The effect of acidosis on peak power after a simulated 4000-m individual pursuit on a bicycle ergometer

By

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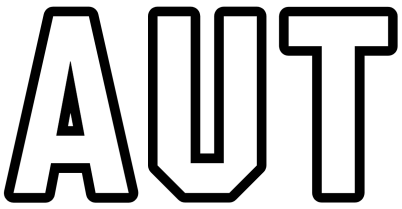

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List of abbreviations

BIC	Sodium bicarbonate intervention trial
PLA	Placebo intervention trial
SPR	Sprint subgroup
END	Endurance subgroup
NH_4Cl	Ammonium chloride
$NaHCO_3$	Sodium bicarbonate
NaCl	Sodium chloride
PCr	Phosphocreatine
P_i	Inorganic phosphate
ATP	Adenosine triphosphate
ADP	Adenosine diphosphate
H^+	Hydrogen ion
$[HCO_3^-]$	Bicarbonate ion concentration
$[La^-]$	Lactate anion concentration
$[K^+]$	Potassium ion concentration
$[Ca^{2+}]$	Calcium ion concentration
$[Na^+]$	Sodium ion concentration
SpO_2	Peripheral oxygen saturation
$\dot{V}O_2peak$	The maximal rate of oxygen uptake
$p\dot{V}O_2peak$	Power at the maximal rate of oxygen uptake
$v\dot{V}O_2peak$	Velocity at the maximal rate of oxygen uptake
APR	Anaerobic power reserve
ASR	Anaerobic speed reserve

PPO	Anaerobic peak power output
°C	Degrees Celsius
m	Meters
W	Watts
s	Seconds
min	Minutes
$mmol.L^{-1}$	Millimoles per liter
μL	Microliter
mL	Milliliters
$mL.kg^{-1}.min^{-1}$	Milliliters per kilogram per minute
$g.kg^{-1}$ BM	Grams per kilogram of body mass
$*g.L^{-1}$	Grams per litre
$N.m^{-1}$	Newton metres
$rev.min^{-1}$	Revolutions per minute

Attestation of authorship

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person (except where explicitly defined in the acknowledgements), nor material which to a substantial extent has been submitted for the award of any other degree or diploma of a university or other institution of higher learning.

Signed……

Mathew J. Mildenhall (Masters candidate)

Date……July 5, 2019

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Ethics approval

Ethical approval was given by the Auckland University of Technology Ethics Committee (AUTEC) on December 1, 2017 (application number 17/386) with no amendments required prior to submission after this approval was granted (see appendix A).

Abstract

Background: Large reductions in plasma and muscle pH have been associated with fatigue during supramaximal exercise. The ingestion of sodium bicarbonate ($NaHCO_3$) has often been used to attenuate plasma acidosis and provide an ergogenic effect. Due to the importance of the end-spurt in supramaximal events an understanding of what limits the ability to produce power late into a race could provide a competitive advantage. Additionally, the anaerobic power reserve (APR) has been put forth as a noninvasive tool for quantification and prediction of the anaerobic capabilities above aerobic capacity. However, limited research has related the model to changes in acid-base balance commonly seen over supramaximal exercise.

Aims: To determine: 1) the effect of $NaHCO_3$ on plasma acidosis and changes in anaerobic peak power output (PPO) after a 3-min fixed-intensity supramaximal cycling time-trial (based on the intensity requirements of a 4000-m individual pursuit) 2) the role of blood lactate concentration (blood [La^-]), plasma potassium concentration (plasma [K^+]), plasma calcium concentration (plasma [Ca^{2+}]), plasma sodium concentration (plasma [Na^+]), and peripheral oxygen saturation (SpO_2) on changes in PPO 3) the relationship between APR and changes in PPO and plasma measures.

Methods: Twelve elite cyclists from both sprint and endurance backgrounds were recruited to participate. Participants performed an initial testing session to determine APR, and two experimental intervention trials. During the intervention trials participants firstly ingested 0.3 $g.kg^{-1}$ body mass (BM) of either $NaHCO_3$ (BIC) or placebo (PLA) in a double-blind, randomized crossover design, 75 min prior to a standardized warm up. Performance testing then included an initial PPO (PPO1) followed by a fixed-intensity time-trial, simulating ~75%

(or 3 min) of a 4000-m individual pursuit at 105% of the power at $\dot{V}O_2$peak ($p\dot{V}O_2$peak) followed by a second PPO (PPO2). All trials were performed on a magnetically braked cycle ergometer.

Results: No difference in the percentage decrease of PPO (between PPO1 and PPO2) (45.7 ± 13.7% PLA vs. 42.3 ± 12.6% BIC, $P > 0.05$), peak torque ($P = 0.34$) or peak cadence ($P = 0.42$) was observed between PLA and BIC. Plasma bicarbonate concentration ($[HCO_3^-]$) was higher in BIC vs. PLA immediately following PPO1 (33.9 ± 2.7 vs. 27.0 ± 2.2 mmol.L^{-1}, $P < 0.001$), and PPO2 (26.7 ± 2.9 vs. 22.1 ± 2.7 mmol.L^{-1}, $P < 0.001$). Plasma pH was also higher in BIC at PPO1 (7.38 ± 0.05 vs. 7.29 ± 0.03 pH units, $P < 0.001$) and PPO2 (7.20 ± 0.12 vs. 7.13 ± 0.14 pH units, $P < 0.001$). No relationship was found between the percentage decrease of PPO and plasma pH at PPO2 ($r = -0.112$, $P > 0.05$) or the absolute change in plasma pH from PPO1 to PPO2 ($r = -0.016$, $P > 0.05$).

Blood $[La^-]$ was higher in BIC at PPO2 (19.6 ± 4.5 PLA vs. 22.1 ± 3.3 mmol.L^{-1}, $P = 0.011$). Plasma $[K^+]$ increased from rest to PPO1 in both trials (1.1 ± 0.6 mmol.L^{-1} PLA, $P < 0.001$ and 0.8 ± 0.7 mmol.L^{-1} BIC, $P = 0.01$), however no further increases were seen from PPO1 to PPO2 (PLA, $P > 0.05$ or BIC, $P = 0.12$). SpO_2 decreased throughout the fixed-intensity time-trial (99.6 ± 0.7 to 96.0 ± 2.5% PLA, $P = 0.02$ and 99.5 ± 0.8 to 96.3 ± 3.7% BIC, $P = 0.02$). However, no difference was seen between trials ($P > 0.05$).

A strong positive correlation was found between APR and the percentage decrease of PPO ($r = 0.79$, $P = 0.002$). APR was negatively correlated to the absolute increase in blood $[La^-]$ from PPO1 to PPO2 ($r = -0.59$, $P = 0.045$), and positively correlated to the absolute decrease in plasma $[HCO_3^-]$ from PPO1 to PPO2 ($r = 0.60$, $p = 0.039$). Moderate but non-significant

relationships were seen between APR and the absolute changes in plasma pH from PPO1 to PPO2 ($r = 0.52$, $P = 0.08$).

Conclusion: Differences in plasma acidosis do not appear to affect the decrease in PPO following a simulated 4000-m individual pursuit cycling time-trial. Additionally, these findings question the rise in plasma [K^+] and decrease in SpO_2 as limiting factors dictating the ability to sprint at the end of a 4000-m individual pursuit. Furthermore, the inverse relationship between the APR and the increase in blood [La^-], as well as a non-significant relationship between APR and the change in plasma pH suggest that a higher APR does not reflect the capacity of the anaerobic glycolytic pathway. Instead this implies that the ATP-PCr pathway is a major determinate of the APR.

Chapter 1 - Introduction and rationale

1.1 Thesis organisation

This thesis is prepared in the traditional research structure (format 1). It is therefore wholly written in accordance to the guidelines outlined in the AUT postgraduate handbook (2018) and includes several distinct chapters:

- ***Chapter 1 - Introduction and rationale***. Briefly outlines the role of plasma acidosis in the context of supramaximal exercise performance. It also provides rationale behind the study design, the aims and hypotheses, and the significance of the thesis.
- ***Chapter 2 - Narrative review of the literature***. Firstly, overviews the importance of the anaerobic system and PPO in supramaximal exercise. It then moves to the current understanding of acid-base balance and the proposals for acidosis' role in fatigue. Focus is then paid to $NaHCO_3$ supplementation and its role in supramaximal exercise performance. Finally, a review of the anaerobic power reserve and anaerobic speed reserve (ASR) showcases the need for further research to validate and strengthen the model's application.
- ***Chapter 3 - Research methods***. Report of the overall study design, measures and statistical analyses used to execute the study.
- ***Chapter 4 - Results***. Collation of results. Firstly, overall and subgroup participant characteristics are outlined to distinguish the elite capabilities and variability between participant subgroups. Secondly, in order to answer the first two research aims the overall and subgroup performance and physiological findings are presented. Finally, correlations between APR and the various performance and physiological measures are also outlined.
- ***Chapter 5 - Discussion***. Provides an explanation of the findings in relation to the aims of the study. This section firstly touches on the key findings of the study. It then moves to

discuss the effectiveness of the $NaHCO_3$ loading protocol and that no ergogenic effect was seen. Several proposals are then presented for why this lack of erogenicity was observed, leading to postulation on the other mechanisms of fatigue. Finally, an investigation into relationships between the APR and both performance and physiological measures are explored.

- ***Chapter 6 – Conclusion, limitations and future areas of stud***y. A reflection on the study design, findings, and potential directions for future research. This section begins with concluding remarks concerning the study's findings. Following this, several limitations are highlighted with justification provided as to why certain decisions were made concerning the study design despite these limitations. Finally, several areas for future research are considered.

1.2 Background

Several prominent Olympic events are competed over durations and intensities that fall within the supramaximal exercise domain (Craig et al., 1995). Supramaximal exercise typically lasts less than 10 min at intensities above the maximal rate of oxygen uptake ($\dot{V}O_2$peak) (Bassett & Howley, 2000). As intensity increases above $\dot{V}O_2$peak the capacity of the mitochondria to produce ATP is exceeded, causing the anaerobic pathway to be upregulated to meet the exercise demand (Brooks, 2010; Lindinger, Kowalchuk, & Heigenhauser, 2005; Robergs, Ghiasvand & Parker, 2004). Previous research has quantified the considerable anaerobic requirements of a variety of supramaximal events (de Campos Mello, de Moraes Bertuzzi, Grangeiro, & Franchini, 2009; Rodríguez & Mader, 2011; Spencer & Gastin, 2001; Zouhal et al., 2012). At durations of ~4 min, several endurance events across different modalities (i.e. 1500-m run, 400-m freestyle swim, 1000-m sprint kayak, and of interest to this study, the 4000-m individual

pursuit in cycling) have been shown to require similar energy system contributions (1% alactic, 14% anaerobic glycolytic and 85% aerobic) (de Campos Mello et al., 2009; Rodríguez & Mader, 2011; Spencer & Gastin, 2001; Zouhal et al., 2012). In addition to their energy requirements, these events are also characterized by the steady development of fatigue or impairment of performance through a reduced power generating capacity, and often an end-spurt or the sprint for the finish (Cairns, 2013; Corbett, 2009; Green, 1997; Place, Yamada, Bruton & Westerblad, 2010; Tucker, Lambert & Noakes, 2006).

Increased reliance on the anaerobic glycolytic pathway during supramaximal exercise increases the production and accumulation of exercising metabolites, namely lactate and protons (H^+) (Robergs, Ghiasvand & Parker, 2004). Produced in the muscle, increases in H^+ during supramaximal exercise results in drops in intramuscular pH from ~7.0 pH units to as low as 6.2 pH units (Hermansen & Osnes, 1972; Debold, Fitts, Sundberg, Nosek, 2016; Fitts, 2016; Robergs, Ghiasvand & Parker, 2004). Although initially neutralized by intracellular buffers, much of the excess lactate and H^+ moves across the sarcolemma and into plasma via MCT proteins and the Na^+-H^+ exchange (Hirakoba, 1999; Juel, 1998; Mason & Thomas, 1988; McGinley & Bishop, 2016; Westerblad & Allen, 1992). The efflux of H^+ can then cause plasma pH to drop from ~7.35 pH units at rest to ~6.8 pH units during and post-exercise (Hermansen & Osnes, 1972; Robergs, Ghiasvand & Parker, 2004). Traditionally, it has been believed that the increased production of both lactate and H^+ (collectively referred to as lactic acid) play a primary role in fatigue (Fitts & Holloszy, 1976; Fletcher & Hopkins, 1906; Hill, Long & Lupton, 1924). However, current belief now views the two components of lactic acid separately, with lactate now viewed as a metabolic substrate while the increase in H^+ during exercise continues to be researched as a causal factor of fatigue (Brooks, 2018; Gladden, 2004).

Many of the proposed mechanisms underlying acidosis' role in fatigue occur intramuscularly (Fabiato & Fabiato, 1978; Favero et al., 1995; Fitts, 2016; Knuth, 2000; Lännergren & Westerblad, 1991; Westerblad & Allen, 1991). However, much of the intramuscular research has been conducted using animal models (Fabiato & Fabiato, 1978; Favero et al., 1995; Fitts, 2016; Knuth, 2000; Lännergren & Westerblad, 1991; Westerblad & Allen, 1991), with little research using isolated human muscle *in vitro* (Sundberg, Hunter, Trappe, Smith & Fitts, 2018). Instead research exploring the fatiguing effects of exercise-induced metabolic acidosis within human subjects has done so through the manipulation of plasma acidosis (Brien, 1982; Carr, Hopkins & Gore, 2011; Correia-Oliveira et al., 2017; Matson & Tran, 1993; McNaughton, Siegler & Midgley, 2008; McNaughton et al., 2016; Robergs, Hutchinson, Hendee, Madden & Siegler, 2005). Such manipulation has occurred through augmenting plasma bicarbonate (HCO_3^-) buffer capacity via exogenous sodium bicarbonate ($NaHCO_3^-$) or by inducing an acidosis via exogenous ammonium chloride (NH_4Cl). The bicarbonate buffer system provides 86% of the extracellular buffer capacity (Poupin et al., 2012), making it critical for maintaining acid-base balance during exercise. Therefore, acute $NaHCO_3$ ingestion has often been used as an effective ergogenic aid (Carr, Hopkins & Gore, 2011; Christensen et al., 2017; Matson & Tran, 1993; Peart, Siegler & Vince, 2012).

Studies investigating acute $NaHCO_3$ ingestion as a means of enhancing exercise performance have ranged in intensity, duration, modality and exercise protocol. However, study designs have predominantly used three types of exercise protocol, including fixed-intensity time-to-exhaustion trials, close-ended time-trials defined by set durations or distances, and repeated-sprint bouts. However, such protocols may lack ecological validity or generalizability to competitive situations (Ansley, Lambert, Scharbort, St Clair Gibson & Noakes, 2004; Amann, Hopkins & Marcora, 2008; Christensen et al., 2017; Faria, Parker & Faria, 2005; Marino, Gard & Drinkwater, 2011; Schimpchen, Skorski, Nopp & Meyer, 2016), particularly

in the context of supramaximal events > 110 s in duration, which generally include an end-spurt (Corbett, 2009; Tucker, Lambert & Noakes, 2006). In these events researchers speculate that the end-spurt is regulated by homeostatic disturbance of physiological variables such as H^+ (Tucker, Lambert & Noakes, 2006). Yet, the effect of $NaHCO_3$ on end-spurt performance during supramaximal exercise has yet to be investigated. Considering that winning margins in Olympic events spanning 45 s to 8 min were commonly separated by a 1% change in average speed at the previous two Games, the ability to change speed or produce PPOs quickly over the late stages of a race is critical to success (Christensen et al., 2017).

Given the anaerobic energy requirements of supramaximal exercise, the measurement and quantification of anaerobic ATP during exercise is of utmost importance for future research and understanding. However, measurement of anaerobic ATP production tends to require more invasive intramuscular measures and is therefore difficult to quantify, especially during whole-body exercise (Bangsbo, 1996; Bangsbo, 1998; Gastin, 2001; Noordhof, Skiba, & de Koning, 2013). Several common measures of anaerobic contribution during exercise include oxygen debt, peak blood [La^-] and maximal oxygen deficit, but each one of these measures has its own limitation (Gastin, 2001; Green & Dawson, 1993; Noordhof, Skiba, & de Koning, 2013). Firstly, oxygen debt has been found to overestimate anaerobic energy release by nearly two-thirds compared with intramuscular measures (Bangsbo et al., 1990). Secondly, the interpretation of blood [La^-] can be limited by acute changes in plasma volume (Coyle et al. 1986; Harrison 1986; Shepley et al., 1992), differences in concentration between plasma and muscle (Jacobs & Kaiser, 1982; Tesch, Daniels & Sharp, 1982), differences between sampling sites (i.e. finger and forearm venous) (Foxdal et al., 1990), an inability to elucidate the influence of production or removal when assessing changes in net blood [La^-] (Belcastro & Bonen, 1975; McGrail, Bonen & Belcastro, 1978), or the inability to capture energy derived from phosphagens, ATP and PCr (Gastin, 2001). Finally, concerns over the oxygen deficit measure

arise due to its reliance in several assumptions. One of which is its use of a linear extrapolation based off of submaximal $\dot{V}O_2$ and workloads, which may be a significant source of error (Åstrand & Saltin, 1961; Bangsbo, 1998; Green & Dawson, 1993).

Due to the limitations identified in the current measures of anaerobic energy contribution, the APR/ASR models has been proposed as a simple measure of anaerobic capabilities above aerobic capacity (Bundle, Hoyt & Weyand, 2003). The APR is defined as the difference between PPO and the power at $\dot{V}O_2$peak ($p\dot{V}O_2$peak), while the ASR is defined as the difference between maximal velocity and the velocity at $\dot{V}O_2$peak ($v\dot{V}O_2$peak) (Blondel, Berthoin, Billat & Lensel, 2001; Bundle, Hoyt & Weyand, 2003; Sanders, Heijboer, Akubat, Meijer & Hesselink, 2017). To date, this model has primarily been used to characterise the exponential reduction in mean power or velocity over increasing supramaximal durations (Blondel et al., 2001; Bundle, Hoyt & Weyand, 2003; Weyand & Bundle, 2005; Weyand, Lin & Bundle, 2006). More recently research has used the model to differentiate elite from sub-elite athletes, predict performance, and as a relative exercise intensity measure (Buchheit & Mendez-Villanueva, 2012; Sanders & Heijboer, 2019; Sanders et al., 2017; Sandford, Allen, Kilding, Ross & Laursen, 2019). However, research utilising the APR is in its infancy and requires more physiological context before it can be confidently applied (Boullosa, 2014; Boullosa & Abreu, 2014).

1.3 Study aims

This thesis aims to:

1) Determine the effect of exercise-induced plasma acidosis (modulated by $NaHCO_3$ supplementation) on changes in PPO after a fixed-intensity supramaximal time-trial, simulating ~75% of a 4000-m individual pursuit on a cycle ergometer.
2) Determine the mechanisms underpinning any changes in PPO towards the end of a simulated 4000-m individual pursuit cycling time-trial with a particular focus on plasma pH, plasma HCO_3^-, blood lactate, peripheral oxygen saturation (SpO_2) and plasma electrolytes, namely potassium (K^+), calcium (Ca^{2+}) and sodium (Na^+).
3) Understand the applicability of the APR in relation to several physiological and performance variables measured in response to fixed-intensity supramaximal cycling time-trial.

1.4 Study hypotheses

Three respective hypotheses are proposed:

1) $NaHCO_3$ supplementation will attenuate exercise-induced plasma acidosis leading to a reduced percentage decrease in PPO as a result of fatigue generated by the fixed-intensity supramaximal cycling time-trial.
2) The decline in SpO_2 from rest (prior to the start of the fixed-intensity supramaximal cycling time-trial) to PPO2 will be correlated with the reduction in PPO between PPO1 and PPO2.
3) A positive relationship will be observed between APR and the decrease in PPO from PPO1 to PPO2 as well as the increase in blood [La^-] from PPO1 to PPO2. A negative relationship

will exist between APR and the decrease in both plasma pH and plasma [HCO_3^-] from PPO1 to PPO2.

