetitions (Gaitanos et al., 1993). Longer rest periods (1:8 - 1:12 work to rest)

enhance sprint performance (Connolly, Brennan and Lauzon, 2003; Spierer et al., 2004), through greater PCr regeneration and this is highly correlated (r = 0.84) with power output in subsequent sprints (Bogdanis et al., 1996). Smilios et al. (2017) reported that if rest period is too long, there is a drop in HR which has a detrimental effect on training adaptations. A work to rest ratio which develops both aerobic and anaerobic elements would be more beneficial to invasion sport athletes, and a 1:8 work to rest ratio has been shown to do this (Kavaliauskas, Aspe and Babraj, 2015). SIT protocols using this ratio have elicited significant improvements in peak power output (PP), time to exhaustion (TTE), critical power (CP), and peak oxygen uptake ($\dot{V}O_{2peak}$) (Burgomaster et al., 2005; Kavaliauskas, Aspe and Babraj, 2015; Lloyd Jones, Morris and Jakeman, 2017; Yamagishi and Babraj, 2017). Most high intensity bouts during invasion sports last less than 10s (Morton, 1978) making short duration sprints appropriate during training. It is assumed that, with SIT protocols involving sprints as short as six seconds producing similar results to longer exercise bouts, metabolic demand in the early section of the sprint is the driver for physiological adaptation (Jakeman, Adamson and Babraj, 2012). Yamagishi and Babraj (2017) (15s versus 30s sprints) and Hazell et al. (2010) (10s versus 30s sprints) both concluded that the shorter sprints were just as effective at improving fitness characteristics as 30s bouts. The magnitude of response to exercise is strongly correlated to the magnitude of the stimulation, with supramaximal bouts of exercise generating a greater training stimulus than moderate intensity continuous training (MICT) (Egan et al., 2010; Nordsborg et al., 2010). There are multiple physiological and metabolic adaptations to SIT including upregulation of proteins such as peroxisome proliferator-activated receptor γ coactivator 1α (PGC-1α) which is a driver of mitochondrial biogenesis (Gibala et al., 2012; Valero-Grinan, 2014), and glucose transporter-4 (GLUT-4) which facilitates skeletal muscle glucose uptake (Little et al., 2010). These adaptations translate to increased levels of physical performance with research showing increases in characteristics desirable in field hockey such as $\dot{V}O_{2peak}$ (Sheykhlouvand et al., 2018), TTE and CP (Kavaliauskas, Steer and Babraj, 2017), acceleration (Serpiello et al., 2011), and repeated sprint ability (RSA) (Taylor et al., 2015; Viaño-Santasmarinas et al., 2018). In adults, HIT (30 - 60s

cycle bouts with 2 mins recovery) has also been shown to increase the skeletal muscle lactate capacity, with increased lactate transporter content in skeletal muscle (Pilegaard et al., 1999), and increased lactate dehydrogenase (LDH) activity in Type II skeletal muscle fibres (6 x 2.5 min run intervals with 90s recovery) (Kohn et al., 2010). Together, these would allow invasion sport athletes to either transport accumulated lactate away from the working muscle for consumption in another metabolic site (Brooks, 2002b), or metabolise it within the mitochondria of the working muscle cell (Brooks, 2002b). The benefit of this is twofold. Firstly, a readily available metabolic substrate during low to moderate intensity recovery periods, reduces reliance on blood glucose and muscle glycogen and preserves these metabolites for further high intensity bouts (Brouns et al., 1995; Fahey et al., 1991). Secondly, if $[La^-]b$ becomes significantly elevated, the ability to rapidly produce adenosine triphosphate (ATP) through glycolysis can be suppressed leading to reduced sprint performance (Bangsbo et al., 1992; Choi et al., 2002).

Interval training can be used to simulate the repeated high intensity efforts seen during invasion sports like field hockey, but to date few studies have investigated the benefits for female field hockey players. Examining changes in $\dot{V}O_{2peak}$ in 14 non-elite female hockey players over a four week training programme, Funch et al. (2017) concluded that both high intensity running and 'Tabata-style' high intensity interval training (HIIT), were effective training modalities to improve $\dot{V}O_{2peak}$. The running protocol consisted of three sessions per week of 30 minutes running at 75 - 85% of age predicted $HR_{max}$ and the HIIT was performed at the same frequency and required four minutes of 20s maximal effort calisthenics separated by 10s recovery. Following the 12 training sessions, both training modalities elicited significant improvement in $\dot{V}O_{2peak}$ (running = 6.2%; HIIT = 6.1%), but HIIT required 20% less time commitment than the running protocol. Jones, Hamilton and Cooper (2015), investigated the effect of HIIT on muscle oxygenation and endurance capacity in 25 elite female field hockey players of Olympic standard. The SIT protocol involved six weeks of one session per week in addition to normal training regime and was performed on a Wattbike Pro (air brake level 3, magnetic brake setting 1). Participants were required to perform 8 (week 1 & 2), 10 (week 3 & 4) or 12

(week 5 & 6) repetitions of a 60s sprint (450 ± 106 W) separated by 75s active recovery (45 ± 10W). Endurance capacity was evaluated using the 30-15 intermittent fitness test and final running speed performance was improved significantly in the training group following SIT (pre 5.34 ± 0.14 $m \cdot s^{-1}$ versus post 5.50 ± 0.14 $m \cdot s^{-1}$). The authors suggest that this improvement may be related to the increased rate at which skeletal muscle was able to extract oxygen from the blood. With time efficiency being a desirable trait in training (Funch et al., 2017), and athletes having limited contact time with strength and conditioning coaches (Sell and Ledesma, 2016), it may be beneficial for invasion sport athletes to utilise a shorter SIT protocol to reduce overall duration, and also the number and duration of repetitions.

### 6.2.3 Lactate Supplementation

Earlier findings in this thesis and prior research have indicated that lactate may be beneficial to physical performance when consumed as an ergogenic aid (Table 2.1). In particular, ingested lactate is metabolised rapidly and extensively during high intensity exercise in well trained individuals ($\dot{V}O_{2peak}$ = 67.4 ± 3.2 $ml \cdot kg^{-1} \cdot min^{-1}$), leading to substantial increases in performance (Azevedo et al., 2007; Morris et al., 2011; van Montfoort et al., 2004). These benefits are less pronounced in individuals with lower aerobic capacity (Peveler and Palmer, 2012; Russ, Schifino and Leong, 2019), or when exercise is less intense (Swensen et al., 1994). The increase in performance seen in the exhaustive protocols may be attributable to preservation of muscle glycogen for use in the exhaustive phase of the exercise through preferential metabolism of the supplemented lactate solutions in the earlier stages (Bryner et al., 1998; Fahey et al., 1991; Swensen et al., 1994). One explanation as to why lactate ingestion may not lead to enhanced performance is the training status of the individual. The enzyme LDH is present in the mitochondria of skeletal muscle (Brooks et al., 1999b; Dubouchaud et al., 2000), and facilitates direct metabolism of lactate within the mitochondria (Brooks, 2002b). Untrained individuals have lower concentrations of LDH and may not respond to exogenous lactate supplementation (Russ, Schifino and Leong, 2019). Increased LDH concentrations following high intensity training (Kohn et al., 2010) may therefore

increase the ergogenic effect of supplemented lactate by allowing it to be used more fully as a metabolic substrate during submaximal exercise.
Supplemented lactate may also act in a buffering capacity by elevating blood bicarbonate concentrations and reducing intramuscular hydrogen ion ($H^+$) concentrations which have been associated with a reduction in contractile efficiency during maximal exercise (Morris et al., 2011; Oliveira et al., 2017; van Montfoort et al., 2004).

### 6.2.4 Aim and hypothesis

The aim of the current study was to determine if supplementing a 2% calcium lactate solution improved performance measures compared to water in adult female field hockey players. Additionally, it sought to determine if a three week, twice-weekly cycle-based sprint protocol, which would improve lactate kinetics, had an effect on the performance differences between and within conditions. It was hypothesised that there would be a small improvement in performance through calcium lactate supplementation, and this difference would be amplified following the training.

## 6.3 Methods

### 6.3.1 Participants

Eleven healthy, active female participants (Table 6.1) were recruited from an amateur women's hockey club. All participants were sub-elite hockey players participating in one training session and one match per week. The Abertay University Ethics Committee approved the study and all testing was carried out in line with the Declaration of Helsinki (www.wma.net, 2013).

***Table 6.1 Participant characteristics prior to, and following SIT.***

| | Pre training | Post training |
|---|---|---|
| Age (y) | 19 ± 1 | 19 ± 1 |
| Height (cm) | 166 ± 4 | 166 ± 4 |
| Mass (kg) | 65.7 ± 10.5 | 66.2 ± 10.2 |
| Fat Free Mass (kg) | 47.7 ± 4.4 | 47.2 ± 4.1 |
| Body Fat (%) | 26.5 ± 8.5 | 27.8 ± 8.2 |
| $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$) | 40.27 ± 6.04 | 44.52 ± 4.11 |

### 6.3.2 Procedure

Participants undertook each test under two conditions, consuming either 500ml flavoured lactate solution containing 2% weight by volume calcium lactate (L), or 500ml flavoured water solution (N) 20 minutes before commencing the tests. The order in which they were performed was randomized and double blinded. Familiarisation trials were performed for each test in order for participants to become oriented with all testing procedures and training devices.

Session 1

Participants were invited to the laboratory where they were given the opportunity to ask any questions they may have had regarding the study. Height was measured (SECA 213 stadiometer, United Kingdom) along with body mass, and body fat percentage using calibrated, bioelectrical impedance analysis (BIA) (SC-330ST Tanita Body Composition Analyser, Tanita Europe BV, Amsterdam, Netherlands), the results of which were recorded (Table 6.1). Saddle height was adjusted for each participant ensuring the knee remained slightly flexed at the completion of the power stroke (approximately 170 - 175° at extension). Toe clips were used to ensure that the participant's feet remained firmly in place and in contact with the pedals throughout the tests. Bike settings were noted for all subsequent sessions.

This session tested critical power (CP) using a Wattbike Pro cycle ergometer (Wattbike Ltd, Nottingham, United Kingdom). Participants cycled at 55W for three minutes to warm up. Participants then began the three-minute aerobic test on the Wattbike Pro which requires them to pedal maximally for three minutes

with no feedback in relation to time elapsed to avoid pacing tactics. Verbal encouragement was provided throughout. Heart rate and respiratory rate were recorded continuously using a BioHarness 2 (Xypher technologies, Annapolis, USA) with gas analysis (Metalyzer®3B gas analyser, Cortex, Leipzig, Germany).

The CP test was conducted before and after the control period, and again after the training period. It was determined using the three-minute aerobic test on the Wattbike Pro which required participants to pedal maximally for three minutes. Participants cycled at 60W for three minutes to warm up, then after a one-minute rest, began the test with no feedback in relation to time elapsed, distance covered, or speed to avoid pacing tactics. Verbal encouragement was provided throughout to ensure maximal effort. Resistance is automatically determined by the Wattbike Pro based on the participant's sex, age and body mass.

Blood lactate concentration was determined via fingertip blood samples (Lactate pro, Arkray Inc., Kyoto, Japan). Blood lactate concentration was measured prior to the CP test and then 0, 1, 5, 10, 15, and 20mins after the CP test. The skin was punctured using an Accu-check single use lancet (Roche Diagnostics, UK) and pressure applied to the finger to draw the blood. The initial drop was discarded, and the second drop was taken for analysis. Blood lactate kinetics were modelled using the same three-pool model as the previous study.

Session 2

The Wattbike Pro Max Ramp Test starts at a pre-programmed initial power output of 55W, with an additional 15W increase every minute until volitional exhaustion, or the participants could not maintain power output despite strong verbal encouragement. Gas analysis (Metalyzer®3B gas analyser, Cortex, Leipzig, Germany) was used to measure $\dot{V}O_{2peak}$. Exercise duration at exhaustion was recorded to the nearest second and defined as TTE. $\dot{V}O_{2peak}$ was taken as the highest 15s average over the incremental test.