1.5 Study rationale

1.5.1 Peak power output as the key performance measure

Although previous research has explored the effect of plasma acidosis (manipulated through $NaHCO_3$ or NH_4Cl supplementation) on supramaximal exercise performance, little attempt has been made to explore it in the context of the end-spurt. Therefore, integration of an end-spurt may improve the ecological validity of this research over simple fixed-intensity time-to-exhaustion trials and close-ended time-trial designs (Ansley et al., 2004; Amann, Hopkins & Marcora, 2008; Marino, Gard & Drinkwater, 2011). The change in PPO is a valid and reliable measure for quantifying fatigue (Glaister, Howatson, Pattison & McInnes, 2008). Therefore, integrating this measure into the exercise protocol through assessment of the change in PPO, measured before and after a fixed-intensity time-trial, will help elucidate the effect of exercise-induced plasma acidosis' role on performance.

1.5.2 Physiological measures

With plasma acidosis being central to this thesis the study of plasma pH, plasma [HCO_3^-], blood [La^-] and SpO_2 will be the main focus. Several reasons exist for inclusion of these measures in the current investigation. Firstly, it is proposed that $NaHCO_3$ supplementation enhances lactate efflux and improves the intracellular/extracellular pH gradient during supramaximal exercise (Siegler, Marshall, Bishop, Shaw & Green, 2016; McNaughton et al.,

2016). Additionally, the effect of acidosis on the desaturation of haemoglobin via the Bohr effect was also considered (Thomas & Lumb, 2012). With decreasing plasma pH during supramaximal exercise the resultant declines in oxygen saturation have been suggested to inhibit central motor drive through hypoxemia (Nielsen et al., 2002; Romer, Dempsey, Lovering, & Eldridge, 2006; Subudhi et al., 2011; Vogiatzis et al., 2011). Finally, due to their proposed interactive effects with H^+, as well as their roles in muscular contraction and the calculation of acid-base balance via the strong ion difference, plasma K^+, Ca^{2+} and Na^+ will also be measured.

1.5.3 Exercise protocol design and participant recruitment

$NaHCO_3$ has been shown to exhibit consistent ergogenic effects during supramaximal cycling time-trials from 1-6.5 min in cohorts of well-trained participants (Carr, Hopkins & Gore, 2011; McNaughton et al., 2016; Peart, Siegler & Vince, 2012), and attenuate fatigue over the later stages of repeated-sprints (Bishop, Edge, Davis & Goodman, 2004; Kozak-Collins Burke & Schoene, 1994; Lavender & Bird, 1989; Peart, Siegler & Vice, 2012; Price, Moss & Rance, 2003; Zinner et al., 2010). Therefore, it is believed that the study of elite cyclists using a protocol that combines a longer supramaximal time-trial and PPO (as a measure of performance) later into the protocol would advance the current body of knowledge. Another reason for targeting an elite population is that the majority of research in this field has been conducted on untrained and recreationally active populations (Carr, Hopkins & Gore, 2011; Gough, 2018; McNaughton et al., 2016; Peart, Siegler & Vince, 2012). Therefore, recruitment of an elite cohort would provide much needed insights into this under-studied population.

Several factors were influential in the design of the exercise protocol. Firstly, it has been noted that the most substantial changes in acidosis occur over durations of 1-10 min (Cairns, 2006a). It was therefore determined that the supramaximal time-trial used in the exercise protocol would aim to simulate a 4000-m individual pursuit in track cycling. This was determined as the event falls within the time frame outlined above (~4 min) and is competed over similar durations and intensities (~105% p$\dot{V}O_2$peak) at the elite level to several other events (i.e. 1500-m in middle-distance running, 400-m freestyle and 1000-m kayak) (Craig & Norton, 2001; Jeukendrup, Craig & Hawley, 2000; de Campos Mello et al., 2009; Rodríguez & Mader, 2011; Spencer & Gastin, 2001; Zouhal et al., 2012). Additionally, as the study looked to explore the ability to sprint, the duration of the simulated time-trial was not meant to bring participants to complete exhaustion. Therefore, in order to find an appropriate duration for the fixed-intensity time-trial the pacing research of Ansley et al. (2004) was utilised. Using several 4000-m time-trials, Ansley and colleagues identified an increase in power generally occurring over the last quarter of the trial. With all these factors considered a fixed-intensity time-trial of 3 min at 105% p$\dot{V}O_2$peak was agreed upon, as it represents ~75% of the world record for the men's individual pursuit (Mexican Cycling Federation, 2018) and would not bring the participant to exhaustion before the final PPO (PPO2).

Despite research not identifying an end-spurt during a 4000-m individual pursuit studying the change in PPO is still a relevant endeavour (Corbett, 2009; De Koning, Bobbert, & Foster, 1999). Due to the unique competitive situation in which two cyclists race directly against one another, starting from opposite sides of the track, it can be assumed that in instances where competition is close an end-spurt may be required to separate the winner. Also, the race is characterised by a slow decrement in power output throughout, therefore the ability to better resist fatigue is critical and has often been measured using the change in PPO in repeated-sprint research (Corbett, 2009; Corbett, Barwood, Ouzounoglou, Thelwell, & Dicks, 2012; De

Koning, Bobbert, & Foster, 1999; Glaister, Howatson, Pattison & McInnes, 2008). Finally, the use of the end-spurt during this research increases its application to several other events such as the Keirin in track cycling and the 1500-m in middle-distance running, both of which include rapid changes in power or velocity throughout the final stages of the race.

To effectively explore relationships between the APR and changes in both performance and plasma measures a spectrum of varied APRs was required. Therefore, a variety of elite cyclists from both sprint and endurance backgrounds were recruited to participate in the study. These subgroups were based on competitive and training focus, which was identified during screening before initial testing. After recruitment it was noted that the twelve participants could be evenly classified into the two subgroups (sprint = SPR and endurance = END). Because of this subgroup analysis was added to the results section to assess whether differences existed between SPR and END participants.

1.5.4 Study of the anaerobic power reserve

Few studies have attempted to characterise the ASR/APR by the physiological responses that occur during supramaximal exercise (Buchheit, Hader & Mendez-Villanueva, 2012; Dardouri et al., 2014; Julio et al., 2019; Mendez-Villanueva et al., 2010; Panissa et al., 2016). Therefore, the third aim of the study arose in an effort to address this the limited research associating the APR/ASR to physiological changes. To date, no studies have examined the relationship between changes in acid-base balance that occur over supramaximal exercise and the APR. Therefore, the current study sort to investigate this much needed area. Given that both PPO and p$\dot{V}O_2$peak were already to be collected during initial testing the addition of the APR to

analysis could be easily added to the study design without requiring anything further from participants.

1.6 Significance of thesis

The aims of the current investigation look to address two areas where currently research is lacking. Firstly, the study will expand on the current understanding of the effects of exercise-induced acidosis and the use of acute $NaHCO_3$ supplementation. Despite being studied across a variety of modalities, intensities and durations the use of $NaHCO_3$ in supramaximal exercise performance has been limited to three types of exercise protocol. Yet, no research to date has explored the effect of exercise-induced plasma acidosis on the ability to change speed or power quickly over the concluding stages of supramaximal exercise. Given the importance of the end-spurt in supramaximal events >110 s this research will have a direct performance application for athletes and coaches and specifically the ability to sprint over the concluding stages of a 4000-m individual pursuit (Corbett, 2009; Tucker, Lambert & Noakes, 2009).

Secondly, the current investigation will look to add much needed research to the understanding of the APR and its relationship to physiological variables associated with supramaximal exercise. The principle use of the APR to date has largely been to help describe the exponential decrement in high-power performance with increasing duration (Sanders et al., 2017; Weyand, Lin & Bundle, 2006). Researchers have hypothesised that such an exponential relationship is not solely determined by the rate of ATP resynthesis (Weyand, Lin & Bundle, 2005). Instead it is possible that metabolite accumulation, and the resulting acidosis progressively reduces exercise intensity with increasing duration (Tucker, & Noakes, 2009). Therefore, exploring relationships between the APR and changes in acid-base balance is important for understanding

and application of the model as a non-invasive tool for estimating supramaximal performance (Sanders et al., 2017; Weyand, Lin & Bundle, 2006). Additionally, the model is based off of two capacity measures (maximal mechanical capacity i.e. maximal velocity or PPO, and aerobic capacity) that do not appear to capture the extent of the recruitment of anaerobic glycolysis in ATP production. Therefore, the study will look to relate changes in blood [La^-], plasma pH, plasma [HCO_3^-], several plasma electrolytes, and SpO_2 resulting from a fixed-intensity supramaximal time-trial to the APR of participants. This will help understand whether such physiological changes associated with the increased recruitment of the anaerobic glycolytic pathway during supramaximal exercise can actually be explained by the model.

Chapter 2 – Narrative review of the literature

2.1 Introduction

Several prominent Olympic events are competed over durations and intensities that fall within the supramaximal exercise domain (Craig et al., 1995). Supramaximal exercise is completed at intensities above aerobic capacity and which last less than 10 min. During such exercise the development of intramuscular and plasma acidosis has often been explored as a potential agent of fatigue (Cairns, 2006a; Cairns, 2006b; Fitts, 2016; Westerblad, 2016; Westerblad, Allen & Lännergren, 2002). Indeed, the use of $NaHCO_3$ to combat changes in plasma acid-base balance has often provided an ergogenic effect (Carr, Hopkins & Gore, 2011; Christensen et al., 2017; Matson & Tran, 1993; McNaughton, Siegler, & Midgley, 2008; McNaughton et al., 2016). Yet, closer examination of the research shows inconsistent results across studies. Erogenicity does appear to be more consistent in studies using close-ended time-trials of 1-6.5 min in well-trained participants and over later repetitions during repeated-sprint protocols (Bellinger, Howe, Shing & Fell, 2012; Driller, Gregory, Williams & Fell, 2012; Gough, et al., 2018; Gough et al., 2017; Kilding, Overton & Gleave, 2012; Sung-Gye, Dong-Sik, Sang Chul & In-Ho, 1990; Thomas et al., 2016). However, much of the research studying $NaHCO_3$ and performance lacks ecological validity, with the majority of studies using one of three exercise protocols (i.e. fixed-intensity time-to-exhaustion trials, close-ended time-trials, and repeated-sprint protocols). One area missing from this research is an exploration of the end-spurt, which may offer more relevant competitive insights (Martin, Davidson & Pardyjak, 2007). Finally, given the importance of anaerobic contribution to ATP production in supramaximal exercise a new measure, the APR/ASR, has arisen to quantify the anaerobic capabilities above $\dot{V}O_2peak$. Defined as the difference between PPO/ maximal velocity and $p\dot{V}O_2peak$/ $v\dot{V}O_2peak$ (Sanders

et al., 2017; Weyand, Lin & Bundle, 2006), research in the area is sparse and has not related the model to acute changes in acid-base balance.

Therefore, the purpose of this review is to:

- Establish the importance of anaerobic energy contribution and the end-spurt in supramaximal single bout events such as the 4000-m individual pursuit.
- Examine the measurement and regulation of acid-base balance, and the role of acidosis in fatigue.
- Summarize the methods of acute $NaHCO_3$ supplementation.
- Assess acute $NaHCO_3$ use in supramaximal whole-body exercise performance i.e. cycling, rowing, running and swimming.
- Explain the lack of ecological validity in study designs used to assess the effect of $NaHCO_3$ ingestion on supramaximal performance and argue the need for integration of the end-spurt.
- Outline the development and current understanding of the APR/ASR and how it could be assessed in future research.

2.2 Supramaximal performance and anaerobic energy contribution

Anaerobic metabolism is pivotal to supramaximal exercise performance. It is well established that the absolute ATP requirement and combination of substrate utilised during exercise is primarily determined by intensity (Brooks, 1997). Activation of the anaerobic pathways such as muscle glycogenolysis and glycolysis occurs as intensity increases and is achieved through the increased recruitment of fast-twitch muscle fibres and activation of the sympathetic nervous system (Brooks, 1997). Anaerobic glycolysis is a multi-step pathway fuelled by the production of glucose-6-phosphate derived from either blood glucose or muscle glycogen (glycogenolysis)

(Brooks, 2010; Lindinger, Kowalchuk, & Heigenhauser, 2005; Robergs, Ghiasvand & Parker, 2004). Although the utilization of glycogen via this pathway yields small amounts of ATP (3 ATP per glycosyl unit) relative to aerobic metabolism via the Krebs cycle (39 ATP per glycosyl unit), it is more rapid and therefore preferred when the required rate of ATP synthesis is high, such as during supramaximal exercise (Allen, 2004; Brooks, 1997; McArdle, Katch & Katch, 2006).

The importance and magnitude of anaerobic metabolism in supramaximal exercise performance is also well established in research. Blondel et al. (2001), showed that performance in supramaximal efforts (120% and 140% of v$\dot{V}O_2$peak) correlated more closely with anaerobic capabilities above aerobic capacity than v$\dot{V}O_2$peak. Additionally, the meaningful contribution of the anaerobic system has been quantified across a range of sports and modalities (i.e. cycling, rowing, sprint and middle-distance running, sprint kayak and short course swimming) (de Campos Mello et al., 2009; Rodríguez & Mader, 2011; Spencer & Gastin, 2001; Zouhal et al., 2012). Primarily concerning track cycling, several prominent events fall within the supramaximal domain (Craig et al. 1993; Craig et al., 1995; Withers et al. 1991; Mulder, Noordhof, Malterer, Foster & de Koning, 2015). The energetics of such events range from 95% anaerobic energy contribution during the 200-m sprint (competed at intensities estimated to be 280% p$\dot{V}O_2$peak for ~9 s), to roughly 50% during the 1000-m time-trial (~60 s and intensity of 180% p$\dot{V}O_2$peak), and 15% in the 4000-m individual pursuit (~4 min and intensity of 105% p$\dot{V}O_2$peak) (Craig & Norton, 2001). Although the percentage contribution of the anaerobic system appears to fall with increasing duration, its importance does not. The notable work of Medbø and Tabata (1993) showed that anaerobic capacity was not fully utilized until at least 2 min into maximal exercise. Despite subsequent research suggesting that the time to reach anaerobic capacity may be event specific, and therefore closer

to 70 s in well-trained athletes (Craig et al., 1995), others have ratified the findings of Medbø and Tabata (Mulder et al., 2015). Needless to say, these findings and the contributions of aerobic and anaerobic metabolism required over longer duration supramaximal exercise, illustrates the need for a robust anaerobic system even as duration and aerobic contribution increases.

One such event highlighting the complexity is the individual pursuit in track cycling. Competed over a fixed distance of 4000 m for males and 3000 m for females, two cyclist race in pursuit of one another from starting positions on opposite sides of a banked track (Union Cycliste Internationale, 2019). In this sense, the winner is determined by either catching the opposing rider or recording the fastest overall time for the fixed distance. The event requires not only requires intensities above p$\dot{V}O_2$peak (with average power outputs ~520 W in elite males) but also the maintenance of a reserve throughout the race in anticipation of fatigue (Corbett, 2009; Corbett et al., 2012). These complex race requirements stress the importance of an ability to resist fatigue throughout, but also a capacity to maintain and produce high rates of anaerobic energy.

The ability to quickly change speed or produce high-powers is critical to success in competition competed at supramaximal intensities and durations. Considering that winning margins in events spanning 45 s to 8 min were commonly separated by a 1% change in average speed at the last two Olympic Games, the ability to change speed or produce PPO quickly over the late stages of a race is critical (Christensen et al., 2017). Many of these races are decided by a final sprint for the line, known as the end-spurt (Noakes, St Clair Gibson & Lambert, 2005). Of the 28 world championship races competed within cycling, four are often decided in the end-spurt (men's and women's road race and scratch race), and two require repeated-sprints (men's and women's points race) (Craig et al., 1995; Martin, Davidson & Pardyjak, 2007). Additionally,

the end spurt has also been shown to occur across other modalities (i.e. running and rowing) at durations > 110 s (Tucker, Lambert & Noakes, 2009). The end-spurt occurs under the accumulated fatigue of a race and is believed to be limited by physiological, psychological and tactical constraints (de Morree & Marcora, 2013; Noakes, St Clair Gibson & Lambert, 2005; Swann et al., 2017). Research has shown that from a physiological perspective the end-spurt is derived from additional anaerobic contribution (Corbett et al., 2012; Stone et al., 2017). Therefore, given the high rates of anaerobic energy production required throughout supramaximal exercise, and the additional requirements of the end-spurt, one hypothesized physiological limiting factor of the end-spurt is the accumulation of anaerobic metabolites (Broker, Kyle, & Burke, 1999; Corbett, 2009; Corbett et al., 2012; Noakes, St Clair Gibson & Lambert, 2005; Siegler et al, 2016). Of which, a popular metabolic proposal for fatigue is the effect of exercise-induced acidosis (Cairns, 2013; Hargreaves, 2006).

2.3 Exercise-induced acidosis and fatigue

2.3.1 Acid-base balance

Regulation of acid-base balance is critical for the maintenance of physiological function and metabolic processes. At rest pH is kept alkaline in plasma with homeostatic values between ~7.35-7.45 pH units, while muscle pH can be as low as ~7.0 pH units at rest (Atherton, 2003; Carter, 1972; Goel & Calvert, 2012; Rogers & McCutcheon, 2015; Street, Bangsbo & Juel, 2001). pH is calculated via the equation below (Hultman & Sahlin, 1980):

$$pH = - \log_{10} [H^+]$$

Put simply, the biochemical definition of an acid is a compound that donates a proton (H^+) whilst a base will instead accept a proton (Brönsted, 1923; Lowry, 1923). Although this conveys the first law of mass action: the conservation of mass, it has been highlighted that this

simplistic definition disregards two other laws (Lindinger, Kowalchuk & Heigenhauser, 2005). These disregarded laws dictate the equilibrium state between water and weak electrolytes, and the maintenance of electrical neutrality within water, and both of which determine the presence of H^+ within the physical milieu in which biochemical reactions take place (Edsall & Wyman, 1958; Harned & Owen, 1958; Van Slyke & Cullen, 1917). Accounting for these, an understanding of acid-base balance requires including the reactions that ultimately determine the H^+ and HO^- within a solution (Lindinger, Kowalchuk & Heigenhauser, 2005; Van Slyke & Cullen, 1917). Additionally, as outlined in the works of Stewart (1981) acid-base status is also determined by the independent effects of PCO_2, the total concentration of noncarbonate weak acid anions, and the strong ion difference (sum of strong cations minus the sum of the strong anions i.e. Na^+, K^+, Ca^{2+} and lactate, less chloride (Cl^-)) (Kowalchuk et al. 1988; Lindinger, 1995). Given this expanded view, it must be acknowledged that H^+ cannot be viewed as the stand-alone causative factor in changes to acid-base balance (Stewart, 1981). That being said, the greatest threats to acid-base balance are still derived from endogenous sources that contribute H^+ (Robergs, Ghiasvand & Parker, 2004).

Various endogenous intermediaries and metabolites have been found to dictate the accumulation of H^+ in both muscle and plasma, and hence a drop in both intramuscular and plasma pH (Robergs, Ghiasvand & Parker, 2004). Traditionally the accumulation of H^+ was believed to arise from the lactate dehydrogenase reaction (reducing pyruvate to lactate and H^+). However, it has been suggested that this final reaction actually consumes two H^+ (Robergs, Ghiasvand & Parker, 2004). Through retracing the glycolytic pathway an alternative hypothesis is that ATP hydrolysis, occurring at other steps along this pathway, causes an increase in H^+. Despite this new view of what contributes to the onset of acidosis, the pyruvate to lactate pathway still plays a role in the onset of acidosis. Instead of contributing H^+ as

traditionally thought, it aids in the onset of acidosis through its inability to buffer H^+ produced via ATP hydrolysis (Robergs, Ghiasvand & Parker, 2004). Although the additional hydrolysis of ATP allows the body to meet the energy demand of exercise, by exceeding the steady state capacity of the Krebs cycle and the pyruvate to lactate pathway protons begin to accumulate with the increasing exercise intensity. This initially results in a drop in intramuscular pH from ~7.0 pH units at rest to as low as 6.2 pH units post-exercise (Hermansen & Osnes, 1972; Debold, Fitts, Sundberg, Nosek, 2016; Fitts, 2016; Robergs, Ghiasvand & Parker, 2004). The subsequent drop in plasma pH is the result of transportation of H^+ and lactate through the sarcolemma and into the extracellular space, where plasma pH has been seen to fall to levels of ~6.8 pH units (Hermansen & Osnes, 1972; McNaughton & Cedaro, 1991). In an effort to control against excessive drops in both intramuscular and plasma pH several regulatory mechanisms are present within the body.

2.3.2 Mechanisms of pH regulation

Endogenous mechanisms for pH regulation generally fall within three categories of varying time courses. These include the combination of chemical buffers, pulmonary ventilation (through the carbonic anhydrase reaction) and excretion via the kidney (Hirakoba, 1999; Juel, 2008; Messonnier et al., 2007; Robergs, 2002). Buffers rapidly regulate pH as proton acceptors within muscle and plasma. Skeletal muscle buffering is dependent on three buffering processes; metabolic (i.e. PCr and glutamate), physicochemical (i.e. histidine-based proteins and dipeptides, inorganic phosphate (P_i) and HCO_3^-) and transmembrane fluxes (Hirakoba, 1999; McGinley & Bishop, 2016). It is estimated that roughly 61% of the H^+ buffered within muscle during exercise is done so through physicochemical mechanisms, while the remaining 39% is buffered by metabolic mechanisms (Hultman & Sahlin, 1980). Of the H^+ that enters plasma via transmembrane fluxes, these can be buffered by one of four plasma-based buffers, including

HCO_3^-, haemoglobin, plasma protein and phosphate (Feher, 2017; Sidebotham, McKee, Gillham, & Levy, 2007). The bicarbonate buffer system is highly effective due to its high concentration in plasma, converting H^+ to CO_2 and H_2O through the reversible reaction with carbonic anhydrase (illustrated below) (Sidebotham et al., 2007; McNaughton, Siegler & Midgley, 2008; Poupin et al., 2012):

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

The concentration of HCO_3^- in plasma is roughly 25 $mmol.L^{-1}$, while lower levels of 10 $mmol.L^{-1}$ have been measured in muscle (Costill, Verstappen, Kuipers, Janssen & Fink, 1984; Hood, Schubert, Keller & Muller, 1988; Sahlin, Alvestrand, Brandt & Hultman, 1978). Given the low HCO_3^- content within muscle and the low permeability of the sarcolemma to HCO_3^-, its effects as a buffer are more prominently seen extracellularly (Mainwood & Cechetto, 1980; Mainwood & Renaud, 1985; Sahlin, 2014). Its effectiveness as a buffer may seem questionable given that the dissociation constant (pK), a measure of the strength of an acid, of carbonic acid and HCO_3^- are 3.77 and 10.2 respectively (Robergs, 2002). Despite this the bicarbonate buffer system is a good buffer at physiological pH values between 7.2-7.4 pH units, providing up to 86% of the total extracellular buffer capacity (Poupin et al., 2012). This is due to the influence of dissolved and gaseous CO_2 in the lungs and blood on both H_2CO_3 and HCO_3^-, altering the pK of the bicarbonate buffer system and bringing it closer to plasma pH of 7.4 pH units.

A second regulatory mechanism is the removal of intramuscular protons by means of transmembrane fluxes (Feher, 2017; Hirakoba, 1999; McGinley & Bishop, 2016). In this case various transporters attenuate the decline of intramuscular pH by providing rapid translocation of H^+ across the sarcolemma (Gough, 2018; Halestrap & Wilson, 2012). Research manipulating these transporters has identified that the primary systems associated with the extrusion of H^+ under intense exercise are the Na^+-H^+ exchange (Juel, 1998; Mason & Thomas, 1988;

Westerblad & Allen, 1992). In addition, the role of MCT's in regulation of H^+ is gaining prominence (Brooks, 2010; Messonnier et al., 2007; Kitaoka, Hoshino & Hatta, 2012). Being concentration dependent, these transporter proteins rely on saturation by accumulated H^+ to establish a pH gradient in order to promote the transport of both lactate and H^+ across the sarcolemma. The primary MCT, MCT4, facilitates the extrusion of lactate out of muscle (Geers & Gros, 2000, Juel, 1995; Juel 1998; Kitaoka, Hoshino & Hatta, 2012). Although the body is generally able to uphold acid-base balance through these regulatory mechanisms, it is speculated that a critical threshold for these exists, above which fatigue ensues (Amann, 2011; Neyroud, Kayser & Place, 2016). Therefore, with the high anaerobic requirements and the ensuant drops in both intramuscular and plasma pH seen in response to supramaximal exercise, the onset of extreme acidosis is an obvious candidate for fatigue in this case.

2.3.3 Overview of fatigue

Performance is determined by the maximal power that can be sustained throughout exercise. This is not only dependent on the rate of energy one can produce, but also the impairment of muscle force or power through the integrative process of fatigue (Weyand, Lin and Bundle, 2005). Fatigue is defined as the decline of muscle force or power output leading to reduced performance (Cairns, 2006b; Green, 1997; Place et al., 2010). Mechanisms eliciting fatigue can be split into two primary branches. The first is central fatigue which involves a diminished motor drive to the periphery as dictated by the central nervous system (Place et al., 2010; Shei & Mickleborough, 2013). The mechanism underpinning central fatigue is not clear. One proposal is the reduced central motor drive dictated by group III and IV muscle afferents when metabolic perturbations exceed a specific critical threshold of high-energy phosphate compounds or metabolites (i.e. PCr, P_i, ADP and H^+) (Amann & Calbet, 2008; Burnley,

Vanhatalo, Fulford & Jones, 2010; Hogan, Richardson & Haseler, 1999; Vanhatalo, Fulford, DiMenna & Jones, 2010). Another central mechanism associated with fatigue at both altitude and under highly anaerobic conditions is hypoxemia or changes to the oxygen saturation of haemoglobin (Thomas & Lumb, 2012). The degree of hypoxemia is shown to severely impact cerebral oxygenation and in turn reduce central drive (Nielsen et al., 2002; Romer, Haverkamp, Lovering, Pegelow & Dempsey, 2006; Subudhi et al., 2011; Vogiatzis et al., 2011). Thirdly, the relationship between fatigue and the decreased concentration of dopamine and accumulation of serotonin (5-hydroxytryptamine, or 5-HT) in the brain (Bailey, Davis & Ahlborn, 1993). Finally, an alternative central governor model, which views the brain as a feedforward central controller preventing catastrophic failure (Noakes 2012, Noakes 2011). In this context the brain interprets the sensations and feedback from the periphery during exercise, modulating effort in order to maintain homeostasis. However, the validity of this model is yet to be fully tested and remains an area of debate (Cairns, 2011; Robergs, 2017).

Peripheral fatigue refers to exercise-induced reductions to contractile performance occurring within the working muscle (Place et al., 2010; Sandiford et al., 2004; Shei & Mickleborough, 2013). Peripheral fatigue manifests at several sites including decreased Ca^{2+} release from the sarcoplasmic reticulum and the reduction of actin-myosin cross-bridge formation (Fabiato & Fabiato, 1978; Fitts, 2008; Westerblad & Allen, 1991; Cairns, Westerblad & Allen, 1993b), or action potential changes in the sarcolemma or transverse-tubular membranes (Green, 1997; Cairns & Lindinger, 2008). Factors eliciting such fatigue could include lowered substrate availability through reduced muscle ATP, PCr and glycogen levels (Allen, Lamb & Westerblad, 2008; Barnes, Gibson & Stephenson, 2001; Casey, Constantin-Teodosiu, Howell, Hultman & Greenhaff, 1996; Dutka & Lamb, 2007; Karatzaferi, de Haan, Ferguson, van Mechelen & Sargeant, 2001; McLester , 1997; Sahlin, Tonkonogi & Söderlund, 1998), large reductions in trans-sarcolemma K^+ gradient (Cairns et al., 1997; Clausen, 2003; McKenna et

al., 2006; Juel, 1986), the accumulation of reactive oxygen species (ROS) (Edwards, Macdonald, van der Poel & Stephenson, 2007; Ferreira & Reid, 2008; Westerblad et al., 1998), and finally raised metabolites such as P_i, ADP, lactate and H^+ in muscle and plasma (Giannesini et al., 2001; McLester Jr, 1997; Jubrias, Crowther, Shankland, Gronka & Conley, 2003; Karatzaferi, Franks-Skiba & Cooke, 2008; Westerblad, Allen & Lännergren, 2002). Of these the most prominent in discussions by sports scientists and coaches alike has been the concept of lactic acid, or more specifically lactate and H^+ (Brooks, 2018; Daniels, 2005; Hill, Long & Lupton, 1924; Livingstone, 2009).

2.3.4 Acidosis' role in fatigue

The exercise-induced acidosis associated with the glycolytic pathway, often referred to as "lactic acidosis", has been a part of the fatigue vernacular since the early 20th century (Fletcher & Hopkins, 1906; Hill, Long & Lupton, 1924). However, research has provided several arguments against the central tenants associating lactic acid, and primarily lactate, to fatigue (Brooks, 2018; Cairns, 2006a; Cairns, 2006b; Fitts, 2016; Gladden, 2004; Pate, Bhimani, Franks-Skiba & Cooke, 1995; Robergs, Ghiasvand & Parker, 2004). Firstly, it has been shown that the accumulation of lactate in blood and tissue is not necessarily due to states of anoxia (Connett, Gayeski & Honig, 1986; Linnarsson, Karlsson, Fagraeus & Saltin, 1974; Richardson, Noyszewski, Leigh & Wagner, 1998). Secondly, studies examining the temporal relationship between lactate, acidosis and fatigue identified originally by Fitts and Holloszy (1976) have shown declines in force and acid-base balance deviate during both exercise and a post-exercise recovery period (Costill et al., 1983; Degroot et al., 1993; Hermansen & Osnes, 1972; Juel et al., 2004; Sahlin & Ren 1989; Saugen et al., 1997; Street, Bangsbo & Juel, 2001; Vøllestad et

al., 1988; Westerblad & Allen, 1992). Finally, it has also been shown that as a compound lactic acid does not exist at physiologically relevant temperatures (Toffaletti, 1991).

With this separation of lactate and H^+ attention has turned to the separate examination of each for their role in fatigue. Over the past four decades the understanding of lactate has progressed from a mechanism of fatigue to now being viewed as a metabolic substrate and signalling molecule (Brooks, 2018). This understanding has been expanded upon by the discovery of an intra- and intercellular lactate shuttle and the presence of lactate dehydrogenase in mitochondria, enabling lactate oxidation (Brooks, 1986a; Brooks, 1986b; Brooks, Dubouchaud, Brown, Sicurello & Butz, 1999). Alternatively, the debate over acidosis as a fatigue agent appears more convincing (Fitts, 2016; Westerblad, 2016). Predominantly conducted intracellularly using animal models, research has provided several proposals for acidosis' role in eliciting both peripheral and central fatigue. Peripherally, the rise in H^+ has been shown to independently inhibit Ca^{2+} release in the sarcoplasmic reticulum and Ca^{2+} sensitivity of the myofilaments reducing shortening velocity (Fabiato & Fabiato, 1978; Favero et al., 1995; Jarvis, Woodward, Debold & Walcott, 2018; Knuth, 2000; Lännergren & Westerblad, 1991; Westerblad & Allen, 1991). Acidosis has also been shown to hamper energy supply through downregulation of glycolytic enzymes (Hollidge-Horvat et al., 1999), inhibit oxidative phosphorylation (Jubrias et al., 2003) and induce dysfunction by increasing cell volume (Bressler & Matsuba, 1991; Lindinger, 2005; Rapp et al., 1998). Alternatively, others have shown acidosis to induce fatigue via interaction with other physiological variables. Firstly, an interaction with P_i has been shown to inhibit myosin function through Ca^{2+} de-sensitivity, impacting shortening velocity and force production (Karatzaferi, Franks-Skiba & Cooke, 2008; Nelson, Debold & Fitts, 2014; Woodward & Debold, 2018). Another interaction is with increased K^+ efflux which is proposed to elicit fatigue via inhibition of ATP-sensitive K^+ channels (Street, Nielsen, Bangsbo & Juel, 2005). While from a central

fatigue perspective, acidosis has been shown to reduce central motor drive via arterial de-oxygenation (Nielsen et al., 2002) and stimulation of group III and IV muscle afferents (Amann, 2011; Blain, 2016).

Several counterarguments have also arisen to provide putative evidence suggesting the appearance of acidosis could actually combat fatigue (Westerblad, 2016). Firstly, researchers have found that addition of lactic acid to K^+ depressed muscle fibres accelerated recovery and restored force (Nielsen, De Paoli & Overgaard, 2001; Pedersen, Nielsen, Lamb & Stephenson, 2004; Pedersen, De Paoli & Nielsen, 2005). To do so the rise in H^+ has been suggested to block chloride channels in the transverse-tubular system, making it possible for action potentials to propagate despite inactivation of Na^+ channels by K^+ depression (Pedersen, Nielsen, Lamb & Stephenson, 2004; Pedersen, De Paoli & Nielsen, 2005). However, one critique of this interpretation is that in these experiments the muscle was depressed prior to the addition of lactic acid which is not physiologically representative of exercise, where K^+ efflux and the onset of acidosis occur simultaneously (Aronson & Giebisch, 2011). Other researchers have proposed that acidosis increases oxygen delivery to vital organs. This is accomplished in part by acidosis stimulating the offloading of oxygen from haemoglobin via the Bohr effect (Arieff, 1991; Sahlin, 1983; Thomas, & Lumb, 2012). Alternatively, the onset of acidosis has been viewed as a signalling mechanism causing increases in local vasodilation via activation of smooth muscle which alters muscle and cerebral circulation (Yoon, Zuccarello & Rapoport, 2012), or by causing increased ventilation in an effort to support the removal of CO_2 (Meyer, Faude, Scharhag, Urhausen & Kindermann, 2004; Ortiz-Acevedo, Rigor, Maldonado & Cala, 2009).

Human studies have also provided evidence supporting the detrimental effects of acidosis in inducing fatigue. Although intramuscular research using human muscle does exist, it is limited.

Of the few studies, Sundberg, Hunter, Trappe, Smith & Fitts (2018) showed that exposing isolated muscle fibres from the vastus lateralis to low levels of pH (6.2 pH units) and high levels of P_i (30 mmol.L^{-1}) additively inhibited cross-bridge cycling and peak power. Despite the lack of intramuscular research the role of acidosis been explored within humans through the manipulation of plasma acidosis. In these cases manipulation has been achieved via exogenous NH_4Cl (inducing plasma acidosis) or exogenous $NaHCO_3$ and sodium citrate (inducing plasma alkalosis). Of the two conditions (acidosis and alkalosis) the negative effects on exercise performance of plasma acidosis via NH_4Cl appear to be more pronounced (Brien, 1982; Correia-Oliveira et al., 2017; Hultman, Del Canale & Sjöholm, 1985; Jones, Sutton, Taylor & Toews, 1977; Robergs, Hutchinson, Hendee, Madden & Siegler, 2005; Sutton, Jones & Toews, 1975). In addition to these reductions in exercise performance eliciting a plasma acidosis via NH_4Cl also reduced plasma [HCO_3^-] lowered peak blood [La^-] at exhaustion. The negative impacts of plasma acidosis on exercise and muscle performance within humans highlights the importance of plasma H^+ regulation in fatigue. With this in mind, the use of $NaHCO_3$ to alternatively induce plasma alkalosis has instead been shown to enhance performance on many occasions.

2.4 Sodium bicarbonate supplementation to modulate plasma acidosis

2.4.1 Mechanisms

Although $NaHCO_3$ supplementation has been utilized acutely in exercise performance research since the first works of the Harvard fatigue researchers in 1931, the mechanisms behind its proposed ergogenic effects have been heavily discussed but are yet to be fully elucidated (Dennig, Talbott, Edwards, & Dill, 1931; McNaughton et al., 2016; Siegler et al., 2016). Although researchers have shown an ability for $NaHCO_3$ to affect intracellular mechanisms, given that the sarcolemma is largely impermeable to HCO_3^- the two popular mechanistic

proposals for $NaHCO_3$ exist extracellularly. These include an increased plasma buffer capacity for H^+ and an improved pH gradient enhancing transmembrane efflux of H^+ (Granier et al., 1996; Heibel, Perim, Oliveira, McNaughton & Saunders, 2018; Mainwood & Cechetto, 1980; Mainwood & Renaud, 1985; Messonnier et al., 2007; Nagesser, Van Der Laarse, & Elzinga, 1994; Raymer et al., 2004; Roos, 1975; Sahlin, 2014). In addition to these two prominent mechanistic proposals, several other proposals exist. First of which is the speculation that $NaHCO_3$ may affect the balance of strong ions, improving motor pathways and muscle function (Raymer et al, 2004; Sostaric et al., 2006; Street, Nielsen, Bangsbo & Juel, 2005, Broch-Lips, Overgaard, Praetorius & Nielsen, 2007). Another proposal is a greater glycolytic ATP production arising from higher plasma and muscle lactate concentrations seen at the same exercise intensity in $NaHCO_3$ conditions (Bouissou, Defer, Guezennec, Estrade & Serrurier, 2003; Hollidge-Horvat, 2000; Spriet, Lindinger, Heigenhauser & Jones, 1986; Raymer, Marsh, Kowalchuk & Thompson, 2004). However, very little research has attempted to quantify the anaerobic and aerobic energy yields during exercise with $NaHCO_3$ (Berger, McNaughton, Keatley, Wilkerson & Jones, 2006; Kolkhorst, Rezende, Levy & Buono, 2004; Soladz, Szkutnik, Krzysztof, Majerczak & Korzeniewski, 2004). Yet, several groups have attempted to dive deeper into this enhanced lactate efflux, attributing it more to an altered pH gradient and enhanced transmembrane transport than an increase in glycolytic activity (Hollidge-Horvat et al., 2000; Raymer et al., 2004; Roos, 1975). Raymer, Marsh, Kowalchuk and Thompson (2004) found no difference in the total measurement of the intracellular accumulation of lactate, H^+ or P_i post-exercise relative to a placebo condition. In this study differences did exist between conditions in the delayed onset of intracellular pH and high-energy phosphate kinetics (illustrated by the ratio of P_i to PCr) however. This therefore suggests that altered energy contribution may come from this increased rate of high-energy phosphate reactions due to the enhancement of H^+ transport (Raymer et al., 2004).

2.4.2 Loading procedure

Given the two primary mechanisms identified to elicit $NaHCO_3^-$ ergogenic effects (i.e. enhanced plasma buffer capacity and increased pH gradient) a significant increase in plasma [HCO_3^-] is required. As a measure of initial plasma changes in response to $NaHCO_3$ ingestion, plasma [HCO_3^-] has been shown to be the most reproducible compared with plasma pH (de Araujo Dias et al., 2015; Gough et al. 2017). Given this, several researchers have highlighted the need for a plasma increase of 6 $mmol.L^{-1}$ post-ingestion for erogenicity (Bishop & Claudius, 2005; Carr et al., 2011; McNaughton & Cedaro, 1991; Van Montfoort et al., 2004; Wilkes et al., 1983). Although some have cautioned this requirement, showing that increases in plasma [HCO_3^-] and performance are weakly associated (Matson & Tran, 1993). Additionally, it does also appear that at the same relative doses an interindividual variability in plasma [HCO_3^-] response does exist (Gough, 2017). Nevertheless, evidence has established several additional recommendations for effective loading of $NaHCO_3$ prior to exercise.