This session tested aerobic capacity and blood lactate threshold of the participants. The test started at an initial power output of 55W, with an additional 15W increase every minute until volitional exhaustion or the participants could not maintain power output despite strong verbal encouragement. Heart rate and respiratory rate were recorded throughout using a BioHarness 2 (Xypher technologies, Annapolis, USA) with gas analysis (Metalyzer®3B gas analyser, Cortex, Leipzig, Germany) being used to calculate $\dot{V}O_{2peak}$. Blood lactate levels were recorded via finger prick sampling using a single use lancet and a spot of blood released from the finger for analysis (lactate pro 2, Arkay Ltd, Japan). The first drop of blood was wiped off to avoid skin surface contamination and the second drop then used for analysis of lactate levels. Samples were taken every 60 seconds throughout the test until LT was reached. Exercise duration at exhaustion was recorded to the nearest second and defined as time to exhaustion. $\dot{V}O_{2peak}$ was taken as the highest 15s average over the incremental test. Lactate threshold was defined as an increase of 1mmol·l$^{-1}$ in [La$^{-}$]b from baseline levels.

Session 3

Peak power and repeated sprint ability were tested using the Wattbike Pro. Following a three-minute warm-up at 55W, participants were required to cycle maximally for six bouts of ten seconds interspersed with ten seconds recovery. PP was taken from the first sprint and total work (TW) was calculated over the six sprints.

Pre 2 and post tests

During the 21-day control period participants maintained their normal routine before repeating sessions 1, 2 and 3 under each condition to determine any changes in $\dot{V}O_{2peak}$, repeated sprint ability or critical power.

Following the 21-day SIT training period testing was repeated along with reassessment of anthropometric data. Post-training testing commenced a minimum of 48hrs from final training and was completed within ten days of the last training session. The test/drink combination was conducted in the same order for each participant as during the Pre 1 tests.

### 6.3.3 Training

Training was conducted on a Wattbike Pro. All participants warmed up for three minutes at 55W followed by a one-minute rest period. To provide a training overload from test intensity, air brake resistance was increased to level 4 for all participants. During the SIT protocol, participants remained seated in the saddle for the duration of the session and maintained a standard overhand handlebar grip. Six bouts of maximal effort cycling were performed consisting of ten seconds all-out cycling followed by a passive recovery period of 80 seconds. The training protocol was completed six times over a period of three weeks.

### 6.3.4 Data Analysis

All data are presented as mean ± standard deviation (SD). The bi-exponential model was fitted using QtiPlot to generate values for A, $k_1$ and $k_2$. Statistical analysis of the remaining data was performed using IBM® SPSS® Version 28.0 (IBM Corp., Armonk, N. Y., USA) and Microsoft Excel (Microsoft Corporation, Redmond, Washington, USA).). Repeated measures analysis of variance (ANOVA) was performed to determine effect of time, condition, and time x condition interaction. Assumptions of homogeneity of variance were tested using Mauchly's sphericity test and where this was violated, the Greenhouse-Geisser estimate value was used to adjust degrees of freedom unless this estimate was > 0.75 in which case the less conservative Huynh-Feldt estimate would be used. Partial eta squared ($\eta_p^2$) was calculated to determine effect size (ES) along with 95% confidence intervals (CI). Effect size was defined as; small = 0.01 - 0.05, medium = 0.06 - 0.13, and large ≥ 0.14 (Cohen, 1988). Pearson's correlations were used to assess the relationships between lactate kinetic parameters and performance measures. Statistical significance for group means difference was set a priori at $p \leq 0.05$.

## 6.4 Results

### 6.4.1 Lactate supplementation prior to training

Critical power

Prior to training, there was no main effect of time on critical power, and no significant interaction between condition and time with ES being small and medium respectively (F (1,10) = 0.32, p = 0.59, $\eta_P^2$ = 0.03 [95%CI: 0.00 to 0.34]; F (1,10) = 0.81, p = 0.39, $\eta_P^2$ = 0.08 [95%CI: 0.00 to 0.41]; Table 6.2). There was no main effect of condition on critical power but a large ES (F (1,10) = 2.21, p = 0.17, $\eta_P^2$ = 0.18 [95%CI: 0.00 to 0.51]) (Table 6.2).

$\dot{V}O_{2peak}$

There was no main effect for condition or time, and no condition x time interaction for $\dot{V}O_{2peak}$ prior to training (F (1,10) = 0.01, p = 0.93, $\eta_P^2$ < 0.01 [95%CI: 0.00 to 0.16]; F (1,10) = 3.97, p = 0.07, $\eta_P^2$ = 0.28 [95%CI: 0.00 to 0.58]; F (1,10) = 0.56, p = 0.47, $\eta_P^2$ = 0.05 [95%CI: 0.00 to 0.38]), with small ES for condition and condition x time interaction, and ES for time was large. There was no difference in magnitude between conditions for $\dot{V}O_{2peak}$ at Pre 1 or Pre 2 (Table 6.2).

Time to exhaustion

There was no main effect for condition or time, and no condition x time interaction for TTE prior to training (F (1,10) = 2.91, p = 0.12, $\eta_P^2$ = 0.23 [95%CI: 0.00 to 0.54]; F (1,10) = 1.45, p = 0.26, $\eta_P^2$ = 0.13 [95%CI: 0.00 to 0.46]; F (1,10) = 0.001, p = 0.97, $\eta_P^2$ < 0.01 [95%CI: 0.00 to 0.00]). ES was large for condition and time, and negligible for condition x time interaction (Table 6.2).

Lactate threshold

Power at lactate threshold (LT) showed no main effect for condition or time, and no condition x time interaction (F (1,10) = 0.30, p = 0.60, $\eta_P^2$ = 0.03 [95%CI: 0.00 to 0.34]; F (1,10) = 0.58, p = 0.47, $\eta_P^2$ = 0.06 [95%CI: 0.00 to 0.38]; F (1,10) = 0.06, p = 0.82, $\eta_P^2$ < 0.01 [95%CI: 0.00 to 0.26]) prior to training. There was no

significant difference between conditions at either test point prior to training (Pre 1 d = -0.13, p = 0.75; Pre 2 d = -0.18, p = 0.54) (Table 6.2).

There was no main effect for condition or time, and no condition x time interaction for $[La^-]b$ at LT prior to training (F (1,10) = 0.26, p = 0.62, $\eta_P^2$ = 0.03 [95%CI: 0.00 to 0.33]; F (1,10) = 0.004, p = 0.95, $\eta_P^2$ < 0.00 [95%CI: 0.00 to 0.10]; F (1,10) = 0.74, p = 0.41, $\eta_P^2$ < 0.07 [95%CI: 0.00 to 0.40]), with a small ES for condition and time but medium for condition by time interaction. There was no significant difference between conditions at either test point prior to training (Pre 1 d = -0.34, p = 0.47; Pre 2 d = 0.09, p = 0.79) (Table 6.2).

Peak power

Prior to training, there was no main effect of condition (F (1,10) < 0.01, p = 0.97, $\eta_P^2$ < 0.01 [95%CI: 0.00 to 0.15]) or time (F (1,10) = 2.26, p = 0.16, $\eta_P^2$ = 0.16 [95%CI: 0.00 to 0.51]) on PP, and no condition x time interaction (F (1,10) < 0.01, p = 0.99, $\eta_P^2$ < 0.01 [95%CI: 0.00 to 0.15]). Effect size was negligible for condition and condition x time but large for time. There was no significant difference in magnitude of PP between conditions, and effect size was trivial at both test points prior to training (Pre 1 d < 0.01, p = 0.98; Pre 2 d = -0.01, p = 0.98) (Table 6.2).

Total work

There was no significant effect of condition on TW prior to training and a large effect size (F (1,10) = 7.25, p = 0.12, $\eta_P^2$ = 0.42 [95%CI: 0.00 to 0.67]). There was also no main effect for time or condition x time interaction, and ES was small and large respectively (F (1,10) = 0.51, p = 0.49, $\eta_P^2$ = 0.05 [95%CI: 0.00 to 0.37]); F (1,10) = 2.47, p = 0.15, $\eta_P^2$ < 0.20 [95%CI: 0.00 to 0.52]) (Table 6.2).

***Table 6.2 Performance without (N) and with (L) lactate supplementation prior to SIT. Values shown are group mean ± SD.***

| Variable | Pre 1 | | | | Pre 2 | | | |
|---|---|---|---|---|---|---|---|---|
| | N | L | p value | Standardised differences, ES ± 95% CI | N | L | p value | Standardised differences, ES ± 95% CI |
| CP | 206 ± 22 | 204 ± 20 | 0.30 | -0.08 ± 0.34 | 210 ± 27 | 203 ± 16 | 0.25 | -0.34 ± 4.82 |
| $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$) | 37.65 ± 5.12 | 38.16 ± 4.93 | 0.71 | 0.10 ± 0.08 | 40.27 ± 6.04 | 39.62 ± 5.39 | 0.44 | -0.11 ± 0.27 |
| TTE (s) | 596 ± 51 | 616 ± 74 | 0.29 | 0.33 ± 0.15 | 613 ± 99 | 631 ± 79 | 0.18 | 0.21 ± 8.42 |
| $LT_{power}$ (W) | 121 ± 7 | 119 ± 22 | 0.75 | -0.13 ± 6.25 | 118 ± 20 | 115 ± 15 | 0.54 | -0.18 ± 2.05 |
| $LT_{[La^-]b}$ ($mmol \cdot l^{-1}$) | 3.29 ± 0.69 | 3.10 ± 0.38 | 0.47 | -0.34 ± 0.13 | 3.15 ± 0.64 | 3.20 ± 0.41 | 0.79 | 0.09 ± 0.10 |
| PP (W) | 712 ± 101 | 712 ± 97 | 0.98 | 0.00 ± .36 | 733 ± 118 | 733 ± 84 | 0.98 | -0.01 ± 14.20 |
| $RSA_{total}$ (kJ) | 23.24 ± 1.65 | 24.10 ± 2.09 | 0.06 | 0.46 ± 0.19 | 23.50 ± 2.38 | 23.44 ± 1.95 | 0.81 | -0.03 ± 0.18 |

***Abbreviations: CP is critical power, $VO_{2peak}$ is the highest rate of oxygen uptake recorded, TTE is time to exhaustion, $LT_{power}$ is power output at lactate threshold, $LT_{[La^-]b}$ is blood lactate concentration at lactate threshold, PP is highest power reached during repeated sprints, $RSA_{total}$ indicates total work done over six sprints.***

Lactate

There was no main effect for condition or time, and no condition x time interaction (F (1,10) = 0.24, p = 0.63, $\eta_P^2$ = 0.02 [95%CI: 0.00 to 0.33]; F (1,10) = 4.60, p = 0.06, $\eta_P^2$ = 0.32 [95%CI: 0.00 to 0.60]; F (1,10) = 0.67, p = 0.43, $\eta_P^2$ = 0.06 [95%CI: 0.00 to 0.39]) for extravascular release of lactate from exercise metabolism (A), and ES was small, large and medium respectively (Table 6.3). For the rate of lactate accumulation ($k_1$) the was no main effect for condition or time, and no condition x time interaction (F (1,10) = 0.26, p = 0.14, $\eta_P^2$ = 0.21 [95%CI: 0.00 to 0.33]; F (1,10) = 0.15, p = 0.70, $\eta_P^2$ = 0.02 [95%CI: 0.00 to 0.30]; F (1,10) = 0.04, p = 0.85, $\eta_P^2$ < 0.01 [95%CI: 0.00 to 0.24]). ES for condition was large, but small and negligible for time and condition x time interaction (Table 6.3). Rate of lactate clearance ($k_2$) showed no main effect prior to training for condition or time, or for condition x time interaction, and ES was small to negligible for each (F (1,10) < 0.00, p = 1.00, $\eta_P^2$ < 0.00 [95%CI: 0.00 to 0.00]; F (1,10) = 0.11, p = 0.75, $\eta_P^2$ = 0.01 [95%CI: 0.00 to 0.29]; F (1,10) = 0.20, p = 0.67, $\eta_P^2$ = 0.02 [95%CI: 0.00 to 0.32]) (Table 6.3). There was no main effect for condition and no condition x time interaction on maximum lactate concentration ($[La^-]b_{max}$) (F (1,10) = 0.24, p = 0.63, $\eta_P^2$ = 0.02 [95%CI: 0.00 to 0.33]; F (1,10) = 4.60, p = 0.06, $\eta_P^2$ = 0.32 [95%CI: 0.00 to 0.60]). ES for condition was small, and ES for condition x time interaction was large (Table 6.3). There was no main effect for condition or time, and no condition x time interaction (F (1,10) = 1.56, p = 0.25, $\eta_P^2$ = 0.18 [95%CI: 0.00 to 0.47]; F (1,10) = 0.33, p = 0.58, $\eta_P^2$ = 0.05 [95%CI: 0.00 to 0.35]; F (1,10) = 0.28, p = 0.61, $\eta_P^2$ = 0.04 [95%CI: 0.00 to 0.34]) for time to maximum lactate concentration ($T[La^-]b_{max}$). ES was large for condition, and small for time and condition x time interaction. Turn point (TP) showed no main effect for condition or time, and no condition x time interaction (F (1,10) = 1.56, p = 0.25, $\eta_P^2$ = 0.18 [95%CI: 0.00 to 0.47]; F (1,10) = 0.33, p = 0.58, $\eta_P^2$ = 0.05 [95%CI: 0.00 to 0.35]; F (1,10) = 0.28, p = 0.61, $\eta_P^2$ = 0.04 [95%CI: 0.00 to 0.36]). ES was large for condition, and small for time and condition x time interaction.