Recommendations focused on eliciting large enough plasma [HCO_3^-] changes have concentrated on dosage and timing. Looking specifically at the dosages required to elicit the desired changes in plasma [HCO_3^-] research has explored a range of doses from 0.1-0.5 $g.kg^{-1}$ BM (Carr, Hopkins & Gore, 2011; Matson & Tran, 1993; McNaughton, Siegler & Midgley, 2008; McNaughton et al., 2016). From this research it has been found that a minimum dose of at least 0.2 $g.kg^{-1}$ BM is required for erogenicity (Ferreira et al., 2019; Gough et al., 2018; McNaughton, 1992), while doses of 0.1 $g.kg^{-1}$ BM do not appear to be high enough to elicit a response (Ferreira et al., 2019; McNaughton, 1992). Despite a dosage of 0.2 $g.kg^{-1}$ BM being shown to be sufficient to elicit a plasma and ergogenic response, the most commonly used dosage remains 0.3 $g.kg^{-1}$ BM (Carr, Hopkins & Gore, 2011; Christensen et al., 2017; Matson

& Tran, 1993). Research has shown this dosage improves performance relative to lower doses, while reducing gastric discomfort compared to higher doses (Ferreira et al., 2019; Ferreira, De Camargo Smolarek & Utter, 2015; Gough et al., 2018; Jones et al., 2016; McNaughton, 1992; Matson & Tran, 1993). However, it is still cautioned as to whether this is the optimal amount across all exercise modalities and participants, with considerable variation in erogenicity existing within modalities and training status (Carr, Hopkins & Gore, 2011; Matson & Tran, 1993; McNaughton, Siegler & Midgley, 2008; McNaughton et al., 2016; Requena, Zabala, Padial & Feriche, 2005; Siegler et al., 2016).

Concerning timing between ingestion and initiation of exercise a great deal of variety exists in research. Some researchers have shown time frames of 60-90 min are required post-ingestion for significant changes in plasma [HCO_3^-] and plasma pH and HCO_3^- to occur (Renfree, 2007; Price & Singh, 2008; Siegler et al., 2010). While others have suggested 120-150 min to induce substantial plasma changes and reduce the symptoms of gastric discomfort (Carr et al., 2011). Studies have also shown that a large interindividual variability exists in the time to peak plasma [HCO_3^-] and pH, ranging from 10-120 min with extreme coefficients of variation of 29% between individuals using standard dosages (Carr et al., 2011; Sparks et al., 2017). To account for this variability, some have called for the use of timing protocols to be based on individualized time to peak plasma [HCO_3^-] (McNaughton et al., 2016; Siegler et al., 2016). Such an approach has been shown promising results across repeated-sprint protocols (Deb, Gough, Sparks, & McNaughton, 2018; Miller et al., 2016) and supramaximal time-trials (Gough et al., 2018). However, such individualized strategies have not been able to fully address the problems that prompted their use in the first place. Some studies have failed to see adequate rises in plasma [HCO_3^-] (Miller et al., 2016), while others have not achieved consistent erogenicity across all participants (Deb et al., 2018). One final critique is that current research has lacked a comparison to traditional timing recommendations, impacting their

validity as the optimal strategy. It is clear that additional research in the area of individualised time to peak protocols is required.

Another consideration includes gastric discomfort. One researcher has proposed that the occurrence of gastric discomfort may help explain variability in ergogenic response to $NaHCO_3$. In this case, Saunders, Sale, Harris and Sunderland (2014) found that by removing participants who experienced moderate to severe gastric discomfort from analysis the total work done by participants during a supramaximal time-to-exhaustion trial was significantly greater than a placebo condition. Although these overall results seem promising, closer examination of individual responses still showed that several participants still failed to improve performance compared to placebo, despite not experiencing any gastric discomfort. Although the results of Saunders and colleagues are not conclusive, gastric discomfort still remains a factor that must be accounted for and hopefully avoided. Generally associated with mild to severe cases of osmotic diarrhoea, bloating, nausea, belching and even headaches, several researchers have highlighted it as a cause of participant drop out (Carr, Slater, Gore, Dawson & Burke, 2011; Gough et al., 2017; Jones et al., 2016; Kahle, Kelly, Eliot & Weiss, 2013; Mohr, 2017; Saunders, Sale, Harris & Sunderland, 2014). A number of tactics have been employed to minimise these negative implications including co-ingestion with food, the use of gelatine capsules for oral delivery, or the spacing out of an absolute dose into smaller stacked segments (Carr et al., 2011; Jones et al., 2016; Sale et al., 2011; Saunders et al., 2014). Such tactics remain an essential consideration in any study looking to minimise participant drop out due to gastric discomfort.

2.4.3 Sodium bicarbonate ingestion and supramaximal exercise performance

2.4.4.1 Consistent ergogenic effects within well-trained cyclist over time-trials of 1-6.5 min

Research exploring the role of $NaHCO_3$ and exercise performance has shown inconsistent responses during supramaximal exercise. This is despite several meta-analyses identifying a small to moderate ergogenic effect (Carr, Hopkins & Gore, 2011; Christensen et al., 2017; Matson & Tran, 1993; Peart, Siegler & Vince, 2012). Some have highlighted publication bias in the early years of research as rationale for several meta-analyses showing an aggregated ergogenic effect, however an increase of more recent studies questioning its efficacy have potentially helped provide a more accurate representation (Christensen et al., 2017; Peart, Siegler & Vince, 2012). With no differences arising between the loading protocols used across research (i.e. dosage and timing) researchers have combined the findings to help elucidate areas where a more consistent ergogenic effect exists. From this it does appear that a consistent beneficial performance effect exists amongst well-trained cyclists over time-trial protocols between 1-6.5 min (Bellinger, Howe, Shing & Fell, 2012; Driller, Gregory, Williams & Fell, 2012; Gough et al., 2018; Gough et al., 2017; Kilding, Overton & Gleave, 2012; Thomas et al., 2016).

Several reviews have acknowledged an influence of training status on erogenicity, although this has also been controversial (Carr, Hopkins & Gore 2011; McNaughton et al., 2016; Peart, Siegler & Vince, 2012). Instead the modality of exercise may be a confounding factor. Several direct within study comparisons have been made between trained and untrained participants using running and cycling. Running is the first case in which studies over supramaximal time-trials of ~60 s to 2 min have shown a consistent benefit with well-trained subjects (Goldfinch, McNaughton & Davies, 1988; Kindermann, Keul & Huber, 1977; Wilkes, Gledhill & Smyth, 1983; McKenzie, 1988). Additionally, over a 3-min time-trial it was also shown that

well-trained cyclists appeared to benefit from $NaHCO_3$ loading, while another study of untrained participants did not (Deb et al., 2017; Vanhatalo, McNaughton, Siegler & Jones, 2010). Alternatively, an effect of training appears to be less apparent in other exercise modalities. Rowing and swimming studies have provided the most thorough examination of elite and well-trained participants across studies. Within the context of rowing the majority of studies have shown no ergogenic effect in time-trial performances of 2 min and 2000 m (Brien, 1982; Carr, Gore & Dawson, 2011; Carr, Slatter, Gore & Burke, 2012; Hobson et al., 2014; Kupcis, Slater, Pruscino & Kemp, 2012). On the other hand, studies examining swimming have provided mixed results. Some have shown overall improvements in 200-m time-trial (Lindh, Peyrebrune, Ingham, Bailey & Folland, 2008), overall time during repeated-sprints (Siegler & Gleadall-Siddall, 2010), and later into repeated-sprint protocols (Gao, Costill, Horswill & Park 1988; Mero et al., 2013). However, other researchers have seen no ergogenic effect over varied distances from 100 m (~60 s) to 400 m (~4.5 min) (de Salles Painelli et al., 2012; Kumstát, Hlinský, Struhár & Thomas, 2018; Pierce, Eastman, Hammer & Lynn. 1992). With no apparent difference between studies with regards to duration of exercise, dosing protocols or even the changes in plasma pH or $[HCO_3^-]$ before or after exercise there is little to explain the differences in these exercise modalities. This aside, the initial suggestion that a training effect exists within cycling does appear consistent.

When examining cycling studies conducted solely on well-trained participants, the ergogenic effects appear more apparent over close-ended time-trials of ~1-6.5 min. McNaughton (1992b) looked at performance over varying durations from 10-240 s with significant improvements in total work and PPO seen over the longer duration bouts (120 and 240 s). Sung-Gye, Dong-Sik, Sang Chul and In-Ho (1990) similarly showed performance improvements in competitive cyclists during $NaHCO_3$ conditions over a longer 3000-m time-trial compared with a 1000-m

time-trial. These findings have been supported by several studies of time-trials over durations of 70 s to ~ 6.5 min (Bellinger, Howe, Shing & Fell, 2012; Driller, Gregory, Williams & Fell, 2012; Thomas et al., 2016) and distances of 3-4 km (Gough et al., 2018; Gough et al., 2017; Kilding, Overton & Gleave, 2012). Although several studies using well-trained participants have questioned the existence of a consistent ergogenic effect, their results may be explained by an inability to achieve the desired plasma changes prior to testing (Callahan, Parr, Hawley & Burke, 2017; Horswill et al., 1988; Linderman, Kirk, Musselmanm Dolinar & Fahey, 1992).

Although an ergogenic effect appears more consistent over close-ended time-trials of greater than 60 s, the effect of training is not as apparent in repeated-sprint cycling (Peinado et al., 2019; Wijen, Verstappen & Kuipers, 1984; Zabala et al., 2008; Zabala et al., 2011). Generally using shorter duration repetitions of 60 s or less, these studies have shown no consistent effect of $NaHCO_3$ in improving performance. Due to the short durations little decrease in pH is seen over the initial repetitions, corresponding to a lack of performance differences early on. However, this initial lack of change may mask performance differences seen later into protocols when overall performance data is considered. In healthy active populations several researchers have highlighted that benefits may be present later into protocols when larger reductions in plasma pH are present (Bishop et al., 2004; Lavender & Bird, 1989; Mündel, 2018; Price, Moss & Rance, 2003). Although not consistent across all studies these findings are supported by several studies using repeated-sprint exercise protocols in trained cohorts (Griffen, Rogerson, Ranchpordas & Ruddock, 2015; Zinner at al., 2010). Taken together these findings provides rationale for $NaHCO_3$ use in resisting fatigue and the ability to sprint later into competition.

Table 2. 1. Studies investigating sodium bicarbonate supplementation in well-trained and elite cohorts in cycling, rowing, running and swimming.

Authors	Year	Training background	Gender	Modality	Intensity	Distance/duration	Protocol	Performance measures			
Gough, Brown, Deb, Sparks & McNaughton	2018	Trained cyclists	Males	Cycling	Maximal	2 x 4000 m with 40 min passive recovery	Repeated sprint	Performance time: Time trial 1: ↑ Time trial 2: ↑	MPO: Time trial 1: ↑ Time trial 2: ↑	The decline in performance from time trial 1 to time trial 2: ↔	
Griffen, Rogerson, Ranchordas & Ruddock	2015	Well-trained	Males	Cycling	Maximal	6 x 10 s with 1 min active recovery	Repeated sprint	MPO: ↑	TWD: ↑	Fatigue Index: ↑	
Peinado et al.	2019	Elite BMX riders	Males	Cycling	Maximal	3 x 400 m BMX races	Repeated sprint	Race time: ↔ Peak velocity: ↔ Time to peak velocity: ↔			
Zabala et al.	2008	International BMX riders	-	Cycling	Maximal	3 x 30 s Wingates with 30 min rest between	Repeated sprint	Time to PPO: ↔	MPO: ↔	PPO: ↔	Fatigue index: ↔
Zabala et al.	2011	Elite BMX riders	Males	Cycling	Maximal	3 x 30 s Wingates with 15 min recovery	Repeated sprint	MPO Wingate 1: ↔ Wingate 2: ↔ Wingate 3: ↔	PPO Wingate 1: ↔ Wingate 2: ↔ Wingate 3: ↔	Fatigue Index: Wingate 1: ↔ Wingate 2: ↔ Wingate 3: ↔	
Zinner at al.	2010	Well-trained cyclists	Males	Cycling	Maximal	4 x 30 s Wingates	Repeated sprint	MPO Rep 1: ↔ Rep 2: ↔ Rep 3: ↑ Rep 4: ↑	PPO Rep 1: ↔ Rep 2: ↔ Rep 3: ↔ Rep 4: ↔		
Wijnen, Verstappen & Kuipers	1984	Healthy well-trained	Males	Cycling	125% pVO_2peak	5 x 60 s sprints with 1min rest period with the 5th rep a time-to-exhaustion	Repeated sprint - with the last rep a time trial to exhaustion	Time-to-exhaustion: ↔			

da Silva et al.	2019	Trained cyclists	Males	Cycling	Repeated sprints completed at 110% $p\dot{V}O_2peak$	4 x 60 s and then 30-kJ time trial	Repeated sprint and time trial	Time to complete the 30-kJ performance test: ↭		
Bouissou, Defer, Guezennec, Estrade & Serrurier	2003	Well-trained healthy, varsity track athletes	Males	Cycling	125% $p\dot{V}O_2peak$		Time-to-exhaustion	Time-to-exhaustion: ↑		
Ferreira et al.	2019	Trained cyclists	-	Cycling	Resistance of 1 kg + 5% body mass		Time-to-exhaustion	Time-to-exhaustion: ↑ (only in 0.3 but not 0.1g.kg^{-1} BM)		
Linderman, Kirk, Musselman, Dolinar & Fahey	1992	Well-trained	Males	Cycling	100% $p\dot{V}O_2peak$		Time-to-exhaustion	Time-to-exhaustion: ↭		
Robergs et al.	2005	Trained cyclists	Males	Cycling	110% $p\dot{V}O_2peak$		Time-to-exhaustion	Time-to-exhaustion: ↭		
Bellinger, Howe, Shing & Fell	2012	Highly-trained cyclists	Males	Cycling	Maximal	4 min	Time trial	MPO: ↑		
Callahan, Parr, Hawley & Burke	2017	Well-trained cyclists	Males	Cycling	Maximal	4000 m	Time trial	Time to complete: ↭	MPO: ↭	
Deb, Gough, Sparks & McNaughton	2017	Trained cyclists	Males	Cycling	Maximal	3 min	Time trial	Critical power: ↑	PPO: ↭	TWD: ↑
Driller, Gregory, Williams & Fell	2012	Well-trained cyclists	Males	Cycling	Maximal	4 min	Time trial	MPO: ↑		
Driller, Williams, Bellinger, Howe & Fell	2012	Well-trained cyclists	Males	Cycling	Maximal	2 min	Time trial	MPO: ↑	PPO: ↭	
Gough, Deb, Sparks & McNaughton	2017	Trained cyclists	Males	Cycling	Maximal	4000 m	Time trial	Time to complete: ↑		

Gough, Deb, Sparks & McNaughton	2018	Well-trained cyclists	Males	Cycling	Maximal	4000 m	Time trial	Time to complete: ↑	MPO: ↑	
Horswill et al.	1988	Endurance trained cyclists	Males	Cycling	Maximal	2 min	Time trial	TWD: ↭		
Kilding, Overton & Gleave	2012	Well-trained cyclists	Males	Cycling	Maximal	3000 m	Time trial	Total time: ↑	MPO: ↑	
McNaughton	1992b	Active - Yet, identified as having mean $\dot{V}O_2$peaks in excess of 5.19 $L.min^{-1}$	-	Cycling	Maximal	10 s, 30 s, 120 s and 240 s	Time trial	PPO 10 s: ↭ 30 s: ↭ 120 s: ↑ 240 s: ↑	TWD: 10 s: ↭ 30 s: ↭ 120 s: ↑ 240 s: ↑	
Sung-Gye, Dong-Sik, Sang Chul & In-Ho	1990	Competitive high-school cyclists	-	Cycling	Maximal	1000 m & 3000 m	Time trial	Performance time: 1000 m: ↭ 3000 m: ↑		
Thomas et al.	2016	Well-trained cyclists (sprint and BMX)	Males & females	Cycling	Maximal	70 s	Time trial	MPO at 20s: ↭ at 50s: ↑ Total: ↑	Fatigue Index: ↭	
Brien	1982	Elite rowers	-	Rowing	Maximal	2 min	Time trial	TWD: ↭	MPO: ↭	
Carr et al.	2012	Well-trained rowers	Males & females	Rowing	Maximal	2000 m	Time trial	Time trial 1: ↭ Time trial 2: ↭		
Carr, Gore & Dawson	2011	Well-trained rowers	Males & females	Rowing	Maximal	2000 m	Time trial	Total time: ↭	MPO: ↭	Highest average power over 500m: ↭
Christensen et al.	2014	Elite rowers	-	Rowing	Maximal	6 min	Time trial	Total distance for lightweight rowers: ↭ Openweight rowers: ↓	MPO for all rowers: ↭	

Kupcis, Slater, Pruscino & Kemp	2012	Highly-trained rowers	Males	Rowing	Maximal	6.5 min	Time trial	Time to complete: ↭	MPO: ↭
McNaughton & Rod	1991	Highly-trained	Males	Rowing	Maximal	6 min	Time trial	Mean distance travelled: ↑	
Ducker, Dawson & Wallman	2013	Competitive team sports athletes	Males	Running	Maximal	3 sets of 6 x 25 m departing every 25 s, with 4 min recovery between sets	Repeated sprint	Total sprint time: ↑	Mean 20 m sprint times each set: Rep 3-6: ↑ Rep 7-12: ↑ Rep 13-18: ↑
Stöggl, Torres-Peralta, Cetin & Nagasaki	2014	Well-trained	Males	Running	19 $km.hr^{-1}$ at a 5% incline	3x time-to-exhaustion runs of ~2 min separated by 25mins	Repeated bouts of time-to-exhaustion	Time-to-exhaustion: Bout 1: ↭ Bout 2: ↭ Bout 3: ↭	
Van Montfoort et al.	2004	Trained distance runners	Males	Running	19-23 $km.hr^{-1}$		Time-to-exhaustion	Time-to-exhaustion: ↑	
Bird, Wiles & Robbins	2008	National-standard middle-distance runners	Males	Running	Maximal	1500 m	Time trial	Performance time: ↑	
Goldfinch, McNaughton & Davies	1988	Trained	Males	Running	Maximal	400 m	Time trial	Performance time: ↑	
Wilkes, Gledhill & Smith	1983	Trained middle-distance runner	Males	Running	Maximal	800 m	Time trial	Performance time: ↑	
Gao, Costill, Horswill & Park	1988	Well-trained college swimmers	Males	Swimming	Maximal	5 x 91.4 m with 2 min rest between	Repeated sprint	Performance time: Rep 1: ↭ Rep 2: ↭ Rep 3: ↭ Rep 4: ↑ Rep 5: ↑	
Mero et al.	2013	Competitive swimmers	Males	Swimming	Maximal	2 x 100 m with 12 min rest between	Repeated sprint	Performance time: Rep 1: ↭ Rep 2: ↑	Difference in performance time from rep 1 to rep 2: ↑

Pierce, Eastman, Hammer & Lynn	1992	Varsity swimmers	Males	Swimming	Maximal	1 x 91.4 m and 2 x 182.8 m with 20 min rest between	Repeated sprint	Performance time: Single 91.4 m: ⥊ Repeated 182.8 m: ⥊	
Pruscino, Ross, Gregory, Savage & Flanagan	2008	Elite swimmers	Males	Swimming	Maximal	2 x 200 m with 30 min rest between	Repeated sprint	Performance time: Rep 1: ⥊ Rep 2: ⥊	Difference in performance time from rep 1 to rep 2: ⥊
Siegler & Gleadall-Siddall	2010	Trained swimmers	Males and females	Swimming	Maximal	8 x 25 m with 5 s rest between	Repeated sprint	Total time: ↑	
Zajac et al.	2009	Well-trained youth swimmers	Males	Swimming	Maximal	4 x 50 m with 1 min rest between	Repeated sprint	Time to complete protocol: ↑	Swim speed: Rep 1: ↑ Rep 2: ⥊ Rep 3: ⥊ Rep 4: ⥊
de Salles Painelli et al.	2012	Competitive swimmers	Males and females	Swimming	Maximal	100 and 200 m	Time trial	Performance time: 100 m: ⥊ 200 m: ⥊	
Joyce, Minahan, Anderson & Osborne	2011	Highly trained swimmers	Males	Swimming	Maximal	200 m	Time trial	Mean performance time: Trial 1: ⥊ Trial 2: ⥊	
Kumstát, Hlinský, Struhár & Thomas	2018	Nationally ranked swimmers	Males	Swimming	Maximal	400 m	Time trial	Performance time: ⥊	
Lindh, Peyrebrune, Ingham, Bailey & Folland	2008	Elite swimmers	Males	Swimming	Maximal	200 m	Time trial	Performance times: ↑	

Table ordered by modality and then exercise protocols. Abbreviations: PPO = Peak power output; MPO = Mean power output; TWD = Total work done; Rep = Repetition; ⥊ = No significant performance difference ($P \geq 0.05$); ↑ = Significant improvement in performance ($P < 0.05$); ↓ = Performance significantly worse ($P < 0.05$); Fatigue index: Decline in power output divided by the time interval between the peak power and minimum power (Kent, 2006).