There was no significant difference between conditions for A, $k_1$, $k_2$, $[La^-]b_{max}$, $T[La^-]b_{max}$ or TP prior to training (Table 6.3).

***Table 6.3 Lactate kinetic variables for N and L following 3-minute critical power test prior to training. * denotes a significant difference between conditions (p ≤ 0.05).***

| Variable | Pre 1 | | | | Pre 2 | | | |
|---|---|---|---|---|---|---|---|---|
| | N | L | p value | Standardised differences, ES ± 95% CI | N | L | p value | Standardised differences, ES ± 95% CI |
| A ($mmol \cdot l^{-1}$) | 21.0 ± 2.7 | 22.2 ± 4.0 | 0.37 | 0.35 ± 0.51 | 19.48 ± 4.89 | 19.34 ± 4.58 | 0.92 | -0.03 ± 0.13 |
| $k_1$ ($mmol \cdot min^{-1}$) | 0.46 ± 0.15 | 0.37 ± 0.14 | 0.16 | -0.62 ± 0.004 | 0.47 ± 0.15 | 0.40 ± 0.18 | 0.32 | -0.42 ± 0.01 |
| $k_2$ ($mmol \cdot min^{-1}$) | 0.08 ± 0.02 | 0.09 ± 0.03 | 0.71 | 0.39 ± 0.004 | 0.09 ± 0.04 | 0.09 ± 0.06 | 0.77 | 0.00 ± 0.01 |
| $[La^-]b_{max}$ ($mmol \cdot l^{-1}$) | 15.66 ± 2.29 | 16.42 ± 2.48 | 0.47 | 0.32 ± 0.08 | 14.52 ± 2.05 | 14.28 ± 2.32 | 0.36 | -0.11 ± 0.11 |
| $T[La^-]b_{max}$ (min) | 4.81 ± 1.02 | 5.50 ± 1.43 | 0.31 | 0.55 ± 0.17 | 4.69 ± 1.06 | 5.98 ± 3.17 | 0.25 | 0.53 ± 0.88 |
| TP (min) | 9.63 ± 2.05 | 10.99 ± 2.86 | 0.32 | 0.54 ± 0.34 | 9.38 ± 2.12 | 11.96 ± 6.33 | 0.25 | 0.53 ± 1.76 |

***Abbreviations: A is extravascular release of lactate, $k_1$ is rate of lactate accumulation, $k_2$ is rate of lactate clearance, $[La^-]b_{max}$ is maximal blood lactate concentration, $T[La^-]b_{max}$ is time to maximal blood lactate concentration, and TP is turn point.***

### 6.4.2 Effect of sprint interval training

$\dot{V}O_{2peak}$

A main effect was evident for time on $\dot{V}O_{2peak}$ with a large effect size ($F(2,20) = 7.88$, $p = 0.003$, $\eta_P^2 = 0.44$ [95%CI: 0.08 to 0.62]) (Figure 6.4). Following six sessions of SIT, there was a significant increase in $\dot{V}O_{2peak}$ and a medium ES for time ($d = 0.82$, $p = 0.02$).

Time to exhaustion

A significant main effect was present for time with a large effect size ($F(2,20) = 7.78$, $p = 0.003$, $\eta_P^2 = 0.44$ [95%CI: 0.07 to 0.62]; Figure 6.4). Following six sessions of SIT, there was a significant increase in TTE and a small ES for training ($d = 0.59$, $p = 0.01$).

Lactate

No main effect was evident for time on power output a LT and effect size was small ($F(2,20) = 0.12$, $p = 0.89$, $\eta_P^2 = 0.01$ [95%CI: 0.00 to 0.12]; Table 6.4). There was no change in power at LT following the control period or training and ES was trivial (control: $d = -0.17$, $p = 1.00$; training: $d < 0.01$, $p = 1.00$; Table 6.4).

No main effect was evident for time on $[La^-]b$ a LT and effect size was medium ($F(2,20) = 1.24$, $p = 0.31$, $\eta_P^2 = 0.11$ [95%CI: 0.00 to 0.33]; Table 6.4). There was no change in $[La^-]b$ at LT following the control period or training and ES was small (control: $d = -0.21$, $p = 0.66$; training: $d = -0.44$, $p = 0.17$; Table 6.4).

Critical power

There was a significant main effect for time with a large effect size for critical power ($F(2,20) = 8.99$, $p < 0.01$, $\eta_P^2 = 0.47$ [95%CI: 0.10 to 0.65]; Figure 6.4). There was no change in critical power during the control period (Pre 1 - Pre 2; $p = 0.33$). Following SIT, there was a significant ($p = 0.01$) mean increase in critical power of 12W (95% CI -4, 27).

Peak power

There was no main effect of time of peak power output and a large effect size ($F(2,20) = 1.79$, $p = 0.19$, $\eta_P^2 = 0.15$ [95%CI: 0.00 to 0.38]; Figure 6.4). There was an increase in PP of 21W following the control period ($d = 0.19$, $p = 0.36$) and a further 23W following training ($d = 0.22$; $p = 0.41$) (Figure 6.4).

Total work

There was no main effect of time of total work and a large effect size ($F(2,20) = 2.48$, $p = 0.11$, $\eta_P^2 = 0.20$ [95%CI: 0.00 to 0.42]; Figure 6.4). No change in total work during control period was shown ($d = 0.13$, $p = 0.61$). Training had a large effect on total work, but the magnitude of change was not significant ($d = 0.35$, $p = 0.13$).

***Table 6.4 Changes in performance for control and sprint training periods.* denotes a significant difference between test points (p ≤ 0.05). Values shown are group mean ± SD.***

| Variable | Control period | | | | Sprint training period | | | |
|---|---|---|---|---|---|---|---|---|
| | Pre 1 | Pre 2 | p value | Standardised differences, ES ± 95% CI | Pre 2 | Post | p value | Standardised differences, ES ± 95% CI |
| CP | 206 ± 22 | 210 ± 27 | 0.33 | 0.18 ± 2.22 | 210 ± 27 | 222 ± 25 | 0.01* | 0.45 ± 0.91 |
| $\dot{V}O_{2peak}$ (ml·kg$^{-1}$·min$^{-1}$) | 37.65 ± 5.12 | 40.27 ± 6.04 | 0.16 | 0.47 ± 0.38 | 40.27 ± 6.04 | 44.52 ± 4.11 | 0.02* | 0.82 ± 0.81 |
| TTE (s) | 596 ± 51 | 613 ± 99 | 0.47 | 0.21 ± 0.33 | 613 ± 99 | 677 ± 118 | 0.01* | 0.58 ± 0.13 |
| $LT_{power}$ (W) | 121 ± 8 | 118 ± 21 | 1.00 | -0.19 ± 5.53 | 118 ± 21 | 118 ± 18 | 1.00 | 0.00 ± 1.06 |
| $LT_{[La^-]b}$ (mmol·l$^{-1}$) | 3.29 ± 0.69 | 3.15 ± 0.64 | 0.66 | -0.21 ± 0.02 | 3.15 ± 0.64 | 2.91 ± 0.47 | 0.17 | -0.43 ± 0.07 |
| PP (W) | 712 ± 101 | 733 ± 118 | 0.36 | 0.19 ± 0.01 | 733 ± 118 | 756 ± 86 | 0.41 | 0.22 ± 0.01 |
| $RSA_{total}$ (kJ) | 23.28 ± 1.65 | 23.50 ± 2.38 | 0.61 | 0.11 ± 0.31 | 23.50 ± 2.38 | 24.22 ± 1.57 | 0.13 | 0.35 ± 0.34 |

***Abbreviations: CP is critical power, $\dot{V}O_{2peak}$ is the highest rate of oxygen uptake recorded, TTE is time to exhaustion, $LT_{power}$ is power output at lactate threshold, $LT_{[La^-]b}$ is blood lactate concentration at lactate threshold, PP is highest power reached during repeated sprints, $RSA_{total}$ indicates total work done over six sprints.***

Lactate

There was no main effect of time on A with a small effect size ($F(2,20) = 0.47$, $p = 0.64$, $\eta_P^2 = 0.04$ [95%CI: 0.00 to 0.23]) following six sessions of SIT (Table 6.5). Magnitude of change during the control period was not significant and ES was small ($d = -0.39$, $p = 0.15$). There was also no change following SIT ($d = 0.17$, $p = 0.71$). No main effect was evident for rate of lactate appearance ($F(2,20) = 0.01$, $p = 0.99$, $\eta_P^2 < 0.01$ [95%CI: 0.00 to 0.00]; Table 6.5). ES for magnitude of difference was trivial for both the control period ($d = 0.07$, $p = 0.85$) and training period ($d = -0.03$, $p = 0.94$).

There was no main effect of time on rate on $k_2$ with a medium effect size ($F(2,20) = 1.17$, $p = 0.33$, $\eta_P^2 < 0.10$ [95%CI: 0.00 to 0.32]; Table 6.5).

***Table 6.5 Lactate kinetic variables for N and L following 3-minute critical power test prior to training. * denotes a significant difference between test points (p ≤ 0.05). Values shown are group mean ± SD.***

| Variable | Control period | | | | Sprint training period | | | |
|---|---|---|---|---|---|---|---|---|
| | Pre 1 | Pre2 | p value | Standardised differences, ES ± 95% CI | Pre 2 | Post | p value | Standardised differences, ES ± 95% CI |
| A ($mmol \cdot l^{-1}$) | 21.0 ± 2.7 | 22.2 ± 4.0 | 0.15 | -0.39 ± 0.90 | 19.48 ± 4.89 | 19.34 ± 4.58 | 0.71 | 0.17 ± 0.29 |
| $k_1$ ($mmol \cdot min^{-1}$) | 0.46 ± 0.15 | 0.37 ± 0.14 | 0.85 | 0.07 ± 0.00 | 0.47 ± 0.15 | 0.40 ± 0.18 | 0.94 | 0.00 ± 0.03 |
| $k_2$ ($mmol \cdot min^{-1}$) | 0.08 ± 0.02 | 0.09 ± 0.03 | 0.54 | 0.32 ± 0.01 | 0.09 ± 0.04 | 0.10 ± 0.06 | 0.24 | 0.20 ± 0.01 |
| $[La^-]b_{max}$ ($mmol \cdot l^{-1}$) | 15.66 ± 2.29 | 16.42 ± 2.48 | 0.04* | -0.52 ± 0.10 | 14.52 ± 2.05 | 14.28 ± 2.32 | 0.79 | 0.02 ± 0.01 |
| $T[La^-]b_{max}$ (min) | 4.81 ± 1.02 | 5.50 ± 1.43 | 0.76 | -0.12 ± 0.02 | 4.69 ± 1.06 | 5.98 ± 3.17 | 0.67 | 0.07 ± 0.02 |
| TP (min) | 9.63 ± 2.05 | 10.99 ± 2.86 | 0.76 | -0.12 ± 0.03 | 9.38 ± 2.12 | 11.96 ± 6.33 | 0.67 | 0.08 ± 0.04 |

***Abbreviations: A is extravascular release of lactate, $k_1$* is rate of lactate accumulation, $k_2$ is rate of lactate clearance, $[La^-]b_{max}$ is maximal blood lactate concentration, $T[La^-]b_{max}$ is time to maximal blood lactate concentration, and TP is turn point.**

***Table 6.6 Correlation between training outcome and lactate kinetic parameter. * indicates p ≤ 0.05 training variable to lactate kinetic response. Values shown are group mean ± SD.***

| | CP (W) | $VO_{2peak}$ (ml·$kg^{-1}$·$min^{-1}$) | TTE (s) | PP (W) | TW (kJ) |
|---|---|---|---|---|---|
| A (mmol·l-1) | 0.13 | -0.39 | 0.16 | 0.21 | 0.32 |
| $k_1$ (min-1) | -0.3 | 0.19 | -0.28 | -0.06 | -0.17 |
| $k_2$ (min-1) | -0.04 | -0.23 | 0.19 | 0.22 | 0.31 |
| [La-]bmax (mmol·l-1) | -0.04 | -0.45 | -0.21 | 0.42 | 0.14 |
| T[La-]bmax (min) | 0.32 | -0.28 | 0.07 | -0.23 | -0.16 |
| TP (min) | 0.32 | -0.28 | 0.07 | -0.23 | -0.16 |

### 6.4.3 Lactate supplementation following training

Critical power

Following SIT there was no difference in CP between conditions (d = -0.21, p = 0.13, Table 6.7).

$\dot{V}O_{2peak}$

Following SIT there was no difference in $\dot{V}O_{2peak}$ between conditions (d = -0.13, p = 0.67, Table 6.7).

Time to exhaustion

TTE was significantly reduced in L following training (d = -0.13, p = 0.03, Table 6.7).

Lactate threshold

Power and blood lactate concentration at LT were unchanged with supplementation following training (d = 0.08, p = 0.80; d = 0.41, p = 0.30, Table 6.7).

Peak power

Peak power was not different between conditions following training (d = 0.32, p = 0.09, Table 6.7).

Total work

Following SIT there was no difference in total work performed over the six sprints between conditions (d = 0.24, p = 0.13, Table 6.7)

***Table 6.7 Performance without (N) and with (L) lactate supplementation following SIT. Values shown are group mean ± SD. * denotes a significant difference between conditions (p ≤ 0.05).***

| Variable | Post | | | |
|---|---|---|---|---|
| | N | L | p value | Standardised differences, ES ± 95% CI |
| CP | 222 ± 25 | 217 ± 26 | 0.13 | -0.21 ± 0.39 |
| $\dot{V}O_{2peak}$ (ml·kg$^{-1}$·min$^{-1}$) | 44.52 ± 4.11 | 43.55 ± 9.69 | 0.67 | -0.13 ± 2.46 |
| TTE (s) | 677 ± 118 | 662 ± 110 | 0.03* | -0.35 ± 0.23 |
| $LT_{power}$ (W) | 120 ± 19 | 122 ± 17 | 0.80 | 0.08 ± 0.99 |
| $LT_{[La^-]b}$ (mmol·l$^{-1}$) | 2.91 ± 0.47 | 3.13 ± 0.60 | 0.30 | 0.41 ± 0.05 |
| PP (W) | 756 ± 86 | 783 ± 85 | 0.09 | 0.03 ± 0.004 |
| $RSA_{total}$ (kJ) | 24.22 ± 1.57 | 24.62 ± 1.76 | 0.13 | 0.24 ± 0.08 |

***Abbreviations: CP is critical power, $\dot{V}O_{2peak}$ is the highest rate of oxygen uptake recorded, TTE is time to exhaustion, $LT_{power}$ is power output at lactate threshold, $LT_{[La^-]b}$ is blood lactate concentration at lactate threshold, PP is highest power reached during repeated sprints, $RSA_{total}$ indicates total work done over six sprints.***

Lactate

There was no significant difference between conditions for A, $k_1$, $k_2$, $[La^-]b_{max}$, $T[La^-]b_{max}$ and TP following training ($p > 0.05$, Table 6.8).

***Table 6.8 Lactate kinetic variables for N and L following 3-minute critical power test post training. Values shown are group mean ± SD.***

| Variable | Post | | | |
|---|---|---|---|---|
| | N | L | p value | Standardised differences, ES ± 95% CI |
| A ($mmol \cdot l^{-1}$) | 20.2 ± 4.2 | 20.7 ± 5.4 | 0.80 | 0.09 ± 0.50 |
| $k_1$ ($mmol \cdot min^{-1}$) | 0.47 ± 0.22 | 0.44 ± 0.15 | 0.72 | -0.16 ± 0.03 |
| $k_2$ ($mmol \cdot min^{-1}$) | 0.10 ± 0.06 | 0.10 ± 0.05 | 0.51 | 0.20 ± 0.01 |
| $[La^-]b_{max}$ ($mmol \cdot l^{-1}$) | 14.57 ± 2.02 | 14.96 ± 1.68 | 0.76 | 0.21 ± 0.14 |
| $T[La^-]b_{max}$ (min) | 4.77 ± 1.11 | 4.61 ± 0.80 | 0.89 | -0.17 ± 0.13 |
| TP (min) | 9.55 ± 2.22 | 9.22 ± 1.60 | 0.89 | -0.17 ± 0.26 |

***Abbreviations: A is extravascular release of lactate, $k_1$ is rate of lactate accumulation, $k_2$ is rate of lactate clearance, $[La^-]b_{max}$ is maximal blood lactate concentration, $T[La^-]b_{max}$ is time to maximal blood lactate concentration, and TP is turn point.***

## 6.5 Discussion

The major findings in the current study are that a three week, twice-weekly cycle-based SIT intervention effectively improves performance indicators in female field hockey players although changes in lactate kinetics are limited. There was a statistically significant improvement in endurance performance ($\dot{V}O_{2peak}$ 10.6%, TTE 10.5%) and critical power (5.6%), and small, non-statistically significant improvements in PP (3.1%), and RSA (3.0%). Changes in lactate exchange rates (A and $k_2$) were small and weakly correlated to performance indices. HIIT has been shown to be more time efficient than SSG or MICT for female invasion sport athletes (Edge et al., 2005; Gabbett and Mulvey, 2008). In this study, training duration was 18 minutes per session which is shorter than that previously reported by Funch et al. (2017) who utilised a

running-based protocol lasting 30 minutes. Therefore, it appears that performing cycling-based SIT training twice-weekly (18 min), in addition to the regular hockey training sessions can help players develop aerobic and anaerobic capacity.

Supplementation of a 2% calcium lactate solution offers no ergogenic benefit for anaerobic capacity performance and may in fact be detrimental to aerobic capacity testing performance.

### 6.5.1 Effect of sprint interval training

Aerobic capacity

Aerobic capacity has been identified as a key element in the development of elite hockey players (Elferink-Gemser et al., 2004). Increased $\dot{V}O_{2max}$ is associated with more high intensity running during game play (Jennings et al., 2012), and greater RSA (Aziz, Chia and Teh, 2000). Following three weeks of SIT, $\dot{V}O_{2peak}$ was comparable to that of amateur female hockey players of a similar age and standard (Funch et al., 2017). The 10.6% mean increase in $\dot{V}O_{2peak}$ following three weeks of SIT was greater than that following four weeks of running-based HIIT (6.1%) or 'Tabata-style' HIIT (6.2%), despite a reduction in total training time of approximately 75%. In meta-analyses of SIT research, Gist et al. (2014) and Weston et al. (2014) reported the average increase in $\dot{V}O_{2max}$ to be ~7% in young healthy participants following HIT interventions lasting between two and ten weeks. This difference may be explained by females being more responsive to SIT in terms of increases in $\dot{V}O_{2max}$ elicited (Bagley et al. 2016).

It has previously been reported that central adaptations such as increased maximal cardiac output ($Q_{max}$) and stroke volume (SV) (Helgerud et al., 2007; Matsuo et al., 2014) which are associated with increased $\dot{V}O_{2max}$, are not evident without a more prolonged training period of six weeks or more (Daussin et al., 2007; Gillen et al., 2016; Warburton et al., 2004). However, increases in $Q_{max}$ (Astorino et al., 2017) and SV (Trilk et al., 2011) have been reported following short duration SIT suggesting these more central adaptations may be occurring within three weeks.

Mechanisms likely responsible for the increase in TTE following SIT include increased capillary density (Jensen et al., 2004), increased mitochondrial enzyme content and activity (Burgomaster et al., 2005), and faster $\dot{V}O_2$ kinetics (Bailey et al., 2009), although these were not directly measured in this study. Another explanation is a rightward shift in the blood lactate curve (Jakeman, Adamson and Babraj, 2012) which may well be the case as there was no difference in [La-]b at cessation of exercise between the pre and post testing, despite terminal exercise intensity being higher in a trained state. This suggests that lactate appearance is reduced at a given intensity of exercise with oxidative metabolism remaining predominant at higher intensities following SIT. PGC-1α has been shown to increase following HIT (Adhihetty et al., 2003; Gibala et al., 2012), and is a primary contributor to mitochondrial biogenesis (Laursen, 2010; Valero-Grinan, 2014; Uguccioni, D'souza and Hood, 2010). There is a concomitant increase in maximal activity of bioenergetic enzymes such as citrate synthase (CS) and cytochrome *c* oxidase (COX) following SIT (Burgomaster et al., 2006; Gibala et al., 2006). This facilitates increased energy production in the tricarboxylic acid (TCA) cycle and electron transport chain (ETC) leading to enhanced endurance performance.

It has been suggested that glycogen depletion plays an important role in skeletal muscle remodelling following training (Philp et al., 2012), with intramuscular glycogen concentrations increasing following short duration SIT (Burgomaster et al., 2005). Depletion of muscle glycogen levels is associated with a decline in field hockey performance in terms of high intensity work performed and number of repeated sprint actions (Spencer et al., 2005). The increase in TTE observed in this study may have been facilitated by increased muscle glycogen stores providing fuel for anaerobic glycolysis in the later stages of exhaustive exercise. Increased glycolysis is accompanied by an increase in lactate production which can have an inhibitory effect on anaerobic energy metabolism (Bangsbo et al., 1992), so for increased glycogen stores to be utilised effectively, the lactate must be either utilised within the muscle more effectively or transported out more rapidly. In the present study, there was only a weak correlation between $k_2$ and TTE (r = 0.19) suggesting increased time at high intensity may have been aided slightly by the increased rate of lactate clearance from the working muscles.

An area which was not controlled in this study was the use of oral contraceptives or menstrual cycle phase. Improvements in aerobic capacity following SIT are lessened through use of oral contraceptives (Schaumberg et al., 2017), and strength training in the follicular phase of the menstrual cycle elicits greater adaptations in muscular performance than training in the luteal phase (Sung et al., 2014). Future study could investigate whether this also applies to SIT and if it would be beneficial to individualise SIT periodisation in female athletes.