2.4.4.2 Lack of ecological validity in current performance research using sodium bicarbonate

Research using $NaHCO_3$ to study performance may be limited in its application to real world competition as research has mainly used three types of exercise protocol. These include fixed-intensity time-to-exhaustion trials, close-ended time-trials defined by set durations or distances, and repeated-sprints. A benefit of utilizing these protocols is they separately assess the effects of duration, intensity and fatigability on performance outcomes. However, these protocols have been suggested to lack ecological validity because they do not replicate the psychological challenge, racing constraints or pacing strategies of competition (Ansley, Lambert, Scharbort, St Clair Gibson & Noakes, 2004; Amann, Hopkins & Marcora, 2008; Christensen et al., 2017; Faria, Parker, & Faria, 2005; Marino, Gard & Drinkwater, 2011; Schimpchen, Skorski, Nopp & Meyer, 2016). One component of supramaximal competition is the end-spurt or the ability to rapidly produce peak powers in order to separate from the competition over the final stages of a race (Corbett, 2009; Corbett et al., 2012). With the end-spurt shown to be dictated by increased anaerobic ATP contribution research into the area of the end-spurt could provide a more specific application of $NaHCO_3$ in a performance context (Corbett et al., 2012; Siegler, 2016).

To date no studies have assessed the efficacy of $NaHCO_3$ to affect the ability to sprint over the concluding stages of longer exercise bouts. Instead the majority of studies have used mean power output and performance times as their key performance measures, with those assessing PPO generally assessing it over the initial stages of testing (Deb et al., 2017; Driller, Williams, Bellinger, Howe & Fell, 2012; Marx et al., 2002; McNaughton, 1992; Vanhatalo et al., 2010). However, with regards to PPO researchers have shown acidosis' effect on the separate components of power, namely force and velocity. Using isolated fibres acidosis has been shown to depress both force production and contraction velocity (Karatzaferi, Franks-Skiba &

Cooke, 2008; Nelson, Debold & Fitts, 2014; Verbitsky, Mizrahi, Levin & Isakov, 1997), as well as the rate of force production (Higgins, Tallis, Price & James, 2013). These effects have been expanded to *in vivo* human studies using supramaximal cycling and $NaHCO_3$, with both maximal force production and the rate of force development impacted by changes in plasma acidosis (Sielger et al., 2013; Verbitsky, Mizrahi, Levin & Isakov, 1997). Considering these, the use of $NaHCO_3$ to increase plasma pH or better regulate intracellular pH may allow for more rapid transition between steady-state exercise and the initiation of an end-spurt.

The end-spurt has been studied effectively on two occasions using an unrestricted maximal sprint to assess either before and after or after a fixed-intensity and fixed-duration bout (Fujii et al., 2018; Etxebarria et al., 2019). Studies like these offer a more ecologically valid exercise protocol in the context of supramaximal exercise performance considering the importance of the end-spurt in many supramaximal events (Christensen et al., 2017; Craig et al., 1995; Noakes, St Clair Gibson & Lambert, 2005; Martin, Davidson & Pardyjak, 2007). Therefore, these study designs, and more specifically the exercise protocol they employ, provide an avenue for future research using $NaHCO_3$ to explore the role of exercise-induced plasma acidosis on the ability to sprint over the concluding stages of supramaximal competition.

2.5 The anaerobic power reserve/ anaerobic speed reserve

Given the importance of anaerobic energy contribution during supramaximal exercise its measurement and quantification is important. Due to limitations in the current measures of anaerobic capacity, the anaerobic power reserve (APR) and anaerobic speed reserve (ASR) models have arisen to provide a simple, non-invasive alternative for characterizing one's anaerobic potential above $\dot{V}O_2peak$ (Blondel et al., 2001; Green, & Dawson, 1993; Noordhof, Skiba, & de Koning, 2013). The model is typically calculated as: APR = PPO - $p\dot{V}O_2peak$ (Sanders et al., 2017) and ASR = maximal velocity - $v\dot{V}O_2peak$ (Weyand & Bundle, 2005). To

date research has predominantly used the model as a descriptive tool for characterising the exponential decay in intensities that can be maintained over increasing supramaximal time-to-exhaustion trials (from 3-350 s) in both running (ASR) and cycling (APR) (Blondel et al., 2001; Bundle, Hoyt & Weyand, 2003; Sanders & Heijboer, 2019; Sanders et al., 2017; Weyand & Bundle, 2005; Weyand, Lin & Bundle, 2006). More recently these models have been proposed as a potential training tool for prescription of relative exercise intensity (Buchheit & Laursen, 2013; Heaney, 2013; Heaney, 2014; Heaney, 2016; Sandford & Maunder, 2018). A recent study has provided the first evidence that the percentage of ASR, as a measure of relative exercise intensity, may be superior in the prescription of supramaximal exercise when compared with traditional exercise prescription tools i.e. percentage of $v\dot{V}O_2peak$ (Julio et al., 2019). However, some have cautioned its use as a measure of relative exercise intensity due to a lack of evidence relating the model to acute physiological changes seen during supramaximal exercise, or the chronic adaptations that occur in response to training (Boullosa, 2014; Boullosa & Abreu, 2014; Buchheit & Mendez-Villanueva, 2014; Mendez-Villanueva, Hamer & Bishop, 2008). Despite a great deal of growth in research concerning the APR/ASR, one area that has yet to be explored is the relationship between the APR/ASR and acid-base balance. Discussed earlier as a prominent physiological change occurring during supramaximal exercise, the ability to connect the APR to disturbances in acid-base balance may enrich the models understanding and application to supramaximal exercise.

2.5.1 Relationship to physiological responses

It has been suggested that use of lower percentages of the APR/ASR during exercise could prevent an excessive peripheral physiological disturbance, thus sparing the anaerobic capacity and neuromuscular function during exercise (Buchheit, Hader & Mendez-Villanueva, 2012; Bundle et al., 2003; Sundberg, Hunter & Bundle, 2016). From this researchers postulated that

a metabolic basis of fatigue could explain the reductions in mean power or velocity able to be maintained over supramaximal exercise of increasing durations (Buchheit, Hader & Mendez-Villanueva, 2012; Bundle et al., 2003; Sundberg, Hunter & Bundle, 2016; Weyand & Bundle, 2005; Weyand, Lin & Bundle, 2006). However, no physiological data was collected to confirm these claims. Additionally, although the model advocates its use as a means of quantifying anaerobic capacity the model lacks any measurement of glycolytic contribution to exercise. Instead it uses two simple capacity measures, one primarily supported by the alactic energy system and the other the aerobic system, between which anaerobic glycolytic contributions are assumed (Jones et al., 1985; Jacobs, Tesch, Bar-Or, Karlsson & Dotan, 1983; Bogdanis, Nevill, Boobis & Lakomy, 1996; Bogdanis, Nevill, Lakomy & Boobis, 1998). With both glycogenolysis/glycolysis and the alactic pathways important to achieving intensities required during supramaximal exercise, the measurement of blood and intramuscular anaerobic metabolites such as lactate, PCr and ATP concentrations would help better characterize the APR/ASR model. This is particularly necessary given several academics have promoted the models use in exercise intensity prescription (Buchheit & Laursen, 2013a; Buchheit & Laursen, 2013b; Heaney, 2013; Heaney, 2014; Heaney, 2016 Sandford & Maunder, 2018).

Considering research that has explored metabolic changes in relation to either the APR or ASR, studies have shown a positive relationship has been observed between ASR and blood lactate accumulation (Buchheit, Hader & Mendez-Villanueva, 2012; Dardouri et al., 2014; Panissa et al., 2016). Additionally, Julio et al., (2019) has suggested that exercise prescribed as a percentage of ASR may reduce the inter-individual variability in blood lactate response to fixed-intensity repeated sprints when compared with exercise prescribed as a percentage of $v\dot{V}O_2peak$. Alternatively, relationships between APR or ASR and other measures of anaerobic metabolism, such as maximal accumulated oxygen deficit, remain unclear (Buchheit, Hader & Mendez-Villanueva, 2012). Additionally, relationships to oxygen delivery are also

questionable, with a small positive relationship seen in haemoglobin deoxygenation of the biceps femoris, and no relationship seen between the ASR and $\dot{V}O_2$ kinetics (Buchheit, Hader & Mendez-Villanueva, 2012). These results illustrate the weak associations to metabolic measures and showcase that the physiological underpinnings of the APR/ASR are not well understood.

2.5.2 A tool for performance prediction

To date the primary use of the APR/ASR has been to explore exponential decrements in the power/speed-duration relationship within cycling and running. Researchers have sought to hone an exponential model using the anaerobic reserve to describe time-to-exhaustion over various powers and durations from roughly 3-350 s. The development of this model was done in an effort to establish a better physiological measure for performance prediction in sprint and middle-distance events (Weyand, Cureton, Conley, Sloniger, & Liu, 1994). Initially, Blondel et al. (2001) suggested that by expressing times to exhaustion at supramaximal velocities as a percentage of ASR that the individual differences in anaerobic work capacity could be better accounted for. Weyand & Bundle (2005) furthered this argument by showing that for runners from different event backgrounds (sprint, middle-distance and long-distance) the absolute differences in maximal average speeds achieved over durations of 10 to 150 s were invariant when expressed relative to ASR. This bolstered the universality of the application of ASR/APR, and as a result, gave rise to an exponential equation used to predict times to exhaustion in both supramaximal running and more recently in cycling. Within a running context this model has been found to predict all-out track runs within 2.5-3.7% over durations from 3-240 s (Bundle, Hoyt & Weyand, 2003), while researchers have predicted mean power outputs in cycling to within an average of 4.1-6.6% over durations of 5-350 s (Sanders & Heijboer, 2019; Sanders et al., 2017; Weyand, Lin & Bundle, 2006). The use of APR/ASR as

a performance predictive tool has also been used repeated-sprint research. In the study of 29 active males Dardouri et al. (2014) found via stepwise regression that ASR was the only significant predictor of total time and the best time over a single repetition during a protocol of 10x15 m shuttle runs, explaining 47% and 50% of the shared variance.

Researchers have also shown that the predictive strength of the APR/ASR is dependent on adjusting for differences in $v\dot{V}O_2peak$. Buchheit, Hader and Mendez-Villanueva (2012) showed that by adjusting for differences in $v\dot{V}O_2peak$ those with larger ASR had greater exercise capacity. This was reiterated in a following longitudinal study of 270 trained youth soccer players, where increases in mean sprint times over the course of the study were not determined by the change in absolute ASR until it was put into context with improvements in $v\dot{V}O_2peak$ and maximal sprint speed (Buchheit & Mendez-Villanueva, 2014). Although this adjustment to account for differences in $v\dot{V}O_2peak$ has only been applied to repeated-sprint research its application to future research exploring the ASR/APR may allow for better comparison between individuals by removing the confounding effects of differences in aerobic capabilities.

2.6 Conclusion and intended research

Accumulated acidosis is a key consideration in supramaximal performance given the importance of anaerobic ATP production in supramaximal events. Although $NaHCO_3$ is a popular ergogenic aid in the fight against accumulated plasma acidosis, its positive performance response is not consistent. However, it is suggested that well-trained cyclists are a key group that has seen a consistent ergogenic effect, specifically over longer duration supramaximal time-trials of 1-6.5 min. Additionally, both recreational and trained participants

have been shown to benefit from $NaHCO_3$ over the later stage of repeated-sprint protocols. However, research appears to lack ecological validity due to the primary use of three types of exercise protocol. Given the established importance of the end-spurt and an ability to change power or speed late into a race, integrating this into the research design may help to expand the understanding of the effect of $NaHCO_3$ on supramaximal performance (Siegler et al., 2016). Therefore, future research should utilize well-trained cyclists and longer supramaximal time-trials (greater than 60 s), with the integration of a final sprint measure to establish whether acidosis is an underlying mechanism of fatigue in this context.

The physiological underpinnings of the APR/ASR are still to be truly elucidated. Previous research has used the APR/ASR to map the exponential decrease in supramaximal performance over increasing durations in both cycling and running, with researchers suggesting a metabolic basis to explain this exponential decrease. Yet, no metabolic data was taken throughout this research (Blondel et al., 2001; Bundle, Hoyt & Weyand, 2003; Sanders & Heijboer, 2019; Sanders et al., 2017; Weyand & Bundle, 2005; Weyand, Lin & Bundle, 2006). Although several studies have attempted to relate the model to both metabolic and central fatigue measures, research is still limited (Buchheit, Hader & Mendez-Villanueva 2012; Buchheit & Mendez-Villanueva, 2014; Dardouri et al., 2014; Julio et al., 2019; Mendez-Villanueva, Hamer & Bishop, 2008; Panissa, 2016). Therefore, before the model can be applied to performance and training contexts more work is needed to understand the relationship between APR/ASR and the supramaximal exercise it attempts to describe. Because of this it is proposed that a next step in research should be to explore acid-base balance in the context of the APR.

Chapter 3 - Research methods

3.1 Participants

Twelve elite male cyclists were recruited to participate in the study. The training and competitive backgrounds of each participant was identified during screening prior to initial testing. Participants came from a variety of event backgrounds including both sprint (i.e. BMX and track sprinters) and endurance (i.e. multistage tour riders and Ironman triathletes) events. Recruitment of such diverse participants was done in an effort to obtain a range of APRs in order to better test the third hypothesis. Several participants had won medals at the Olympics and world championship level, while others had competed professionally in road tour events, as cycling sailors or in long course triathlons. All participants were healthy and did not highlight any injuries which could place them at risk given the high intensity protocols required during the study. Each participant provided informed written consent prior to the commencement of the study. The study itself was approved by the Auckland University of Technology Ethics committee (AUTEC).

An overall and separate examination of the endurance and sprint participants better illustrates their elite status (Table 4.1). Concerning the endurance participants their aerobic abilities were showcased by an average $\dot{V}O_2peak$ of 68.6 ± 5.5 $mL.kg^{-1}.min^{-1}$ and $p\dot{V}O_2peak$ of 442 ± 37 W. Of these participants, one achieved a world class absolute $\dot{V}O_2peak$ of 6.07 $L.min^{-1}$ (Jeukendrup, Craig & Hawley, 2000). Alternatively, those recruited from a sprint background achieved an average PPO of 1331 ± 238 W (averaged over three pedal strokes) with one participant showcasing a world class peak torque of 266 $N.m^{-1}$ and PPO of 1768 W, which were both achieved over one pedal stroke (Gardner et al., 2005; Gardner et al., 2007). These results were similar to those presented in van der Zwaard et al. (2017) whose mix of elite

participants achieved average $\dot{V}O_2$peaks of 62.7 mL.kg^{-1}.min^{-1} and PPO of 1327 W compared to 59 mL.kg^{-1}.min^{-1} and 1114 W achieved in the current study. After recruitment was complete it was established that participants could be classified evenly into two subgroups, endurance and sprint.

3.2 Research design

The study adopted a double-blind, placebo controlled, randomized, crossover design and was conducted over three separate laboratory visits; an initial session and two subsequent intervention trials. Prior to each testing session participants were advised to avoid strenuous exercise for the 48 hr preceding testing, as well as the consumption of caffeine, alcohol or other ergogenic substances 24 hr prior to testing. Participants were also instructed to arrive at the laboratory in a rested and well-hydrated state, having fasted for at least 2 hr prior to arrival (Driller et al., 2012; McNaughton, Strange, & Backx, 2000). In an effort to ensure that nutrition was kept consistent throughout sessions participants were required to keep a food diary over the 24 hr prior to testing and asked to mimic this diet leading into each intervention session (Carr et al., 2011; Driller et al., 2012). Testing sessions were separated by at least 48 hr and were completed within two weeks of the initial session. Participants were verbally encouraged throughout the exercise testing.

3.3 Experimental procedures

3.3.1 General procedures

All trials were conducted at the same time of day under similar environmental conditions using the same equipment and standardized warm up protocol. An electromagnetically braked cycle

ergometer which also measured pedal forces (Lode Excalibur Sport, Lode, Netherlands) was used throughout testing. Saddle height, headset height and seat position were measured during initial testing, recorded and then maintained for subsequent tests. For each participant, testing was performed over the same time period in the afternoon or evening. The time of testing was set in an effort to be as convenient for the participant as possible, however once set during initial testing it was kept consistent throughout the remaining trials. Rationale for the afternoon or evening trials was due to differences in performance seen between morning and evening testing within high-powered efforts (<60 s) (Lericollais, Gauthier, Bessot, Sesboüé & Davenne, 2009). Finally, environmental temperature (18 ± 0, 18 ± 1 and 18 ± 1 °C, $P = 0.28$), relative humidity (63 ± 7, 63 ± 9 and 63 ± 5%, $P > 0.05$) and barometric pressure (754 ± 4, 754 ± 5 and 753 ± 5 mmHg, $P = 0.25$) were kept similar across the initial session and both $NaHCO_3$ (BIC) and placebo (PLA) intervention trials.

On arrival to the laboratory participants were explained the protocols for each trial. They were then given a general health questionnaire (Appendix B.4) to ensure that they met the inclusion criteria for the study and to establish that there were no pre-existing contraindications for both the intended supplementation or exercise. Following provision of written informed consent (Appendix B.2), body mass and height were measured. Throughout testing a standardized warm up was used across each trial. This warm up consisted of 10 min of cycling at 100 W followed by 3 progressive 6-s efforts separated by 60 s of active recovery at 100 W. These progressive efforts corresponded to 80%, 90% and then 100% of the participants' perceived maximal effort. This structure of warm up aligned with that used by several track cyclists spoken to during pilot testing. An added benefit of the progressive efforts was it allowed for participants to familiarize themselves with the intensity required during the PPO testing, in an effort to minimize the learning effect. After the standardized warm up participants cycled for

1-min at 100 W and then rested passively for 5 min prior to the start of exercise testing on each occasion.

3.3.2 Initial trial

The first laboratory visit was used to establish APR, determine the relative intensities used in the subsequent two intervention trials, and familiarise participants with the intensity requirements of the intervention's fixed-intensity time-trial while validating the prior established $\dot{V}O_2$peak.

3.3.2.1 Initial peak power output testing

After the standardized warm up participants then completed two maximal tests to generate their APR. The first of which was a measure of PPO. During this testing participants sat on the cycle ergometer and were instructed to progressively increase their cadence to roughly 90% of their maximal effort while the flywheel remained unresisted (no breaking forces applied). From this point participants indicated to one of the researchers that they were ready to begin the test, to which the researcher initiated a count down before the test began. During this countdown participants were to increase their cadence to a maximal effort. On the instruction of the researcher the test began, resistance was applied to the mechanically braked fly-wheel, and the participant cycled maximally for the 6 s. The test was initiated at maximal cadence for three reasons. Firstly, this was to avoid any inertial contribution that was found to inflate PPO during pilot testing. Secondly, this procedure is similar to that used by several other researchers in establishing APR (Capmal & Vandewalle 1997; Sanders & Heijboer, 2019; Sanders et al., 2017; Weyand, Lin & Bundle, 2006). Finally, during intervention testing the PPO conducted post the 3-min fixed-intensity time-trial (PPO2) was to be conducted from a maximal rolling

start and therefore, for effective comparison, all PPO testing was done in a similar fashion. This protocol used during initial testing was also used in the initial PPO two intervention trials. After the PPO was conducted participants underwent a 10-min recovery period that included 3 min of cycling at 100 W and then 7 min of passive recovery, which has been determined sufficient time (~10 min) for which near full recovery of PCr can be achieved (Bogdanis et al., 1995; Casey et al., 1996; Dawson et al., 1997).

3.3.2.2 Graded step test to establish $\dot{V}O_2$peak and p$\dot{V}O_2$peak

Following this recovery period, the second exercise test was a graded step test to volitional exhaustion to obtain both $\dot{V}O_2$peak and p$\dot{V}O_2$peak. The graded step test started at 100 W and involved 2-min stages increasing by 50 W until volitional exhaustion. The test was terminated either by volitional exhaustion or when cadence fell below 70 rev.min^{-1} despite verbal encouragement (Lucia et al., 2006). To establish a ventilatory peak, breath by breath analysis of pulmonary gas exchange was recorded over 15-s intervals and was then averaged over 30 s using a calibrated metabolic cart (Parvo Medics TrueOne 2400) (Crouter, Antczak, Hudak, DellaValle & Haas, 2006). The p$\dot{V}O_2$peak was calculated in a pro rata fashion. This was done using the calculation below (Faria, Parker & Faria, 2005; Kuipers et al., 1985):

p$\dot{V}O_2$peak (W) = Last completed stage (W) + [(time completed during final non-completed stage/ step duration) x step increment (W)]

p$\dot{V}O_2$peak was calculated for two reasons. Firstly, to determine the relative intensity of the 3-min fixed-intensity time-trial (to be completed at 105% p$\dot{V}O_2$peak) and secondly to establish the lower boundary of the participants APR.

It was identified from research and during pilot testing that the type of graded protocol (step vs. ramp and the step increases in power) used to determine p$\dot{V}O_2$peak had a significant effect on the power achieved at $\dot{V}O_2$peak which affected participants ability to complete the 3-min fixed-intensity time-trial used during the intervention sessions (Barton et al., 2014; Bentley & McNaughton, 2003; Bishop, Jenkins & Mackinnon, 1998). Pilot testing highlighted the intraindividual differences in p$\dot{V}O_2$peak that could be achieved with differing protocols (i.e. ramp of 25 W.min^{-1} starting at 100 W, a 2 min ramp increasing by 50 W starting at 100 W, and a 3-min step increasing by 35 W starting at 95 W). In an effort to establish the best protocol to enable well-trained participants to cycle for at least 3 min at 105% p$\dot{V}O_2$peak and sprint after this; an assessment of graded test protocols previously used to test time-limits-to-exhaustion at 100% of $\dot{V}O_2$peak was undertaken (Billat et al., 1996; Costa et al., 2011; Laursen, Shing & Jenkins, 2003). From this it was agreed that the step protocol used by Billat et al. (1996) was the most appropriate as it allowed well-trained participants to cycle close to ~4 min at 100% p$\dot{V}O_2$peak compared to other protocols that showed participants riding considerably less (Billat et al., 1996; Costa et al., 2011; Laursen, Shing & Jenkins, 2003). The calculated p$\dot{V}O_2$peak was then pilot tested to ensure that participants could consistently complete 3 min at the protocols prescribed 105% p$\dot{V}O_2$peak and that a reduction in plasma pH was seen.

3.3.2.3 Familiarization/ $\dot{V}O_2$peak validation

The final component of the initial trial was the familiarization bout commencing 20 min after the graded step test. This 20-min recovery period included 3 min of active recovery at 100 W and then 17 min of passive recovery. During the familiarization bout participants cycled for 2 min at 105% p$\dot{V}O_2$peak. This bout was preceded by a short stepped warm up of 1-min at 50%

p$\dot{V}O_2$peak and then 1-min at 70% p$\dot{V}O_2$peak (Nolan, Beaven & Dalleck, 2014). The familiarization bout was included in an effort to prepare participants for the intensities required during the upcoming intervention trials and also to verify the $\dot{V}O_2$peak achieved during the graded step test (Pool & Jones, 2017). This verification process requires a participant to complete a supramaximal bout to ensure that the $\dot{V}O_2$ reading obtained is not higher than the one achieved during the graded step test (Pool & Jones, 2017). To ensure that the bout accounted for the $\dot{V}O_2$ slow component present during severe exercise (intensities above critical power) the duration was suggested to be slightly longer than 80-100 s (Barstow, 1994; Jones & Poole, 2013). Hence the bout duration was set at 2 min (Billat et al., 1996; Costa et al., 2011; Laursen, Shing & Jenkins, 2003). Finally, the intensity of 105% p$\dot{V}O_2$peak was appropriate as it not only aligned with the desired intensity required to familiarize the participant, but it also matched the intensity recommended by Nolan, Beaven & Dalleck (2014) in their study of $\dot{V}O_2$ verification. Verification was met if the $\dot{V}O_2$peak seen during the exercise was equal to or lower than the recorded $\dot{V}O_2$peak during the graded step test (Midgley, McNaughton & Carroll, 2006; Pool & Jones, 2017).

3.3.2.4 Sodium bicarbonate and placebo supplementation (preparation and blinding)

Following the conclusion of the initial testing session the two intervention supplements ($NaHCO_3$ and placebo) that would be randomly assigned to either intervention trial was measured and constructed by hand and encased in gelatin capsules. The relative dosage of 0.3 $g.kg^{-1}$ BM of $NaHCO_3$ was used in the current investigation as it is the most commonly used dosage in research. This dosage has been shown to elicit desired plasma changes and improve performance relative to lower doses, while reducing gastric discomfort compared to higher doses (Carr, Hopkins & Gore, 2011; Ferreira et al., 2015; Ferreira et al., 2019; Gough et al.,

2018; Jones et al., 2016; Matson & Tran, 1993; McNaughton, 1992a; McNaughton et al., 2016). Alternatively, an equal dose of corn flour was used as the placebo condition as this has successfully been used in prior research (Kilding, Overton & Gleave, 2012). Dosages were based on initial body mass, measured during initial testing. The success of the $NaHCO_3$ supplementation was evaluated through measurement of the absolute change in plasma [HCO_3^-] measured at rest and post initial PPO (PPO1) during the relevant intervention trial. The absolute change in plasma [HCO_3^-] was selected as the measure has been shown to be highly reproducible amongst individuals post $NaHCO_3$ ingestion (de Araujo Dias et al., 2015; Gough et al., 2017).

To ensure the randomized, double-blind condition was upheld a third part independently constructed both supplements in private. Randomisation was done at their discretion. A complete list of the supplement allocations was not revealed to the blinded researchers until after the last trial by the final participant. Additionally, it has been highlighted that $NaHCO_3$'s erogenicity may be accounted for by an expectancy of ergogenic effect (Higgins & Shabir, 2016; McClung & Collins, 2007). Therefore, to assess whether participants remained blinded throughout testing in hopes of avoiding any cognitive bias, each was asked to guess which supplement was allocated to which trial on completion of their final intervention trial (Kilding, Overton, & Gleave, 2012).

3.3.3 Intervention trials

The two intervention trials were designed in a similar fashion with the only difference between them being the supplement assigned. Each trial was therefore labelled according to this supplement assignment; with the $NaHCO_3$ trial referred to as BIC and the placebo trial was referred to as PLA.

3.3.3.1 Resting measures

On arrival participants body mass was measured to ensure no excessive fluctuations in mass were seen between trials. If fluctuation did occur this could have affected both the PPO and supplement quantity calculations. Resting blood measures were then taken prior to distribution of supplementation. Blood measures were taken via two sites, one from the antecubital vein of the arm and the second via fingertip capillary sample, extracted via sterilized puncture using a lancet. Because two blood samples were taken during testing two researchers were present. This was done in an effort to take measures as soon as possible post-exercise and in close proximity to each other, taking into account the safety of the participant. The venous sample was roughly 1-mL obtained via venipuncture and drawn into a non-heparinised vacutainer using a syringe (i-STAT analyser Immuno-ready, Abbot, USA). The sample was then carefully expelled into an i-STAT analyser CG8+ cartridge for analysis within 60 s to avoid coagulation (Abbott Laboratories, 2012). The second blood draw was taken via fingertip capillary sample, measured roughly 0.3 μL. This sample was then used for the analysis of blood [La^-] (Lactate Pro, Akray, Japan).

In addition to blood measures SpO_2 was also measured at rest and at several times throughout testing using a pulse oximeter (Masimo Corporation, California, USA). To do this a sensor was applied to the forehead using an adhesive plastic strip that aided the connection between the sensor and the skin. The sensor was cleaned and sterilized with an alcohol wipe prior to application to also help with adhesion. A manufacturer supplied bandage was then wrapped around the head to support the sensors adherence to the skin and to cover the sensor from any light exposer that could create artefact readings (Masimo Corporation, 2006). The use of the forehead sensor has been promoted for more reliable measurement during maximal exercise

over other sites (i.e. ear or finger) (Tokuda, Hayamizu, Ogawa, Hirai & Irita, 2007; Yamaya, Bogaard, Wagner, Niizeki, Hopkins, 2002). This position was also chosen as it was believed to be the most comfortable for the participant during maximal cycling. It also allowed the cable connecting the sensor to the pulse oximeter device to be held by the researcher behind the participant, reducing the risk of the cable getting caught and the sensor dislodged as the participant transitioned from the ergometer to an adjacent bed post-exercise. For resting measures the sensor was applied for several minutes to ensure a stable reading was collected. Alternatively, for exercise testing the sensor was applied after completion of the warm up and remained on throughout testing until the blood measures were taken post the final PPO (PPO2).

3.3.3.2 Sodium bicarbonate and placebo loading (ingestion and absorption) and monitoring of gastric discomfort

After resting measures were obtained, participants then ingested their randomly allotted $NaHCO_3$ or placebo supplement. Participants were then given a 30-min period with which to consume the allotted capsules with 330 mL of water (Kilding, Overton & Gleave, 2012). After complete ingestion of the supplement participants then waited passively for 75 min to allow for supplement absorption (Renfree, 2007; Price & Singh, 2008; Siegler et al., 2010). After this passive absorption period body mass was re-measured to ensure that no major fluctuations had occurred since arrival, potentially resulting from gastric discomfort secondary to the $NaHCO_3$ ingestion (Carr et al., 2011). No participants reported any voidance issues relating to gastric distress and bodyweights were not seen to fluctuate. As a result of the supplementation loading protocol only a mild discomfort was experienced by participants throughout testing (Fig. 4.6).

Several efforts were made to minimise of gastric discomfort. Firstly, both $NaHCO_3$ and placebo supplements were consumed via gelatin capsules (Carr et al., 2011; Junior, de Salles Painelli,

Saunders, & Artioli, 2015; Matson & Tran, 1993; McNaughton, Siegler, & Midgley, 2008; Price, & Singh, 2008; Siegler et al., 2013). Pilot testing using this mode of delivery saw little to no gastric disturbance compared with other methods (i.e. mixing $NaHCO_3$ straight into water or other carbohydrate rich beverages). Secondly, participants were given a 30-min ingestion period in an effort to space out the share quantity of capsules required to be consumed (Kilding, Overton & Gleave, 2012). Finally, gastric discomfort was measured throughout testing to identify the onset of any severe gastric disturbance and avoid jeopardizing the health and performance of the participants (Carr et al., 2011; Gough et al., 2017; Jones et al., 2016; Kahle et al., 2013; Mohr, 2017; Saunders et al., 2014). Measures were taken at rest, every 10-min interval after ingestion until the warm up was commenced, as well as pre and post each exercise test. This was done via a 7-point Likert scale (ranging from *1 = no problem to 7 = very severe*) (Carr et al., 2011, Van Zanten et al., 2006). One final consideration was the reduced incidence of gastric disturbance found when $NaHCO_3$ is co-ingested with carbohydrate rich foods (Carr et al., 2011). Despite this research it was agreed that participants would remain fasted for at least 2 hr prior to ingestion of either intervention supplement. This was done in an effort to minimise the potential confounding ergogenic effects of other foods or supplements and to ensure that foods were completely digested in case the intensity of the exercise protocol itself caused gastric issues (de Alcantara Santos, 2013; Skare, Skadberg & Wisnes, 2001; Stellingwerff, Maughan & Burke, 2011).

3.3.3.3 Initial peak power output and initial blood and SpO_2 measures (PPO1)

The exercise protocol was then initiated by the standardized warm up, which was followed by a 5-min passive recovery period. Therefore, the subsequent testing commenced ~90 min post-supplement ingestion. The pulse oximeter was then reapplied to the forehead and remained on and recording throughout the remainder of the trial. Testing began with a 6-s

all-out maximal sprint to establish an initial PPO (PPO1) which was conducted in the same fashion to that of the PPO measured during initial testing. Following this a second ~1-mL antecubital venous and 0.3 μL fingertip capillary blood sample was collected. For post-exercise blood draws participants would dismount from the cycle ergometer and lie supine on a bed located 1 m away. Blood collection was completed within 15-30 s post-exercise, which has been shown to minimally effect the variability in concentration of plasma pH, electrolytes, hematological variable and blood lactate (Bishop et al., 2008; Lindinger, Heigenhauser, McKelvie & Jones, 1992). Participants then recovered for 10 min, which included 3 min of cycling at 100 W and 7 min of passive recovery.

3.3.3.4 Fixed-intensity time-trial and final PPO, plus final blood and SpO_2 measures (PPO2)

At the completion of the recovery period participants moved to a 3-min fixed-intensity time-trial at 105% p$\dot{V}O_2$peak, followed directly by a final PPO measurement (PPO2). The fixed-intensity time-trial was intended to simulate ~75% of a 4000-m individual pursuit event in track cycling. Therefore the duration of the bout (3 min) was determined as it equates to ~75% of the world record time for the men's 4000-m individual pursuit (Mexican Cycling Federation, 2018) and the intensity was defined from previous research (Craig & Norton, 2001; Jeukendrup, Craig & Hawley, 2000). Directly following the completion of the 3-min fixed-intensity time-trial participants immediately commenced a 6-s maximal-sprint to capture a post-exercise PPO (PPO2). This 6-s maximal-sprint was performed in a continuous fashion with no break in between that and the time-trial. SpO_2 was measured throughout both the 3-min fixed-intensity time-trial and PPO2. A final ~1-mL antecubital venous sample and 0.3 μL fingertip capillary blood sample was then taken immediately following PPO2.

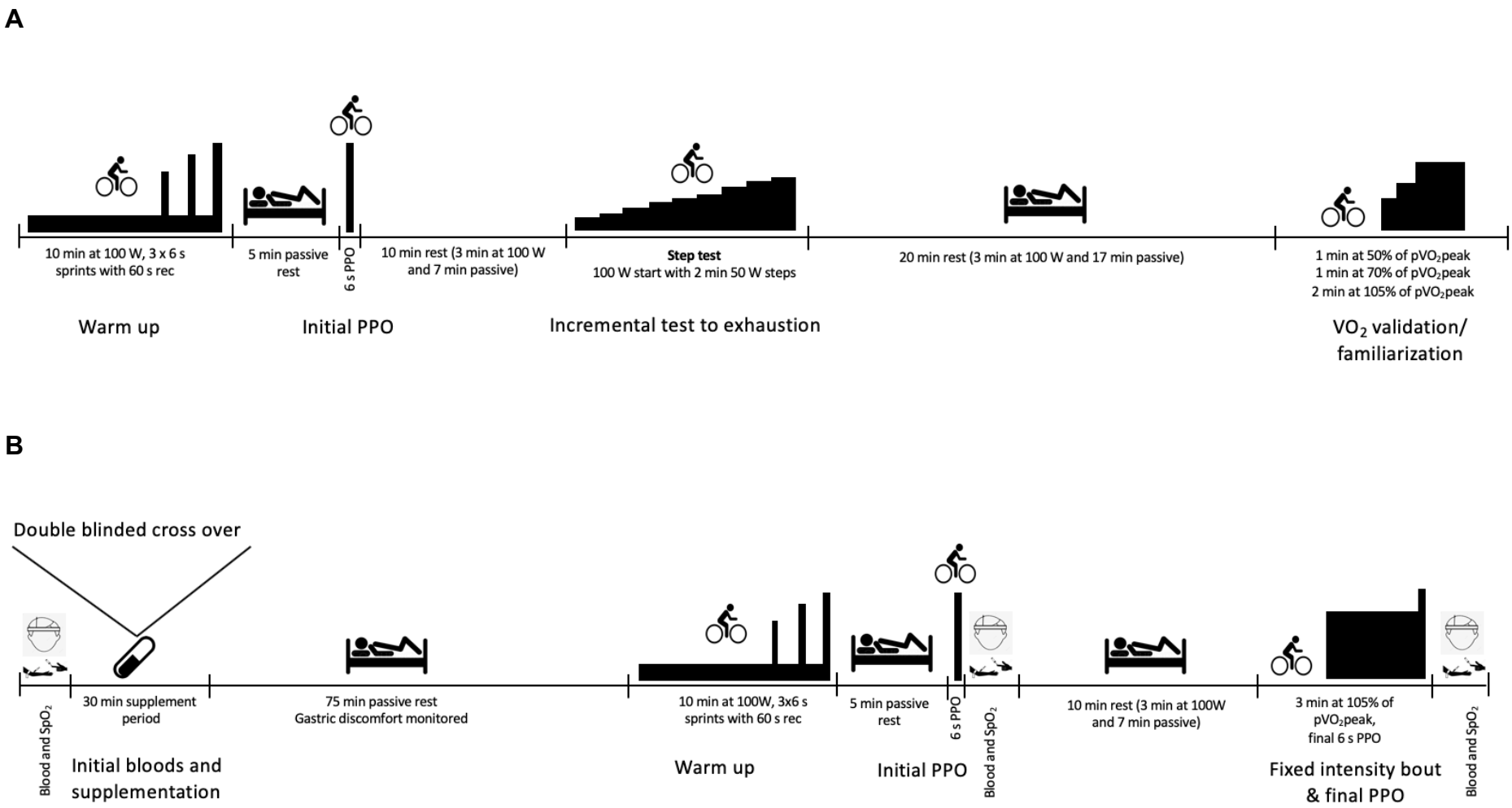

Figure 3. 1. Schematic representation of the study design, illustrating both A) initial and B) intervention sessions.

3.4 Analysis

3.4.1 Blood analyses

Venous whole blood samples were analysed using an i-STAT hand-held analyser and a single CG8+ i-STAT cartridge (i-STAT analyser Immuno-ready, Abbot, USA). Using the i-Stat system the concentration of variables was determined from the plasma fraction of the venous whole blood sample taken. Several plasma concentration measures were taken during the intervention trials. These included blood gases such as HCO_3^-, pH, lactate, K^+, Na^+, Ca^{2+} and haematological measures such as haematocrit and haemoglobin. Each of these variables was chosen due to their involvement in acid-base balance, oxygen transport and as proposed agents in fatigue (Cairns & Lindinger, 2008; Lindinger, 1995; Thomas & Lumb, 2012). Plasma ions were measured by ion-selective electrode potentiometry, pH and HCO_3^- was measured by direct potentiometry, and both haematocrit and haemoglobin were determined conductometrically (Abbott Laboratories, 2012). The reliability and validity of the i-STAT handheld analyser has been described previously and has been found to be an acceptable measure of plasma pH (Pruscino et al., 2008; Silverman & Birks, 2012). Additionally, the technical error of the i-STAT analyser has been reported to be relatively low (~2.5%) (Dascombe, Reaburn, Sirotic & Coutts, 2007). Measurements were analysed at 37 °C in accordance with manufactures settings. Through discussion with the manufacturers a range of 1 °C was given to ensure that measurement was still able to be reliably analysed at 37 °C. Therefore, in an effort to ensure that plasma temperatures did not rise above this threshold testing was conducted to determine changes in core temperatures resulting from exercise completed in the intervention trial. Rectal temperatures taken from a small sample (n = 3) showed that core temperatures did not rise above the 38 °C threshold (37.4 ± 0.2 °C). Additionally, other studies in rowing and running who have measured both muscle and plasma

temperatures provided further evidence that plasma temperatures would not have risen above 38 °C in the current investigation (Byrne et al., 2006; Nielsen et al. 2002).