Critical power

CP is an important aspect for determining work rate during invasion sports with improvements in CP allowing a more intense pace without depleting the anaerobic work capacity reserves (W') (Jones and Vanhatalo, 2017). HIIT has previously been employed as a training modality to improve CP and does so effectively within four weeks (Kendall et al., 2009). However, the protocol used was time consuming (115min·wk$^{-1}$) compared with the current SIT protocol (36min·wk$^{-1}$) and achieved the same increase of approximately 6%. Increases in CP are linked to enhanced aerobic capacity through more efficient oxidative pathways (Jones and Vanhatalo, 2017). This includes increased cardiac output and blood haemoglobin concentrations transporting greater quantities of oxygen to the working muscle (Montero et al., 2015), improved oxygen extraction within the working skeletal muscle through increased capillary density (Raleigh et al., 2018; Tan et al., 2018), and increased fat oxidation facilitated by increased fat transporters such as FAT/CD36 (Perry et al., 2008) alongside reduced glycogen use (Burgomaster et al., 2008).

Conversely, whilst HIIT training increases CP, it has been shown to have a negative effect on W' (Kendall et al., 2009; Vanhatalo, Doust and Burnley, 2008). Following eight weeks of strength training (24 sessions), Sawyer et al. (2014) reported a 40% increase in W' with no change in CP. They attributed this change to increased glycogen stores allowing greater TTE during exercise >CP. Indeed, reduction in W' is linked to depletion of intramuscular glycogen reserves (Miura et al., 2000) and although W' was not measured in this study, the substantial increase in TTE seen is likely linked to increased glycogen stores (Perry et al. 2008) and it is possible there would also have been an increase in

anaerobic work capacity as seen in Study 2. This suggests that SIT is effective for improving both the $\dot{V}O_2$ related CP and the anaerobic work capacity of invasion sport athletes.

Peak power

Invasion sports like field hockey require players to repeatedly perform high-intensity sprint actions which, during the most intense periods of play, can be on a 1:1 work to rest ratio (McLean, 1992). The ability to generate muscular power is strongly correlated to sprint speed in female invasion sport athletes (Vescovi and Mcguigan, 2008) and increasing power to weight ratio is effective at improving speed during sport (Baker and Nance, 1999; Cronin and Hansen, 2005). Thom, Kavaliauskas and Babraj (2019) reported an increase in both cycle ergometer peak power and running speed following six weeks of SIT using a similar training protocol to this study so it could be assumed that running speed may also have increased in these participants too.

The 3.1% increase seen in PP was less than previously noted following 2-4 weeks of SIT; 5% (Burgomaster et al., 2007), 6.4% (Jakeman, Adamson and Babraj, 2012), 6.1-6.8% (Zelt et al., 2014). This may have been due to the level of resistance provided by the Wattbike Pro differing to the traditional 0.075 kg·kg body mass$^{-1}$ used in the other studies and perhaps not providing sufficient overload to develop power to the same extent.

During very short, supramaximal bouts of exercise of 6-10s, there is a high rate of ATP, PCr, and glycogen utilisation (Bogdanis et al., 1998; Gaitanos et al., 1993). This leads to a super-compensatory response resulting in elevated glycogen stores in skeletal muscle following training (Burgomaster et al., 2005; Gibala et al., 2006; Rodas et al., 2000). Following two weeks of 1:8 work to rest SIT, Gibala and colleagues (2006) reported a 28% increase in resting skeletal muscle glycogen levels and it may be this elevated substrate availability which is facilitating the increased power production (Jakeman, Adamson and Babraj, 2012). Another likely explanation for this increase in power is the neuromuscular adaptations seen after this type of training. Following two weeks of SIT, significant increases in motor unit conduction velocity and discharge rate for high-threshold motor units in the leg extensor muscles (Martinez-Valdes et al. (2017, 2018). Astorino and Schubert (2014) and Gurd et al. (2016) have

previously reported ‘non-responders’ following SIT training protocols with the percentage of non-responders reducing as protocol duration increases. It may be that this study training duration was not sufficient to elicit change in some participants in the time given.

RSA

High intensity training has previously been shown to enhance RSA within a short period of time (Viaño-Santasmarinas et al., 2018). Edge et al. (2005) found that in amateur female invasion sport athletes, cycling based HIIT improved RSA in terms of total work done by 13% after five weeks of three sessions per week. In this study, improvement was only 3%. Increased concentrations of MCT 1 and 4 following SIT (Burgomaster et al., 2004) facilitating an increased rate of lactate clearance may have allowed a higher rate of glycolysis in subsequent sprints therefore increasing total work. Increased aerobic capacity also facilitates faster regeneration of PCr allowing better performance in subsequent sprints.

### 6.5.2 Lactate supplementation

Aerobic capacity

It had previously been reported that 120mg·kg body mass$^{-1}$ calcium lactate supplementation increased both TTE and total work performed during cycling to exhaustion by approximately 17% (Morris et al., 2001). In the present study, supplementation of a 2% calcium lactate solution (~152 mg·kg body mass$^{-1}$) showed no clear ergogenic effect on $\dot{V}O_{2peak}$ or LT. This was similar to the results of Russ, Schifino and Leong (2019) who reported no significant increase in $\dot{V}O_{2peak}$ or exercise intensity at the onset of blood lactate accumulation (OBLA) following ingestion of 16 mg·kg body mass$^{-1}$ calcium lactate and magnesium lactate solution. Studies employing smaller doses of lactate of approximately 17 mg·kg body mass$^{-1}$ such as Northgraves et al. (2013) and Russ, Schifino and Leong (2019) have found far smaller, if any, differences in performance with lactate supplementation compared to studies employing high doses (120 mg·kg body mass$^{-1}$) such as Morris et al. (2011). Contrary to the hypothesis of this study, there was no clear improvement in aerobic capacity

through lactate supplementation and there was a small but significant performance decrement with lactate supplementation following SIT for TTE (Table 6.7). Supplementation of lactate also resulted in diminished performance during the critical power test in both tests prior to training (Figure 5.1), and during the post training test (Figure 6.9). Cycling performance over 20km was affected in a similar fashion following ingestion of 14.1 mg·kg body mass$^{-1}$ magnesium lactate and calcium lactate (Peveler and Palmer 2012) and these authors suggest that supplemented lactate is not being utilised by the working muscles but is being metabolised in other biological systems therefore not producing an ergogenic effect. Another possibility is a lack of bioavailability due to dose size and is supported by several studies. Supplementing calcium lactate, sodium lactate and potassium lactate, both Brouns et al. (1995) and Peronnet et al. (1997) reported no increase in bioavailability. No increase in plasma lactate was evident following ingestion of an 80% polylactate solution (Fahey et al. (1991) or when polylactate was combined with a glucose solution Swensen et al. (1994). One study which did report an increase in bioavailability following infusion of sodium lactate failed to find any performance difference during 20km time trials (Ellis et al., 2009). While Pre 1 critical power test showed a significantly higher blood lactate concentration at baseline, most other test points were equal for both conditions. It would appear supplementation of calcium lactate is not an effective ergogenic aid for improving aerobic capacity and any attempt to increase concentration further would likely result in severe gastrointestinal distress (Swensen et al., 1994).

Anaerobic capacity

Calcium lactate supplementation may benefit anaerobic work capacity in a buffering capacity through conversion to bicarbonate (Fahey et al., 1991; Morris et al., 2011; Oliveira et al., 2017; Painelli et al., 2014; van Montfoort et al., 2004), or through increased rate of lactate clearance (Gharbi et al., 2008; Messonnier et al., 2001, 2006; Pilegaard et al., 1999b). However, Painelli et al. (2014) and Olivira et al. (2017) both concluded that supplementation of calcium lactate provided no enhancement of peak power or total work during either upper or lower body 30s WAnT. In line with these findings, neither peak power nor total work performed during repeated sprints were benefitted by lactate

supplementation (Table 6.2) prior to SIT. However, following training both peak power and total work were slightly increased in L (PP 3.5%, TW 1.7%) (Table 6.7) during repeated sprint work. Additionally, immediately after the 3-minute critical power test, [La-]b was elevated to a greater degree in L suggesting a greater maximal power facilitated through elevated levels of anaerobic glycolysis (Edge et al., 2005; Thom, Kavaliauskas and Babraj, 2019). Lactate exchange rates increase with training status (Bergman et al., 1999) and are associated with increased high intensity exercise tolerance (Messonnier et al., 2002). While the improvements seen in performance were less than those reported by either Azevedo et al. (2007) and Morris et al. (2011), participant $\dot{V}O_{2peak}$ was also substantially less but results do indicate that response to lactate supplementation may be linked to aerobic fitness.

### 6.5.3 Conclusion

The findings of this present study have shown that a universal SIT protocol can be used to improve markers of match performance in female field hockey players such as critical power, aerobic capacity, and RSA. In terms of a practical training modality, it is very time efficient with only six minutes of exercise over the three-week period, carries very low injury risk to players, and players could utilise this training outwith club training sessions. Increased aerobic capacity is linked to increased total distance and high intensity work performed during invasion sport match play (Castagna et al., 2009; Al Haddad et al., 2015) suggesting this training modality would increase match related performance indicators (Mendez-Villanueva et al., 2012). It would also be a useful tool to compliment pre-competition training for sporting events where teams will travel to the host venue for a short period of time prior to commencing the tournament.

Improvements in lactate kinetics were less than those recorded in study 2 of this thesis and it is possible that a three-week protocol is not sufficient to elicit an adaptation of these components of metabolism. Future study is required to establish a minimum does to significantly alter lactate transport and metabolism. This study addresses the third aim of the thesis to determine whether supplementing lactate provides an acute increase in sport specific laboratory-

based performance tests performance, and whether this benefit is enhanced following training to alter the rate of lactate kinetics. While there were significant improvements in $\dot{V}O_{2peak}$, TTE, critical power, and large but non-significant increases in PP and RSA, there appears to be no ergogenic benefit of supplementing lactate solution for invasion sport performance tests. In fact, there appears to be a negative impact on some aspects of performance. The study has helped to further develop the current understanding of ergogenic aids to enhance invasion sport performance and the role lactate kinetics and metabolism play in this area.

# Chapter 7

# General Discussion

## 7.1 Introduction

This aim of this PhD thesis was to determine the role of lactate supplementation and sprint interval training on performance indicators in invasion sports athletes. It sought to determine whether sprint speed would be increased and if intermittent sprint performance would be preserved to a greater degree through supplementation of calcium lactate solution compared to either traditional sports drinks or water, during invasion sport play. It also sought to determine if sprint interval training (SIT) is effective for increasing the rate at which athletes metabolise lactate and, if so, whether performance would then be increased with supplemented lactate after training. Traditionally, sports drinks have been glucose based with many studies reporting some degree of benefit during endurance components of invasion sports (Foskett et al., 2008; Patterson and Gray, 2007) primarily though preservation of muscle glycogen (Russell, Benton and Kingsley, 2012). However, results were equivocal regarding their ability to attenuate the drop in repeated sprint performance seen during invasion sport match play (Abbey and Rankin, 2009; Ali and Williams, 2009). Few studies had previously investigated the ergogenic benefits of lactate supplementation despite indications it may be metabolised preferentially to glucose (Azevedo et al., 2007), and none in an invasion sport context. Skeletal muscle is the predominant consumer of lactate during periods of recovery between high intensity bouts which increase blood lactate concentration (Gladden, 2000; Kelley et al., 2002). Lactate produced either within (Brooks, 2002b), or from a separate muscle (Brooks, 1999), is transported into the muscle cell mitochondria where it undergoes further oxidative processing in the tricarboxylic acid (TCA) cycle (Brooks, 2002b). The ability to metabolise lactate and therefore reduce blood lactate concentration $[La^-]b$ has a strong correlation with performance in endurance sports (Baldari et al., 2007; Jacobs et al., 2011). At an exercise intensity between 70 - 90% maximal oxygen uptake ($\dot{V}O_{2max}$) (Jones and Vanhatalo, 2017), which equates to a mean $[La^-]b$ of 4 $mmol \cdot l^{-1}$ (Billat et al., 2003), oxidative phosphorylation alone can no longer supply sufficient adenosine triphosphate (ATP) for the demand by working muscles, and this intensity is termed maximal lactate steady state (MLSS) (Denadai and Higino, 2004). Increasing $\dot{V}O_{2max}$ is therefore desirable as it will increase the

intensity of work at which this noticeable rise in blood lactate will occur. Providing a readily available source of lactate through supplementation should provide fuel for skeletal muscles during the lower intensity recovery phases throughout invasion sports and preserve blood glucose and muscle glycogen for passages of play at an intensity greater than MLSS (Brouns et al., 1995), thus preventing the performance decline associated with diminished levels of these metabolic substrates (Nielsen et al., 2011).

Many studies using short duration supramaximal sprints as a training paradigm, have focused on physiological performance markers such as $\dot{V}O_{2max}$ (Raleigh et al., 2018) or rate of force development (Kinnunen, Piitulainen and Piirainen, 2019). While these physiological characteristics correlate well with performance in invasion sport, the studies do not directly address whether SIT improves key indices of sports performance. Therefore, it is unclear whether SIT is an effective training modality within invasion sports. Approaching this from a practitioner point of view, it was also important to determine whether a generic SIT resistance protocol would be as effective as a tailored approach more commonly used in the laboratory environment.

## 7.2 Summary of main findings and practical implications

### 7.2.1 Lactate supplementation

In studies 1 and 3, lactate solution was administered orally in a 1% and 2% weight by volume calcium lactate solution respectively. It was hypothesised that during simulated match play and laboratory-based performance tests that increased substrate availability in the form of calcium lactate would increase sprint speed, help attenuate the decline in performance associated with diminishing intracellular glycogen reserves, and increase performance in both aerobic and anaerobic performance testing.

During invasion sports, sprinting actions are performed multiple times, often without sufficient time to replenish intracellular phosphocreatine (PCr) (Bogdanis et al., 1996; Spencer et al., 2004). These actions are performed far beyond power output at $\dot{V}O_{2max}$ and cannot be fuelled by oxidative phosphorylation alone. As repetitions of these maximal efforts continue, power

output is reduced and the muscle fibres fatigue (Mendez-Villanueva, Hamer and Bishop, 2008). Glycogen is stored in three distinct locations within the muscle cell and utilisation is dependent on factors such as training status of the individual along with exercise duration, intensity and mode (Marchand et al., 2007; Nielsen et al., 2009, 2011). Intramyofibrillar glycogen stores are used preferentially during high intensity activity (Nielsen et al., 2011), and their depletion is linked to calcium ($Ca^{2+}$) release rate (Ørtenblad et al., 2011), which is associated with power loss (Nielsen et al., 2010). Commercially available sports drinks designed to meet the energy requirement of physical activity most commonly supply energy in the form of sugars such as glucose, fructose and sucrose (Coombes and Hamilton, 2000) but many other supplements such as caffeine and nitrate have been used. It was previously shown that exercising skeletal muscle will oxidise lactate preferentially to glucose and fructose, therefore making it a potential supplement to help attenuate fatigue (Azevedo et al., 2007). An example of this, demonstrated supplemented lactate resulted in 25% greater performance than a glucose-based sports drink during high intensity activity to exhaustion that followed a period of moderate intensity exercise (Azevedo et al., 2007). Van Montfoort et al. (2004) and Northgraves et al. (2013) had also reported elevated performance during high intensity exercise following ingestion of lactate salts.

It may be possible that supplemented lactate acts as a catalyst for activation of aerobic metabolism by simulating endogenously produced lactate which would be present following a bout of high intensity exercise, allowing a more rapid transition to aerobic pathway predominance (Gurd et al., 2005; Gurd et al., 2006). Once this aerobic process had been activated, the consumed lactate would then be readily available for further processing as fuel within the skeletal muscle cell mitochondria.

### 7.2.2 Supplementation prior to training

In study 1, initial investigation into absorption rate following ingestion of a 1% calcium lactate solution found blood lactate was noticeably elevated from a resting concentration of 1.2 ± 0.5 $mmol \cdot l^{-1}$ within 10 minutes, peaked at 2.3 ± 1.0 $mmol \cdot l^{-1}$ 30 minutes after ingestion, and was still significantly elevated at the

60 minute blood test (Figure 4.3). This significant increase ($p = 0.01$) of 91.7% to peak level surpassed the lactate threshold of a 1 $mmol \cdot l^{-1}$ increase from baseline (Weltman et al., 1990), and is a blood lactate concentration associated with an exercise intensity >65% $\dot{V}O_{2max}$ (Wall et al., 2011). These findings were consistent with de Vrese, Koppenhoefer and Barth (1990), and de Vrese and Barth (1991) who reported peak $[La^-]b$ within 40 minutes. During this time, blood glucose concentration was unaltered (Figure 4.4) indicating the fate of ingested lactate is not likely to be transformation to glucose through gluconeogenesis in the liver as suggested by Brouns et al. (1995) but rather oxidation in other tissues. This is consistent with the idea that lactate is produced and consumed by various tissues and organs even while at rest, without prior conversion to glucose (Gladden, 2004).

Simulation of rugby union match play was achieved using the Bath University rugby shuttle test (BURST) created by Roberts et al. (2010a). Performance was measured through 17 evenly spaced performance tests which included a 15m sprint (Figure 4.2). The duration of these sprints was used to evaluate sprint speed and maintenance of performance since the ability to maintain sprint performance is seen as a decisive factor for success in invasion sports (Austin, Gabbett and Jenkins, 2011a; Bishop et al., 2006, 2011; Gabbett, 2010b; Girard, Brocherie and Millet, 2015; Rampinini et al., 2007). Sprint speed was not significantly different between conditions (Table 4.1), nor was total time spent sprinting in each exercise block or half (Table 4.2). However, there was a trend for the decrement in performance to be lower in the lactate condition compared to both glucose and water with a small to medium effect size (Table 4.3). It is likely that performance would be maintained to a greater degree during match play through supplementation of calcium lactate, and that this small benefit may influence match outcome. Heart rate and respiratory rate were consistently lower during the lactate condition than either glucose or water and effect size was large (Table 4.4). This may be due to lactate being readily consumed by the highly oxidative lactate dehydrogenase (LDH) rich cardiac muscle fibres (van Hall, 2000), or the cardiorespiratory system working slightly more efficiently in supplying required oxygen to working skeletal muscles (Concha et al., 2009; Salinas et al., 2006).

In study 3, lactate supplementation possibly had a negative effect on aerobic capacity in terms of critical power (CP) and $\dot{V}O_{2peak}$ although time to exhaustion (TTE) may have been slightly improved (Table 6.2). These findings were consistent with previous research where lactate supplementation had been found to be ineffective as an ergogenic aid for improving moderate intensity exercise performance (Peveler and Palmer, 2012; Russ, Schifino and Leong, 2019; Swensen et al., 1994). Low intensity exercise is fuelled predominantly through oxidation of fat (Lazzer et al., 2010) and it may be that when exercise consists of low to moderate intensity, lactate is not required by the working skeletal muscles for additional fuel and is utilised in other areas such as the cardiorespiratory system. In study 1, peak heart rate (HR) and respiratory rate (RR) were lower in the lactate condition suggesting that it was being utilised in this way.

There was also no clear influence of lactate supplementation on either peak power (PP) or repeated sprint ability (RSA) prior to SIT (Table 6.2). This is also compatible with previous research where calcium lactate increased neither upper nor lower body Wingate anaerobic test (WAnT) performance (Oliveira et al., 2017; Painelli et al., 2014).

### 7.2.3 Supplementation following training

Following three weeks of SIT, any ergogenic benefit from lactate supplementation was still not evident and it appeared to slightly diminish CP, $\dot{V}O_{2peak}$ and TTE (Table 6.7). Lactate supplementation also had no effect on anaerobic work capacity although, peak power and total work done were both slightly greater with lactate compared to water alone (Table 6.7). Increased aerobic fitness is associated with improved lactate exchange rates (Messonnier et al., 2002), and research which has shown the greatest ergogenic effect of lactate supplementation has been conducted with individuals with high aerobic capacity (Azevedo et al., 2007; Morris et al., 2011).

### 7.2.4 Practical implications of supplementation

Based on these findings, it appears that supplementing calcium lactate may provide some small benefit for maintaining intermittent sprint performance in invasion sport athletes. Maximal effort bouts of activity during matches serve a critical role in determining match outcome and sporting performance (Austin, Gabbett and Jenkins, 2011a; Bishop et al., 2006, 2011; Gabbett, 2010b; Girard, Brocherie and Millet, 2015; Little and Williams, 2005; Rampinini et al., 2007; Reilly, Bangsbo and Franks, 2000). During invasion sports, there is a decrement in sprint performance associated with fatigue through declining muscle glycogen (Krustrup et al., 2006), and this was attenuated to a greater degree with lactate supplementation suggesting it plays an active role in exercise metabolism. Use of calcium lactate as an ergogenic aid prior to invasion sports may help maintain intermittent sprint performance throughout the match although any ergogenic benefit would be small. At an elite level, any small benefit garnered may have a large effect on performance and match outcome (Comfort, Haigh and Matthews, 2012).

However, the increased high intensity exercise duration reported by Azevedo et al. (2007) and Morris et al. (2011) was not seen either before or after SIT. In fact, following training, lactate supplementation appeared to have a detrimental effect on TTE and CP performance. Similarly, anaerobic performance was also unchanged, and findings are in line with those of Oliveira et al. (2017) and Painelli et al. (2014). It is therefore the conclusion of this thesis that supplementation of calcium lactate would not be recommended prior to or during invasion sport match play as any ergogenic benefit would be very small and there may also be undesirable reductions in performance levels. Additionally, this thesis suggests further research into the use of lactate as an ergogenic aid would be unnecessary.

The conflicting conclusions of research on lactate supplementation appear to be mirrored in the literature on other supplements. While some papers report supplements such as caffeine (Del Coso et al., 2013; Lara et al., 2014; Schneiker et al., 2006; Stuart et al., 2005) and sodium bicarbonate (Durkalec-Michalski et al., 2020) have significant positive effects on sprint and invasion sport performance, others report the opposite (Brown, Brown and Foskett,

2013; Macutkiewicz and Sunderland, 2018; Paton, Hopkins, and Vollebregt, 2001). A variety of factors may influence outcomes beyond the supplement being tested. For example, a systematic review on the effect of the combination of creatine monohydrate and HMB on sports performance noted the tests used to assess strength, aerobic performance, and anaerobic performance were very different between studies making comparison of conclusions problematic (Fernández-Landa et al., 2019). Habitual use of supplements can also affect the outcome of studies and Evans et al. (2018) reported that only invasion sport athletes with low habitual caffeine consumption experienced an ergogenic benefit from caffeine supplementation during repeated sprint testing. Participant selection criteria may also affect the outcome of studies. Supplementation research is often conducted with participants of varying age, sex, and ability and the habitual use of supplements outwith the focus of the study may have confounding effect on the study outcome (Sobal and Marquart, 1994). Intra-participant variation can also have a confounding effect on supplementation research with normal between-trial variation leading sports scientists to false conclusions (Maughan, Shirreffs and Vernec, 2018).

To improve sports science research on supplementation, a more consistent or unified approach should be considered, and there are published recommendations on how this can be achieved (Pyne, 2014). It is important for recruitment criteria to be clearly set out and test specific outcomes reported. Recommendations should also not be overgeneralised but instead offer population specific conclusions in a clear and transparent manner.

## 7.3 SIT

The findings from study 2 and 3 indicate that cycle-based SIT using 6 x 10s sprints and a 1:8 work to rest ratio is a time effective and potent training modality for improving performance indices in invasion sport athletes. This was true in both adolescent male and adult female athletes, and whether baseline cardiovascular fitness was high (baseline: 56.1 ± 5.5ml·kg$^{-1}$·min$^{-1}$) or moderate (40.3 ± 6.0ml·kg$^{-1}$·min$^{-1}$). Invasion sports are by nature intermittent, characterised by repeated bouts of high intensity actions divided by periods of low to moderate activity (Faiss, Girard and Millet, 2013; Lemos et al., 2017).