Blood lactate was analysed using a Lactate Pro II portable analyser (Lactate Pro, Akray, Japan). This handheld device uses an enzymatic amperometric detection method which interprets an electrical signal produced as a result of the reaction between lactate and lactate oxidase and therefore corresponds to the [La^-] (Bonaventura et al., 2015). The Lactate Pro II portable analyser has repeatedly been shown to be a valid and reliable measure of blood lactate, while exhibiting a high degree of agreement with other lactate analysers (Baldari et al., 2009; Pyne, Boston, Martin & Logan, 2000). It must be noted that on 3 occasions during measurement after PPO2 an error reading of "HI" was observed using the Lactate Pro II. This error indicates that the readings were outside of the upper limit of the device's measurement range (0.5-25 $mmol.L^{-1}$) (Akkray, 2013). Due to a need for consistency of measurement timing a resample was not taken because of the slight delay required for retesting via capillary sample. Additionally, venous blood taken at the same time as the original capillary sample was also not used for analysis. This is due to previous comparisons between venous and capillary blood at submaximal intensities showing significant differences between the two measurement sites (Foxdal et al., 1990). Therefore in cases where the "HI" error was displayed a reading of 25 $mmol.L^{-1}$ was recorded in accordance with the upper limit outlined by Akray (2013).

3.4.2 Peripheral oxygen saturation

SpO_2 was measured using the Rad 8 pulse oximeter (Masimo Corporation, California, USA). This device has been shown to be reliable against other devices in the reduction of reading artefact (Barker, 2002; Kist et al., 2002; Shah, Taleghani, Chitkara & Miller, 2005; Witucki & Bell, 2000) and valid compared to laboratory grade equipment (Hanson, Mottram & Scanlon, 2003). Oxygen saturation was recorded at 2 s intervals throughout exercise. This allowed for

the time course of SpO_2 to be determined throughout the fixed-intensity time-trial and PPO2 (Fig. 4.3) and then compared between interventions.

3.4.3 Heart rate

Heart rate was recorded continuously throughout testing using two devices. During initial testing a Polar RS800cx heart rate monitor (RS800cx; Polar Electro, Kempele, Finland) with chest strap was used to determine maximal heart rate. The other device was the Rad 8 pulse oximeter (Masimo Corporation, California, USA) which was measured at the forehead. This device was used in conjunction with the Polar heart rate monitor during both intervention trials. However, the pulse oximeter was preferred for analysis of the intervention trials due to its 2-s recording rate. This allowed for the time course of heart rate data to be accurately modelled (Fig. 4.5) throughout each intervention trial and then compared between interventions.

3.5 Performance measures

3.5.1 Peak power output, torque and cadence

Performance was assessed as the percentage change in PPO, peak torque and peak cadence from PPO1 to PPO2 (Glaister et al., 2008). PPO was defined as the highest average power achieved over three consecutive, complete pedal strokes. This was calculated firstly by multiplying total pedal torques ($N.m^{-1}$) with cadence (or the rotational speed of the crank ($rad.s^{-1}$)). Total torque was the sum of individual pedal torques measured every 2° (180 times) over a single revolution of both right and left cranks. In the case where negative torques were measured, indicating negative parts of the angle-torque curve, these were transformed to their absolute value prior to calculation of total torques (Sarre, Lepers & Van Hoecke, 2005). The average power was then derived from this by dividing the total by 180 (Appendix B.8). Finally,

the three pedal stroke average was then calculated by adding the highest three consecutive pedal strokes and dividing these by three. Averaging the performance measures over three pedal strokes was done in an effort to nullify the effects of measurement noise and better align measurements with those used by other researchers (Sanders & Heijboer, 2019; Sanders et al., 2017; Weyand, Lin & Bundle, 2006). This technique was also applied to the calculation of peak torque and peak cadence.

Within the Lode software the linear mode was selected during PPO testing as this setting had previously been recommended for sprint testing (Appendix B.7; Driss & Vandewalle, 2013; Capmal & Vandewalle, 1997). This setting applies a linear factor or braking force against the electromagnetic flywheel. This linear factor can vary between 0.001 and 1 $N.m.s.rad^{-1}$. In this study a linear factor of 0.042 $N.m.s.rad^{-1}$ was used and this was then adjusted for differences in body mass through the use of a set torque factor of 0.7 $Nm.kg^{-1}$ (Capmal & Vandewalle, 1997). Use of these factors was determined through communications with the manufacturer who outlined that these would be appropriate for healthy trained participants and also would ensure the resistance was not too excessive for PPO2 given the potential for fatigue resulting from the fixed-intensity time-trial (Appendix B.9).

Several inclusion criteria existed in order for a pedal stroke to be included in the study. A complete pedal strokes was considered one that measured torques over at least 350°. Those that failed to meet this requirement were discarded. Such cases occurred when PPO testing commenced partway through a pedal stroke or if there was a skip in the reading. This was done in an effort to eliminate inflated readings due to incomplete pedal strokes that were averaged over smaller sets of torque data. Considering PPO2, although 6 s was allotted for the generation of PPO, in anticipation of this several participants begun to sprint before the end of the fixed-

intensity time-trial portion of intervention testing. In such cases pedal strokes were included in analysis of PPO2 only if they fell within the last second of the fixed-intensity time-trial.

It must also be noted that selecting the linear mode provided an additional output for the calculation of PPO, separate to the procedure outlined above. This output was disregarded as the power output is based on the constant braking force placed against the electromagnetic flywheel and therefore is directly proportional to cadence (Capmal & Vandewalle, 1997). Because of this fixed braking force, the linear mode output did not accurately represent the differences between participants, mainly considering differences in body mass and event background (Burnley et al., 2010). This was determined through comparison of the results derived from the linear mode with the PPOs achieved by world class sprint cyclists (Dorel et al., 2005). Therefore, given the processing power of the Lode ergometer, which enables the accurate and variable measurement of the two constituents of power, force (torque) and velocity (cadence), the pedal force data was instead used to calculate PPO. This was also shown to be a more accurate representation of differences in body mass and event background, confirmed through comparison of results with the power outputs achieved in Dorel et al. (2005).

3.5.2 Anaerobic power reserve

The APR was defined as a ratio of (Mendez-Villanueva et al., 2010; Sandford, Allen, Kilding, Ross & Laursen, 2018):

$$\frac{\text{PPO}}{\text{p}\dot{V}O_2\text{peak}}$$

The APR was calculated as a ratio in an effort to account for differences in $p\dot{V}O_2peak$, as it is not the absolute difference between PPO and $p\dot{V}O_2peak$ that is of interest but instead the amplitude in relation to $p\dot{V}O_2peak$ (Buchheit, Hader & Mendez-Villanueva, 2012; Buchheit & Mendez-Villanueva, 2014).

3.6 Statistical procedures

Data values are given as mean ± standard deviation. Two separate statistical packages were used to conduct several statistical procedures. Firstly, SPSS Statistics was used to conduct two-way repeated measures analysis of variance, three-way mixed model analysis of variance and Pearson's correlations (SPSS Statistics, version 24, SPSS Inc., Chicago, Illinois). Alternatively, Microsoft Excel was used for manual post hoc testing, paired and unpaired samples *t*-tests, and the creation of several figures (Microsoft Excel 2018, Version 16.20, Microsoft Inc., Redmond, Washington). Plasma data collected during resting, PPO1 and PPO2 for both intervention trials were considered separately. Previous research using a priori power calculation has shown that the sample size of 12 would allow for detection of a less than 1% change with high statistical power ($\beta = 0.80$; $0.05 = \alpha$ level) (Gough, 2018). Statistical significance was inferred when $P < 0.05$. Normality testing was conducted on individual datasets using the Shapiro-Wilk test (Shapiro & Wilk, 1965), which is considered robust for sample sizes of $n < 50$ (Rahman & Govindarajulu, 1997). This was conducted in an effort to ensure the relevancy of the models used for analysis.

Subsequently, overall between-interventions (i.e. BIC and PLA) and within-trials (i.e. rest, PPO1 and PPO2) comparisons were made using a two-way repeated measures analysis of variance (Fisher, 1925; Fujikoshi, 1993). This was done to answer the first two research

questions and establish a causal mechanistic role for acidosis' effect on PPO. Additionally, subgroup (i.e. END and SPR) between-interventions (i.e. BIC and PLA) and within-trials (i.e. rest, PPO1 and PPO2) comparisons were also made using a three-way mixed model analysis of variance. This was done to help showcase any differences that existed between endurance and sprint subgroups prior to analysis of the APR by Pearson's correlations. In accordance with the requirements of these statistical models the assumptions of normality, homogeneity of variances and sphericity were determined prior to running each procedure. The homogeneity of variances was assessed using Levene's testing in which a statistically significant result illustrates a non-equal variance and therefore a violation of the assumption. Sphericity was then assessed via Mauchly's Test of Sphericity. Data was deemed non-spherical if the Mauchly's test was statistically significant ($P < 0.05$) showing that the variances of the differences were not equal (i.e. sphericity has been violated). In this case data was corrected for using the Greenhouse-Geisser (Epsilon < 0.75) or Huynh-Feldt (Epsilon > 0.75) adjustments (Atkinson, 2001; Maxwell & Delaney, 2004). Post hoc pairwise comparisons with Bonferroni stepwise correction was applied to locate the separate effects. This correction is deemed a conservative method helping to protect against type I error (Dunn, 1959; Dunn, 1961). The adjustment involved computing paired *t*-tests for each significant comparison and then multiplying the resultant *P* value by the number of comparisons made (i.e. 2 for between-intervention and 2-3 for within-trial).

Two-tailed paired and unpaired samples *t*-tests were used to compare several variables. Paired *t*-tests were used in the study of gastric discomfort (Fig. 4.6) (Gosset, 1908). Alternatively, unpaired samples *t*-tests were used for simple comparisons between END and SPR used in Table 4.1.

Additionally, several relationships between physiological and performance measures as well as the APR were explored using Pearson's correlation coefficients. For this analysis only measures taken during PLA were used. This was done to avoid the potential confounding effects of $NaHCO_3$. The direction and strength of these relationships between variables was made using the correlation coefficient (r). The strength of the relationship was determined as weak positive: r = 0.1 to 0.29, weak negative: r = -0.1 to -0.29, moderate positive: r = 0.3 to 0.69, moderate negative: r = -0.3 to -0.69, strong positive: r = 0.7 to 0.9 and strong negative: r = -0.7 to -0.9, with r = 0 representing no relationship and r = 1.0 a perfect relationship (Akoglu, 2018). The level of significance was then considered to determine the level of confidence in the measure (Pallant, 2013).

Chapter 4 - Results

4.1 Participant characteristics

Participants categorised into END had lower body mass, higher $\dot{V}O_2$peak and p$\dot{V}O_2$peak; conversely SPR had a higher PPO and APR, with resting plasma haematocrit and haemoglobin concentrations not significantly different between-subgroups (Table 4.1). Individual responses showed that END achieved $\dot{V}O_2$peak values ranging from 62-73 mL.kg^{-1}.min^{-1} and PPO ranging from 585-1268 W. While SPR achieved $\dot{V}O_2$peak values ranging from 45.6-52.2 mL.kg^{-1}.min^{-1} and PPO ranging from 1111-1754 W.

Table 4. 1. Participant characteristics - Overall and by subgroup

		By specialty		
	Overall (*n* = 12)	**END (*n* = 6)**	**SPR (*n* = 6)**	***P* value**
Age (y)	27 ± 5	25 ± 4	29 ± 4	0.13
Height (cm)	183.9 ± 7.2	185.7 ± 7.7	182.1 ± 6.8	0.42
Body mass (kg)	84.7 ± 13.7	74.0 ± 8.1	95.5 ± 8.1	0.001
Max HR (beat.min^{-1})	187 ± 12	190 ± 16	184 ± 3	0.37
$\dot{V}O_2$peak (mL.kg^{-1}.min^{-1})	59.0 ± 10.8	68.6 ± 5.5	49.5 ± 2.6	< 0.001
p$\dot{V}O_2$peak (W)	413 ± 48	442 ± 37	385 ± 43	0.03
PPO (W)	1114 ± 318	897 ± 232	1331 ± 238	0.01
Peak torque (N.m^{-1})	71.8 ± 17.8	60.0 ± 10.0	83.6 ± 15.7	0.01
Peak cadence (rev.min^{-1})	188 ± 11	185 ± 12	191 ± 11	0.39
APR (PPO/p$\dot{V}O_2$peak)	2.76 ± 0.98	2.02 ± 0.42	3.51 ± 0.76	0.002
105% p$\dot{V}O_2$peak (W)	434 ± 51	464 ± 39	384 ± 43	0.03
105% p$\dot{V}O_2$peak as a % of APR (%)	4 ± 2	6 ± 2	2 ± 1	0.006
[Hb] (*g.L^{-1})	149 ± 10	147 ± 11	152 ± 10	0.73
Haematocrit	0.44 ± 0.03	0.43 ± 0.03	0.44 ± 0.03	0.71

Values are presented as mean ± standard deviation. Abbreviations: END = Endurance subgroup; SPR = Sprint subgroup; $\dot{V}O_2$peak = maximal oxygen uptake; p$\dot{V}O_2$peak = power at $\dot{V}O_2$peak; Max HR = maximal heart rate during initial testing; APR = anaerobic power reserve; PPO = peak power output; 105% p$\dot{V}O_2$peak = the power calculated at 105% p$\dot{V}O_2$peak to be used in the fixed-intensity time-trial; % APR = the percentage of APR represented by the absolute workload calculated at 105% $\dot{V}O_2$peak; Hb = haemoglobin. All plasma values were taken at rest during the placebo trial.

4.2 Performance measures

PPO (Fig. 4.1A) showed a significant decrease between PPO1 and PPO2 (44.7 ± 13.0%, $P < 0.001$). However, the decline of was not significantly different between-interventions (45.7 ± 13.7% PLA vs. 42.3 ± 12.6% BIC, $P > 0.05$, Fig. 4.1A). There was no significant difference in the decrease in peak torque (Fig 4.1B) (9.9 ± 17.2% PLA vs. 5.4 ± 17.6% BIC, $P = 0.34$) or cadence (Fig. 4.1C) (42.8 ± 6.2% PLA vs. 42.4 ± 7.1 BIC, $P = 0.42$) between-interventions. However, there was a significant decrease in cadence within-trials from PPO1 and PPO2 (42.6 ± 6.5%, $P < 0.001$).

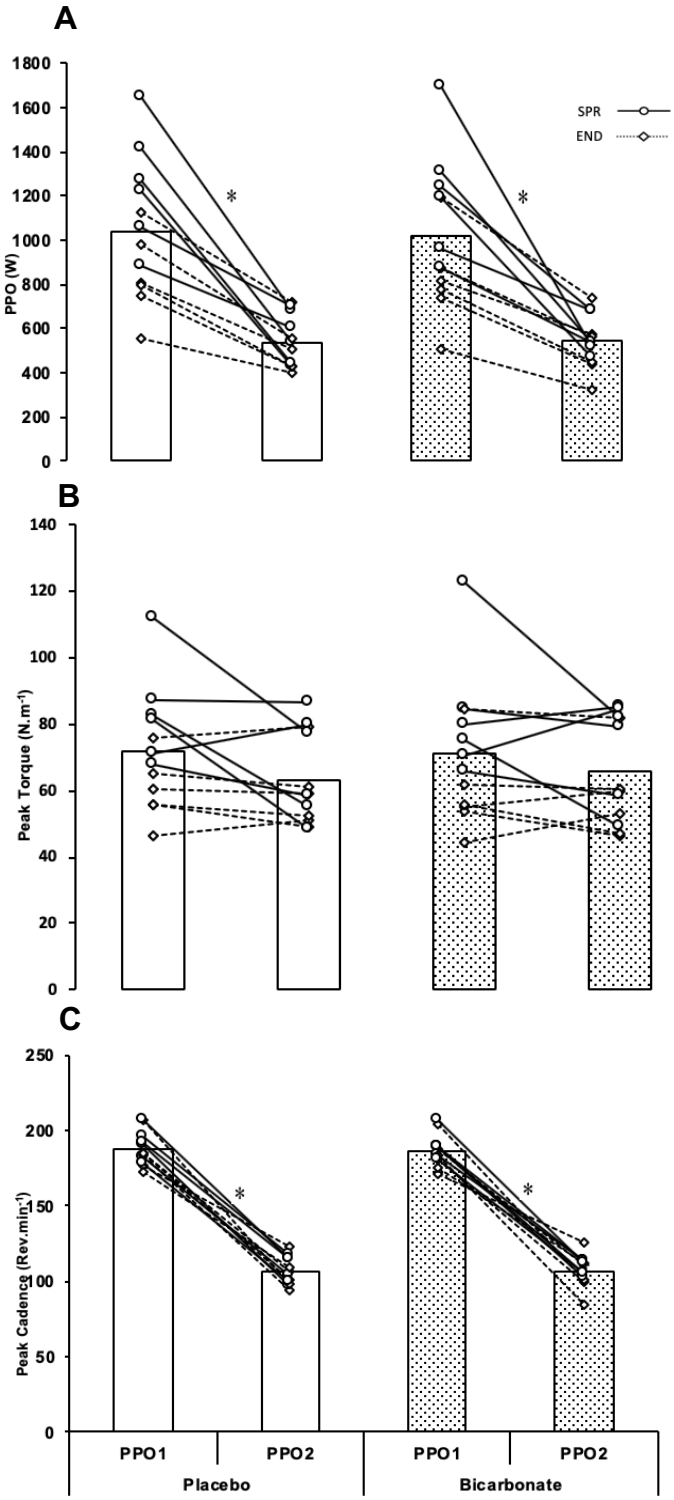

Figure 4. 1. Individual and mean responses for A) Peak power output (PPO) B) Peak torque and C) Peak cadence averaged over three pedal strokes for initial PPO (PPO1) and final PPO (PPO2) timepoints between PLA and BIC (n = 12). Bars indicate overall mean data. Individual responses are identified by lines and are separated into sprint (SPR) and endurance (END) subgroups (refer to key in top right-hand corner). This is done to illustrate the variance between both individuals and subgroup (Weissgerber, Milic, Winham & Garovic, 2015). * denotes $P < 0.05$ or a main effect of time from PPO1 and PPO2.

4.3 Physiological measures

4.3.1 Plasma bicarbonate, pH and lactate

4.3.1.1 Plasma bicarbonate

Plasma [HCO_3^-] was measured initially to assess the efficacy of the supplement loading protocol during BIC (Table 4.2). At rest plasma [HCO_3^-] was not significantly different between-interventions ($P > 0.05$). Plasma [HCO_3^-] was significantly higher in BIC at PPO1 compared to PLA ($P < 0.001$) due to a within-trial increase from rest to PPO1 of 3.4 ± 2.2 mmol.L^{-1} in BIC ($P < 0.001$) and decrease of 3.1 ± 1.8 mmol.L^{-1} in PLA ($P = 0.001$). Plasma [HCO_3^-] remained significantly higher between-interventions in BIC at PPO2 ($P < 0.001$) despite a decrease within-trials from PPO1 to PPO2 in PLA of 4.9 ± 2.6 mmol.L^{-1} ($P < 0.001$) and a 7.2 ± 2.7 mmol.L^{-1} decrease in BIC ($P < 0.001$).

4.3.1.2 Plasma pH

Plasma pH was not significantly different between-interventions at rest ($P > 0.05$). Plasma pH was significantly higher in BIC between-interventions at PPO1 ($P < 0.001$) because of a decrease within-trials in PLA (0.06 ± 0.04, $P < 0.001$) and increase in BIC (0.04 ± 0.03, $P = 0.01$) from rest to PPO1. A similar overall decrease from PPO1 to PPO2 (0.16 ± 0.12 PLA and 0.18 ± 0.10 pH units BIC, $P = 0.31$) meant that plasma pH remained significantly higher in BIC at PPO2 ($P < 0.001$). Individual responses to exercise showed that plasma pH reached levels as low as 6.84 pH units during PLA and 6.93 pH units during the BIC at PPO2.

4.3.1.3 Blood lactate

Blood [La^-] was not significantly different between-interventions at rest ($P > 0.05$) or PPO1 ($P > 0.05$). However, significant increases from PPO1 to PPO2 in both trials (12.2 ± 4.7 PLA, $P < 0.001$ and 15.3 ± 3.2 mmol.L^{-1} BIC, $P < 0.001$) led to a significantly higher blood [La^-] at

PPO2 in BIC (19.6 ± 4.5 vs. 22.1 ± 3.3 mmol.L^{-1}, P = 0.01). It is important to note that two participants recorded blood lactates exceeding 25 mmol.L^{-1} at PPO2 in BIC, with one of these participants also exceeding 25 mmol.L^{-1} in PLA.