Matches last for a prolonged period (field hockey = 60 minutes, rugby union = 80 minutes, soccer = 90 minutes) with an average heart rate of ~85% maximum HR ($HR_{max}$), meaning most athletes are close to their anaerobic threshold (Macutkiewicz and Sunderland, 2011; McGuinness et al., 2017). A highly developed aerobic capacity, measured though $\dot{V}O_{2max}$, is required to maintain this level of performance for the duration of a match (Gabbett, 2010a; Mohr et al., 2008; Roberts et al., 2008). In addition to this aerobic capacity requirement, athletes also need to exhibit highly developed anaerobic capacity, since nearly a third of energy contribution during play is from the anaerobic pathways (Osgnach et al., 2010; Paul, Bradley and Nassis, 2015; Stolen et al., 2005). Invasion sports require athletes to repeatedly perform high intensity activities such as sprinting, jumping and tackling, the duration and volume of which are a key component separating high and low level performers (Gabbett, 2010a; Mohr, Krustrup and Bangsbo, 2003), and match outcomes (Bradley et al., 2009). While single bouts of high intensity work producing high power outputs are predominantly anaerobic in nature (Gastin, 2001), aerobic metabolism becomes increasingly important as the number of high intensity actions increases (Bogdansi et al., 1996; Bogdanis et al., 1998, Gaitanos et al., 1993; Parolin et al., 1999). The ability to repeatedly perform high intensity actions is strongly linked to aerobic capacity (Gharbi et al., 2015), and athletes with greater $\dot{V}O_{2max}$ can maintain peak power to a greater extent over repetitions (Hamilton et al., 1991).

To optimise training benefits, exercise stimulus should replicate the demands of match play (Gabbett, 2010a; Smart et al., 2014; Vaz et al., 2014), and strength and conditioning coaches should strive to develop this in invasion sport athletes. It is now well documented that high intensity interval training (HIIT) and SIT produce equal, or even greater adaptations than moderate intensity continuous training (MICT) (Milanović, Sporiš, and Weston, 2015; Smilios et al., 2018), and that running based protocols are effective whether these are interval based or small sided games (Kavaliauskas, Kilvington and Babraj, 2017; Owen et al., 2012). However, high speed running has been shown to significantly increase non-contact injury prevalence in invasion sports for both youth and non-elite adult athletes (Bowen et al., 2017; Gabbett, 2002). Cycle-based protocols have been shown to have a far lower rate of injury (Willoughby et al.,

2016), and importantly performance improvements translate well to running performance in terms of maximal speed (Thom, Kavaliauskas and Babraj, 2019) and repeated sprint ability (Hamlin et al., 2017). Cycle-based SIT is therefore a useful training modality to improve desirable performance criteria whilst reducing the risk of lower limb injury for invasion sport athletes, with the additional benefit of low time commitment for fitness (15 - 20 minutes per week) which will free time for technical and tactical training.

### 7.3.1 Physiological and performance adaptations

In study 2 and 3, participants were tested for peak rate of oxygen uptake ($\dot{V}O_{2peak}$) using breath by breath gas analysis (Metalyzer®3B gas analyser, Cortex, Leipzig, Germany), TTE using cycle ergometer incremental tests, PP through cycle ergometer maximal effort sprints, RSA (study 2; 6 x 20m running sprints on a 15 second cycle; study 3; 6 x 10s cycle sprint on a 1:1 work to rest ratio). This was done both before and after SIT. In addition to this, in study 2, running speed and jump height were also tested and in study 3, CP was tested. In both studies there was a statistically significant increase in $\dot{V}O_{2peak}$ following SIT (Table 4.2 and 5.4). Changes in aerobic capacity following training can be considered as either central or peripheral in nature (Raleigh et al., 2018). Following traditional MICT, central adaptations include increased heart structure and function (Baggish et al., 2008; Daussin et al., 2008; Helgerud et al., 2007; Matsuo et al., 2014; Montero, Diaz-Canestro and Lundby, 2015; Montero et al., 2015), and an increase in total plasma volume and red blood cell volume (Bonne et al., 2014; Swaka et al., 2000). The induced peripheral adaptations through MICT include a decrease in vascular resistance (Weng et al., 2013), increased skeletal muscle capillarisation (Montero et al., 2015; Murias et al., 2011), increased mitochondrial content and function (Jacobs et al., 2013b; Jacobs and Lundby, 2013; Jørgensen et al., 2007; Little et al., 2010; Montero et al., 2015), improved oxygen extraction (Murias, Kowalchuk and Paterson, 2010), and more efficient blood flow distribution (Kalliokoski et al., 2001). The increase in $\dot{V}O_{2max}$ seen following MICT is mostly attributable to an increase in cardiological (Arab-Zadeh et al., 2014; Montero, Diaz-Canestro and Lundby, 2015) and haematological (Montero et al., 2015) adaptations, while oxidative

phosphorylation remains largely unchanged (Montero et al., 2015). Many of these adaptations are also evident following high intensity training (HIT) with Matsuo et al. (2014) and Efandiari, Sasson and Goodman (2014) each reporting an increase in stroke volume (SV) following either HIIT or SIT. There is evidence to suggest central adaptations do occur following short duration HIT, with Astorino et al. (2017) reporting significant increases in cardiac output following three weeks of HIIT/SIT, and Trilk et al. (2011) reporting increased SV after four weeks of Wingate-based training. TTE was also significantly increased in both studies (Table 4.2 and 5.4). Despite some studies (Astorino et al., 2017; Trilk et al., 2011) evidencing central adaptations following short duration interventions, central adaptations generally require a period of training in excess of six weeks to become meaningful (Gillen et al., 2016 Warburton et al., 2004). The performance adaptations such as TTE following short duration SIT interventions are more likely to be facilitated by peripheral adaptations (Jacobs et al., 2013; Raleigh et al., 2018). Oxidative capacity of skeletal muscle is increased following HIT (Burgomaster et al., 2008; Gillen et al., 2013, 2014; Jacobs et al., 2013; Ma et al., 2013; MacPherson et al., 2011; Perry et al., 2008; Talanian et al., 2006). One aspect of this improvement is increased capillary density and capillary contacts per muscle fibre within skeletal muscle, allowing increased oxygen delivery within the cells (Cocks et al., 2013; Raleigh et al., 2018; Tan et al., 2018). The increase in skeletal muscle oxidative capacity is also reflected in biochemical measurements of maximal protein activity/content of mitochondrial enzymes, and markers of mitochondrial respiration (Bishop et al., 2019; MacInnis and Gibala, 2017). Following SIT, mitochondrial content and activity of proteins such as citrate synthase (CS), pyruvate dehydrogenase (PDH) (Burgomaster et al., 2005, 2006), and peroxisome proliferator-activated receptor γ coactivator (PGC-1α) (Ma et al., 2013) increases.

PP was increased by a statistically significant amount in study 2 (Table 4.2) and SIT was possibly beneficial for PP in study 3 (Table 5.4). PP may be increased through elevated glycogen availability following training (Burgomaster et al., 2006) or through neuromuscular changes improving muscle recruitment efficiency (Creer et al., 2004). RSA in study 3 was determined in terms of total work done. Participants improved 3% ($p = 0.13$) and there was a large effect

size ($\eta_P^2$ = 0.16), with magnitude of change being possibly beneficial (Table 5.4). This may be due to more sustained anaerobic glycolysis facilitated by the increased concentrations of lactate dehydrogenase (LDH) following exercise (van Hall, 2000; Xu et al., 2016). This would also explain the greater magnitude of blood lactate seen following maximal effort bouts. It may also have been facilitated by phosphofructokinase (PFK) levels which are shown to increase following sprint training (Fournier et al., 1982). PFK has been cited as the rate limiting factor for anaerobic glycolysis during maximal exercise (Alves and Sola-Penna, 2003; Hearris et al., 2018), and is strongly correlated with sprint performance during invasion sports (Mohr et al., 2016) and RSA (Iaia et al., 2011).

Changes in lactate kinetics following training

Another major finding from this thesis is that twice-weekly cycle-based SIT improves whole body lactate kinetics following maximal exercise. Additionally, these changes in lactate kinetics are correlated with different aspects of performance in adolescent soccer players. With most players performing near or above MLSS for substantial portions of the game, an accumulation of lactate is evident throughout play (Roberts et al., 2010a). Match mean [$La^-$]b of 4mmol·$l^{-1}$ in soccer players (Aslan et al., 2012), 4.5mmol·$l^{-1}$ in rugby union players (McLean, 1992), and 4.9 ± 2.1 mmol·$l^{-1}$ in hockey players (Buglione et al., 2013) have been recorded. Elite players show greater levels due to the higher volume and duration of high intensity actions they perform (Mohr, Krustrup and Bangsbo, 2003). Improvements in lactate metabolism may aid invasion sport performance by allowing superior playing intensity at reduced lactate concentrations (Best et al., 2013; Edwards et al., 2003). Training to improve whole body lactate kinetics and lactate metabolism could therefore be beneficial to invasion sport performance. Previous studies have shown a rightward movement of the lactate curve following HIT (Best et al., 2013; Jakeman, Adamson and Babraj, 2012; Zelt et al., 2014), and intermittent training was shown to be more effective at increasing the rate of lactate removal from the blood than continuous training (Gharbi et al., 2010).

### 7.3.2 Practical implications of SIT for invasion sport athletes

These data signify that short duration, low volume cycle-based SIT improves several performance variables related to success in invasion sports and, importantly, has transference to running-based performance measures like maximal sprint speed and RSA (Hamlin et al., 2017; Thom, Kavaliauskas and Babraj, 2019). Cycle-based SIT additionally offers a non-load bearing alternative to running with minimal eccentric contraction of leg muscles (Gist et al., 2014). This serves the dual purpose of minimising risk of overuse injuries (Jones, Hamilton and Cooper, 2015), and allowing athletes to maintain aerobic capacity during rehabilitation which prevents them from running. Care should still be taken to include eccentric training as this reduces the likelihood of hamstring injury during match play (Petersen et al., 2001). This is an effective training paradigm for both novice (Martin et al., 2016) and highly trained athletes (Jones, Hamilton and Cooper, 2015), and the use of a generic training load could easily be implemented by coaches who do not have the time or expertise to tailor training plans to individual athletes. The short duration required to elicit significant benefits makes SIT an effective training method during periods of constraint within the training programme such as pre-season, tapering and travel for competition.

## 7.4 Considerations of experimental protocol

### 7.4.1 Gastric emptying and lactate shuttling

The lactate absorption rate used in these studies was based on the recorded time for blood lactate concentration to peak in participants at rest following ingestion of a 1% calcium lactate solution. Whilst there has been no prior research conducted on gastric emptying speed of lactate specifically, there is evidence to suggest that the rate of gastric emptying is influenced by multiple factors. Leiper et al. (2001) reported that the rate at which a carbohydrate (CHO) solution is absorbed during moderate to high intensity exercise is less than that which occurs during low intensity exercise. This reduction became even more distinct at exercise intensities over 75% $\dot{V}O_{2max}$. In both studies 1

and 3, exercise intensity was designed to be greater than this. The BURST protocol used in study 1 is designed to replicate rugby union match play throughout which, average intensity is ~80% $\dot{V}O_{2max}$ (Coutts, Reaburn and Abt, 2003; Cunniffe et al., 2009), and in study 3 the laboratory-based tests were maximal effort working up to, or surpassing, $\dot{V}O_{2max}$. Gastric emptying is also affected by solution concentration, with higher concentrations emptying at a slower rate. Murray et al., (1999) found that gastric emptying was significantly slower when an 8% CHO solution was ingested during prolonged moderate intensity exercise compared to water, 4%, and 6% CHO solutions. It may therefore be that the rate at which lactate was reaching the blood was reduced through a combination of exercise in each study, or the increased concentration of calcium lactate used in study 3, and this may have resulted in a dampening of any possible ergogenic effect during testing.

Additionally, no account was taken of participant cardiovascular fitness in the absorption rate data. It is documented that individuals with high levels of cardiovascular training exhibit a superior rate of lactate clearance from the blood (Aziz, Chia and Teh, 2000; Bishop and Edge, 2006). This was seen in studies 2 and 3 following SIT, with participants in study 2 demonstrating a significantly increased rate of lactate clearance which correlated strongly with increases in $\dot{V}O_{2peak}$. It has also been shown that LDH and monocarboxylate transporter (MCT) levels and activity are elevated following training (Dubouchaud et al., 2000; Kohn et al., 2010) which could also affect the rate lactate is absorbed and cleared from the blood.

### 7.4.2 Training intensity on Wattbike Pro

In sports science, the gold standard for assessing anaerobic power is the 30s WAnT (Herbert et al., 2015), with Monark and SRM cycle ergometers frequently used due to their reputation for being highly accurate and reliable (Astorino and Cottrell, 2012; Bertucci, Grappe and Crequy, 2011; Hopket et al., 2015). The validity and reliability of the Wattbike Pro cycle ergometer is also now well established for accurately assessing anaerobic power and monitoring adaptations elicited through training (Bellinger and Miahan, 2014; Cushman, Bott and Highton, 2018; Driller et al., 2013; Driller et al., 2014; Herbert,

Sculthorpe and Grace, 2015; Hopker et al., 2010; Wehbe et al., 2015b). It has been shown to produce highly reliable results for invasion sport athletes during short duration (6s) maximal power test (Wehbe et al., 2015b) which are highly correlated to traditional 30s WAnT ($r = 0.9$, $p < 0.001$) (Herbert, Sculthorpe and Grace, 2015), and also suitably accurate compared to the SRM powermeter (Hopker et al., 2010).

The Wattbike Pro generates pedalling resistance through both air (level 1 - 10) and magnetic (level 1 - 7) braking systems, with performance test resistance automatically prescribed based on participant body mass. SIT research utilising the mechanically resisted Monark cycle ergometers generally prescribes a resistance of 0.075 kg·kg body mass$^{-1}$, described as the ideal resistance by Ayalon, Inbar and Bar-Or (1974). This standardisation makes comparison between variations in protocol such as changes in resistance, sprint or rest duration, and number of sprints easier. There is far less SIT research using Wattbike Pro, and training load varies widely between studies. Jones, Hamilton and Cooper (2015) investigated the effect of six sessions of SIT in elite female hockey players using air brake level 3, magnetic brake level 1. Hamlin et al. (2017) prescribed training resistance at air brake level 3 and magnetic brake level 3, increasing to air brake level 5 and magnetic brake level 3 to provide additional overload for non-elite male rugby players. Wehbe and colleagues (2015a) increased resistance even further with air brake level 10 and magnetic brake level 4 in elite level Australian rules footballers. Other studies lack information on level of resistance for training protocols relying instead on either prescribed power outputs or heartrate-based intensity (Beard et al., 2019; Devin et al., 2016). Training intensity for SIT utilising Wattbike Pro in this thesis was therefore an estimation based on the limited prior research (Jones, Hamilton and Cooper, 2015), whilst simultaneously applying the principle of overload (Goldspink, 1999) to elicit desirable performance outcomes.

### 7.4.3 Evolution of the thesis narrative

Due to the extended period of time over which this research project was undertaken, there were unavoidable changes required to the direction and narrative of the project. One of the main criteria within a doctoral thesis is the

production of novel research which contributes to and expands the current understanding within the field (Duc et al., 2020). Study 2 and 3 were initially designed to incorporate a training group who would perform SIT in hypoxic conditions. Training in the hypoxic conditions of simulated altitude leads to an increase in blood haemoglobin levels and has been associated with greater improvements in $\dot{V}O_{2max}$ (Bailey and Davies, 1997; Park, Shin and Lim, 2017) and anaerobic performance for activities such as sprint swimming and upper body WAnT (Martino, Myers and Bishop, 1996). This suggested hypoxic training may be beneficial to the repeated sprint performance which is linked to the intermittent high intensity movement patterns seen during invasion sport. However, prior to completion of this study, research was published in this area with a similar demographic of invasion sport athletes demonstrating the addition of a hypoxic environment to HIT provided no additional benefit to intermittent endurance performance, RSA, lower-limb explosive power, or maximal sprint performance compared to normoxic HIT (Brocherie et al., 2015; Gatterer et al., 2015). It was therefore decided not to include this element within the studies due to the substantial cost implication and the possibility the findings would not provide original information. However, the remaining SIT groups/period still provided the required information to address the aims of the thesis, so the overall objective was unaltered.

Study 2 also saw a change in participant group from study 1. The opportunity to work with an elite level of athlete arose and it made methodological sense to employ this type of group. Elite level athletes within a sport can generally be considered a homogeneous population (Ackland et al., 2003;) although within sports such as rugby union this may need to be considered by position rather than across the whole team (Ackland, 2006). In sports such as soccer or field hockey though, outfield players exhibit only low to moderate variation in physical characteristics such as body mass (Silvestre et al., 2006), and compared to non-elite athletes, elite level players also exhibit lower variation for performance measures such as vertical jump, sprint speed, and agility (Rebelo et al., 2013). By using a relatively homogeneous group, standard deviation is reduced and therefore small changes in performance are more likely to be detected (Lockie et al., 2013). It was anticipated recruitment for study 3 would

be done within the same population but a change within the management structure of the club meant this was not possible. Management in elite sport environment changes frequently and this often results in a disruption of established philosophies, practices, and routines (Cruickshank and Collins, 2012). One of the weaknesses of sports science research bridging to practice described by Finch (2011) is the failure to adopt new evidenced ways of training. The new management team demonstrated a reluctance to adapt their coaching philosophies despite the evidence presented to them and permission to work with this group of participants was withdrawn prior to study 3 commencing so recruitment was required in a new demographic. However, due to the similarities and shared components of invasion sports, recruitment of participants from a different sport did not affect the ability to address the main aims of the thesis.

## 7.5 Future direction

### 7.5.1 Lactate supplementation

Absorption rate and lactate concentration

In study 1, absorption rate of lactate was measured through the increase in blood lactate concentration. This was significantly elevated after 20 minutes and peaked at 30 minutes post ingestion. However, with the rate of gastric emptying potentially varying with both exercise intensity (Leiper et al., 2001) and supplement concentration (Murray et al., 1999) further study could be conducted to better define the parameters of time course for lactate absorption. This could be done in terms of; the influence of exercise intensity on the rate at which lactate is absorbed from the gut and appears in the blood, the effect of varying the concentration of calcium lactate on absorption rates both at rest and during exercise, and also the concentration threshold before gastrointestinal distress occurs. A solution of 2% has previously been used with no report of discomfort (Bryner et al., 1998), but Swensen et al., (1994) reported all participants experienced severe abdominal cramping, diarrhoea, and, in some cases, vomiting within a short period after a 5% solution was administered. They suggested a maximum solution strength of 2.5% which is still substantially

more than the initial dose used in this thesis. Carbon-labelled lactate solution could be used to determine the fate of ingested lactate and differentiate between exogenous and endogenously produced lactate present in the blood.

### Supplemented lactate to reduce heart rate

In study 1 it was observed that peak HR and RR were reduced through lactate supplementation compared to glucose and water. It has been demonstrated that in several sports such as golf (Boutcher and Zinsser, 1990), archery (Wang and Landers, 1986), and target rifle shooting (Konttinen, Lyytinen and Viitasalo, 1998) that a decrease in heart rate is evident in the preparatory phase. Lower heart rate is associated with improved performance in target pistol shooting (Kayhan et al., 2013; Thompson et al., 2015) and archery (Kolayiş and Mimaroğlu, 2008). Elite level shooters trigger during diastole (Helin, Sihvonen and Hänninen, 1987), and shooting in high pressure situations can result in elevated HR (Oudejans, 2008) which reduces the duration of this phase. Therefore, it would be advantageous for competitors in these types of sport to reduce their heart rate in order to optimise performance. In fact, the use of Beta-blockers during competition archery and shooting is banned both within and outwith competition by the World Anti-Doping Agency (wada-ama.org, 2020) showing the potential for lactate ingestion to play an important role in enhancing performance in these sports.

### Lactate interactions during high intensity exercise

Miller and colleagues (2002) investigated the role and destination of lactate during moderate intensity exercise (55 - 65% $\dot{V}O_{2max}$) by maintaining $[La^-]b$ of $4mmol \cdot l^{-1}$ through lactate infusion marked with [3-$^{13}$C] lactate tracers. They found that during exercise at these intensities, lactate oxidation rate increased, blood glucose levels were maintained, and glucose production was reduced. Currently there appears to be no research on this set of interactions at high intensity or intermittent high intensity exercise. In study 1, supplementation of calcium lactate appears to have slightly attenuated the drop in sprint performance during simulated rugby union play and, in study 3, to subtly

improve performance in supramaximal bouts during repeated sprints. During the incremental TTE test however, lactate seems to inhibit exercise duration. Muscle biopsies could be used to help determine the destination of ingested lactate, and whether there was in fact sparing of muscle glycogen because of the ingested lactate solution.

### 7.5.2 SIT: Comparability of training intensities for Wattbike Pro

There is a paucity of research utilising the Wattbike Pro for SIT compared to Monark cycle ergometers. These systems use different methods to create resistance, with the Monark being mechanically braked and the Wattbike using a combination of magnetic and air braking. More research is required to determine equivalent workload on the Wattbike to the industry standard 0.075 kg·kg body mass$^{-1}$ described by Ayalon, Inbar and Bar-Or (1974). This could be accomplished by comparing power output or total work performed during repeated sprints (Abbiss et al., 2009; Bertucci et al., 2005; Franklin et al., 2006), heart rate and blood lactate response at a prescribed exercise intensity (Mognoni et al., 1990), or through electromyography of the lower limb muscles to establish comparable workloads (Coelho et al., 2015).

## 7.6 General conclusion and practical implications

The aims of this thesis were to determine whether sprint performance will be enhanced through supplementation of calcium lactate solution compared to either traditional sports drinks or water during invasion sport play. It also sought to determine if SIT is effective at increasing the rate at which athletes metabolise lactate and if so, whether performance increases following training. In study 1, it was found that supplementing a 1% calcium lactate solution prior to, and during, simulated rugby union play attenuated the decrement in sprint performance seen throughout matches to a greater extent than water or a CHO sports drink. It was also evident that lactate supplementation resulted in lower peak HR and RR, suggesting lactate increases stroke volume during intermittent exercise. Study 2 demonstrated that short duration SIT is an

effective training paradigm for improving indices of fitness and match performance in young soccer players, with significant improvements in their aerobic and anaerobic capacity. It was also found that these physiological adaptations were accompanied by improved whole body lactate kinetics with increases in lactate appearance and clearance from the blood. SIT elicited similar physiological adaptation in study 3, although it appears that ingested lactate solution provides no ergogenic benefit to sport specific performance measures during laboratory based anaerobic and aerobic testing.

This thesis provides an insight for practitioners for enhancing performance in invasion sport athletes. Contact time with athletes is often limited and SIT provides a potent training modality to develop both the aerobic and anaerobic capacity required for success in these sports. As few as six sessions significantly increased $\dot{V}O_{2peak}$ and TTE in female athletes. Even in highly trained soccer players, twelve sessions increased aerobic capacity significantly. SIT also provided meaningful practical improvements in anaerobic power which translated to improvements in running speed, making cycle-based SIT suitable for running-based sports. SIT has been shown to be as effective as small-sided games (SSG) for improving fitness and, more importantly, meets the demand for maximal intensity efforts required to maximise training benefit. High volumes of maximal intensity running can increase injury risk through both overuse and stress related injuries. Cycle-based training poses a greatly reduced injury risk to both these elements and would result in fewer lost training hours through injury and rehabilitation.

It would also appear that supplementation of calcium lactate solution provides no meaningful ergogenic effect nor helps attenuate the drop in sprint performance seen throughout invasion sport matches. The trend seen in study 1 for better intermittent sprint performance is most likely accomplished by lactate providing a readily available, and rapidly metabolised energy substrate which helps preserve muscle glycogen for anaerobic glycolysis during maximal effort bouts. It is also clear any ergogenic effect is not amplified as aerobic capacity increases along with improved whole-body lactate kinetics.

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