4.3.1.4 Relationships between performance and plasma bicarbonate, pH and lactate

No significant linear relationship was found between the percentage decrease of PPO and plasma pH at PPO2 (Fig. 4.2A) or the absolute change in plasma pH from PPO1 to PPO2 (Fig. 4.2B). No significant linear relationship was found between the percentage decrease of PPO and the change in blood [La$^-$] from PPO1 to PPO2 (r = 0.32, P = 0.13), peak blood [La$^-$] at PPO2 (r = -0.051, P > 0.05), plasma [HCO_3^-] at PPO2 (r = 0.024, P > 0.05) or the absolute change in plasma [HCO_3^-] from PPO1 to PPO2 (r = -0.35, P > 0.05).

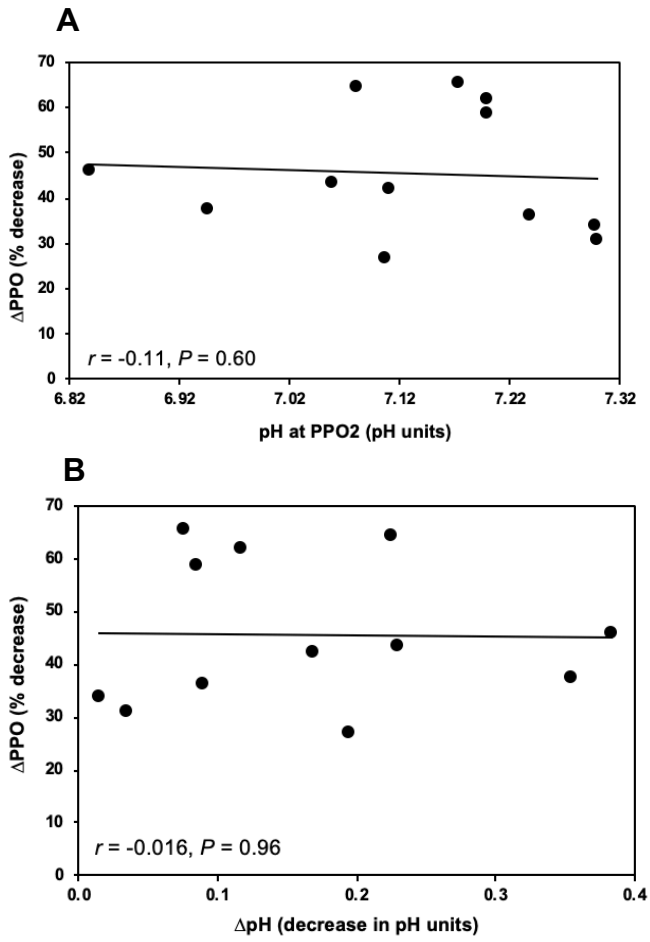

Figure 4.2. Pearson's correlation - Scatterplot comparing percentage change (Δ) in PPO from initial PPO (PPO1) and final PPO (PPO2) and A) plasma pH at PPO2, B) the absolute Δ in

plasma pH from PPO1 to PPO2. Strength of association: $0.1 \leq r \leq 0.29$ = weak positive correlation, $-0.1 \leq r \leq -0.29$ = weak negative correlation, $0.3 \leq r \leq 0.59$ = moderate positive correlation $-0.3 \leq r \leq -0.59$ = moderate negative correlation, $r \geq 0.7$ = strong positive correlation, $r \leq -0.7$ = strong negative correlation (Akoglu, 2018).

4.3.2 Plasma electrolytes

4.3.2.1 Plasma potassium

Plasma [K^+] increased from similar concentrations at rest ($P > 0.05$) to PPO1 within-trials in both PLA (1.1 ± 0.6 mmol.L^{-1}, $P < 0.001$) and BIC (0.8 ± 0.7 mmol.L^{-1}, $P = 0.01$). However, this rise was significantly greater in the PLA, meaning plasma [K^+] was higher in PLA between-interventions at PPO1 ($P = 0.02$). No significant increase was seen within-trials between PPO1 and PPO2 ($P = 0.12$ BIC and $P > 0.05$ PLA). Individual plasma K^+ responses varied, with some rising from PPO1 to PPO2, while others decreased over the same time points. Notwithstanding, several participants reached levels between 6.4-6.7 mmol.L^{-1} at PPO2.

4.3.2.2 Plasma sodium

Higher plasma [Na^+] were seen between-interventions in BIC at both PPO1 ($P = 0.001$) and PPO2 ($P = 0.006$). This was due to a larger within-trials increase from rest to PPO1 of 1.7 ± 1.4 PLA ($P = 0.005$) vs. 3.6 ± 0.8 mmol.L^{-1} BIC ($P < 0.001$).

4.3.2.3 Plasma calcium

Plasma [Ca^{2+}] was higher between-interventions in PLA at PPO1 ($P < 0.001$) and PPO2 ($P < 0.001$). This was due to a significant increase within-trials from rest to PPO1 of 2.2 ± 2.4 mmol.L^{-1} PLA ($P = 0.04$) and significant decrease of 8.7 ± 3.7 mmol.L^{-1} in BIC ($P < 0.001$). However, no further significant changes were seen within-trials from PPO1 to PPO2 in either PLA ($P = 0.08$) or BIC ($P = 0.11$).

4.3.2.4 Relationships between performance and plasma electrolytes

Significant moderate negative linear relationships were found between the percentage decrease of PPO and the absolute change in plasma [K^+] from PPO1 to PPO2 as well as the absolute change in plasma [Ca^{2+}] from PPO1 to PPO2 (r = -0.61, P = 0.36). No significant relationship was seen between plasma [Na^+] and the percentage decrease of PPO (r = 0.18, P = 0.42).

Table 4. 2. Overall blood measures.

Overall (n = 12)	**PLA**			**BIC**		
	Rest	**PPO1**	**PPO2**	**Rest**	**PPO1**	**PPO2**
[HCO_3^-] (mmol.L^{-1})	30.1 ± 1.3	27.0 ± 2.2†	22.1 ± 2.7*	30.5 ± 1.7	33.9 ± 2.7¥†	26.7 ± 2.9¥*
pH (pH units)	7.35 ± 0.02	7.29 ± 0.03†	7.13 ± 0.14*	7.34 ± 0.03	7.38 ± 0.05¥†	7.20 ± 0.12¥*
[La^-] (mmol.L^{-1})	1.2 ± 0.5	7.4 ± 2.7†	19.6 ± 4.5*	1.2 ± 0.3	6.8 ± 1.9†	22.1 ± 3.3¥*
[K^+] (mmol.L^{-1})	4.2 ± 0.3	5.3 ± 0.5†	5.4 ± 0.7	4.3 ± 0.4	5.1 ± 0.6¥†	5.3 ± 0.7
[Na^+] (mmol.L^{-1})	142 ± 1	143 ± 2†	146 ± 3*	142 ± 1	145 ± 1¥†	148 ± 2¥*
[Ca^{2+}] (mmol.L^{-1})	1.28 ± 0.02	1.31 ± 0.04†	1.32 ± 0.05	1.29 ± 0.03	1.18 ± 0.05¥†	1.20 ± 0.04¥

Values are presented as mean ± standard deviation. Abbreviations: PLA = Placebo intervention trial; BIC = Sodium bicarbonate intervention trial; [HCO_3^-] = Plasma bicarbonate concentration; [La^-] = blood lactate concentration; [K^+] = Plasma potassium concentration; [Na^+] = Plasma sodium concentration; [Ca^{2+}] = Plasma calcium concentration. ¥ denotes P < 0.05 difference between PLA and BIC at the same timepoint; † denotes P < 0.05 differences within trial between rest and PPO1; * denotes P < 0.05 differences within trial between PPO1 and PPO2; All P values were adjusted post hoc using the Bonferroni method.

4.3.3 Peripheral oxygen saturation and heart rate

4.3.3.1 Peripheral oxygen saturation

No significant differences between-trials were seen in SpO_2 (Fig. 4.3) over the course of the trial (P > 0.05). A significant within-trials decrease (P = 0.02) during the fixed-intensity time-trial from rest to PPO2 was present in both PLA (99.6 ± 0.7 to 96.0 ± 2.5%) and BIC (99.5 0.8 to 96.3 ± 3.7%). Individual responses showed that only one participant fell below 90% (87%) throughout testing, which was achieved during BIC. A moderate negative linear

relationship was found between the percentage decrease of PPO and the change in SpO_2 from rest to PPO2 (Fig. 4.4).

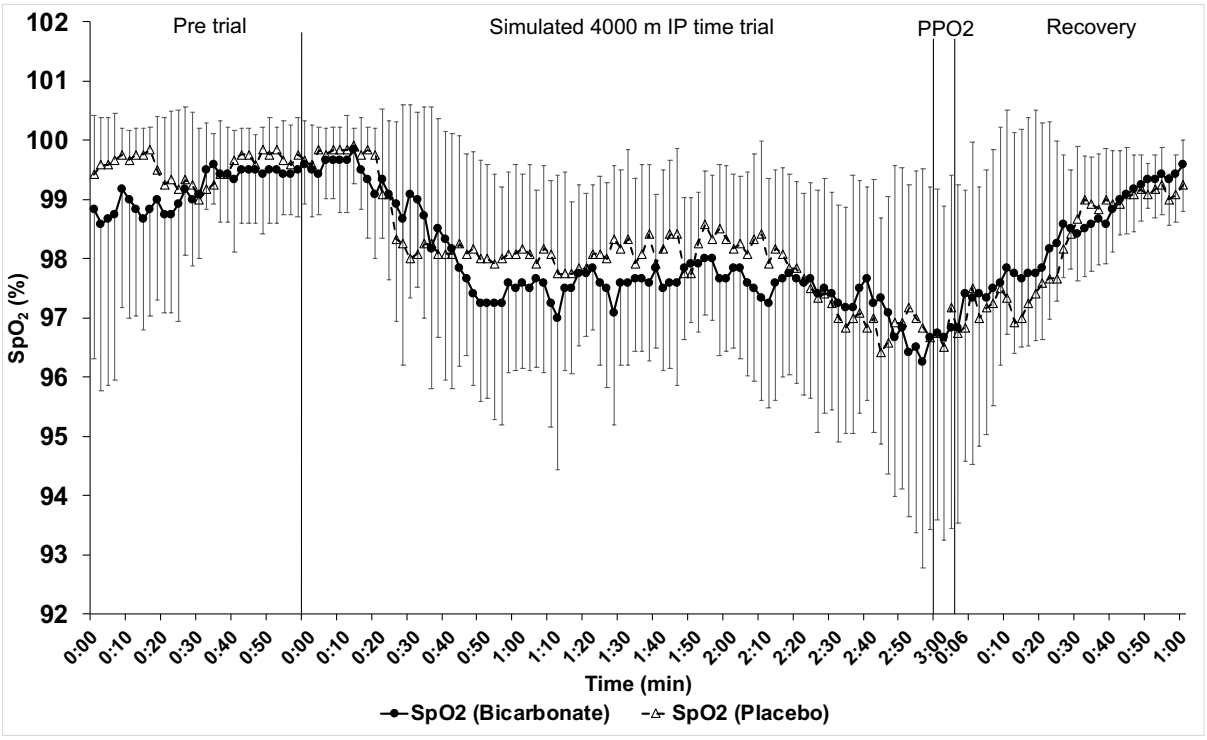

Figure 4. 3. Time course of SpO_2 throughout the 3-min fixed-intensity time-trial and final PPO (PPO2) as well as 60 s pre and post-exercise - Data was recorded every 2 s and was averaged over all participants for both PLA and BIC. Error bars indicate the standard deviation at a particular timepoint. Includes the fixed-intensity time-trial and PPO2, as well as 60 s pre and post-exercise.

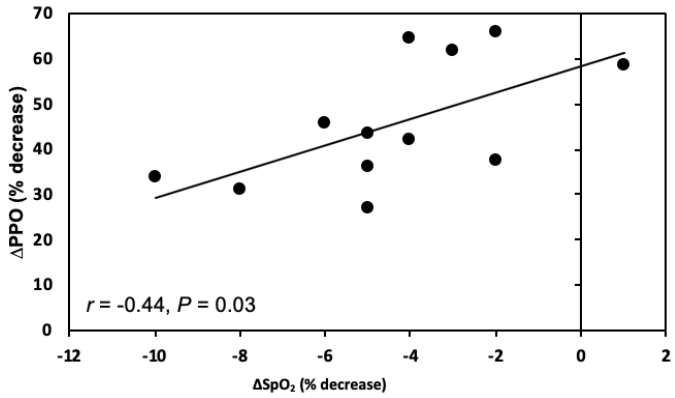

Figure 4. 4. Pearson's correlation – Scatterplot comparing the percentage change (Δ) in PPO from initial PPO (PPO1) and final PPO (PPO2) and the absolute decrease of SpO_2 from PPO1 to PPO2. Strength of association: $0.1 \leq r \leq 0.29$ = weak positive correlation, $-0.1 \leq r \leq -0.29$ = weak negative correlation, $0.3 \leq r \leq 0.59$ = moderate positive correlation $-0.3 \leq r \leq -0.59$ = moderate negative correlation, $r \geq 0.7$ = strong positive correlation, $r \leq -0.7$ = strong negative correlation (Akoglu, 2018).

4.3.3.2 Heart rate

Heart rate was also measured throughout the fixed-intensity time-trial and PPO2 (Fig. 4.5). No significant differences between PLA and BIC were seen over the course of the trial ($P > 0.05$). Within-trials heart rate increased significantly ($P < 0.001$) throughout the fixed-intensity time-trial during both PLA (108 ± 23 to 182 ± 12 beat.min^{-1}) and BIC (108 ± 16 to 181 ± 11 beat.min^{-1}). These heart rates were 96.9% and 97.2% of the maximum values achieved in the graded step tests during initial testing (Table 4.1).

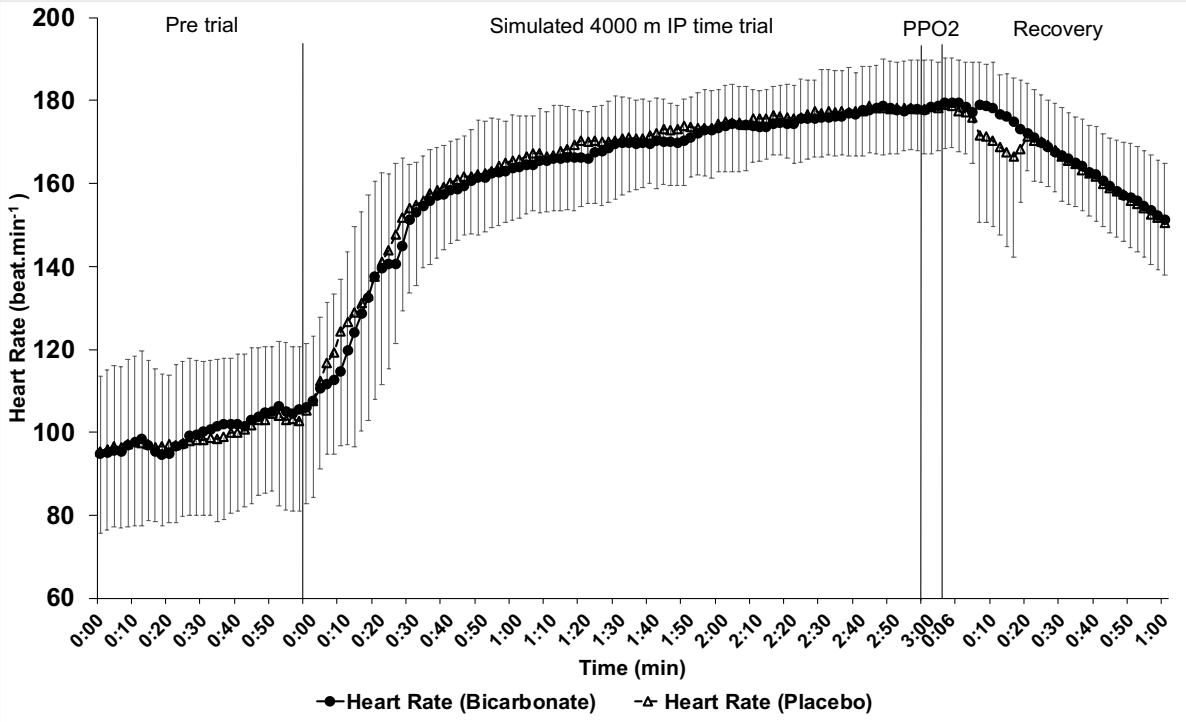

Figure 4.5. Time course of heart rate throughout the 3-min fixed-intensity time-trial and final PPO (PPO2) as well as 60 s pre and post-exercise - Data was recorded every 2 s and was averaged over all participants for both PLA and BIC. Error bars indicate the standard deviation at a particular timepoint. Includes the fixed-intensity time-trial and PPO2, as well as 60 s pre and post-exercise.

4.4 Gastric discomfort and condition blinding

Self-reported gastric discomfort scores were significantly higher in BIC at two points, 50 min post-ingestion (2 ± 1 vs. 1 ± 0 AU, $P = 0.038$) and 60 min post-ingestion (2 ± 1 vs. 1 ± 0 AU, $P = 0.027$. No reports of headache, diarrhoea or serve gastric discomfort were highlighted. Analysis of post-testing participant responses indicated that 92% of participants guessed the correct session for the BIC.

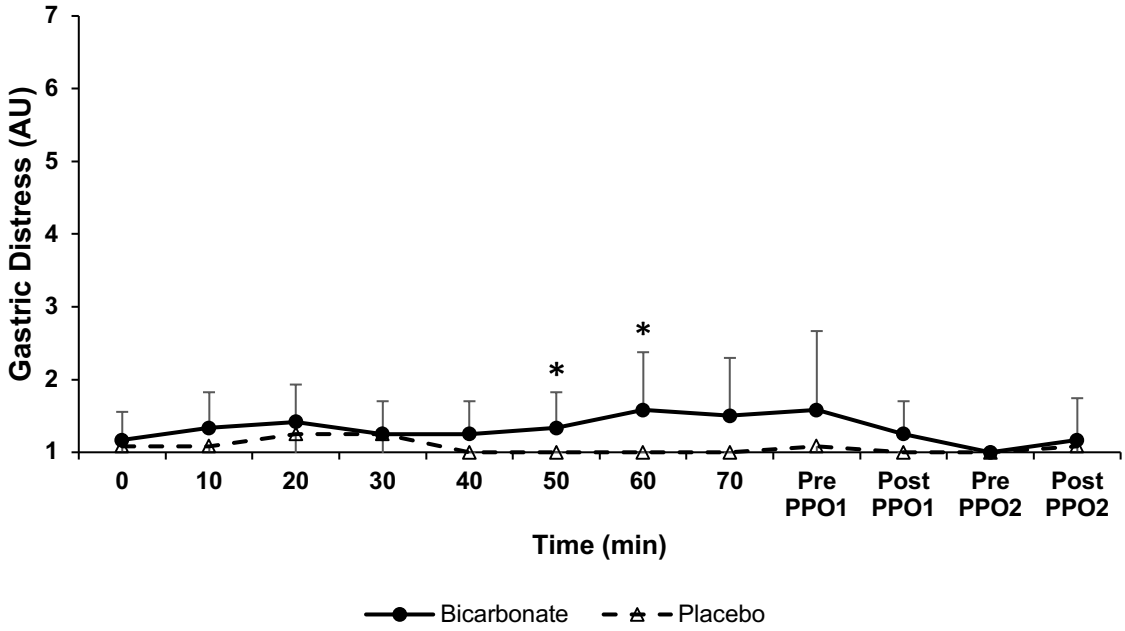

Figure 4. 6. Gastric Discomfort (1-7, AU) measured over the post-ingestion period in PLA and BIC - Data is displayed for the overall mean of both PLA and BIC at each timepoint. Error bars indicate the standard deviation at a particular timepoint. Reported on a scale of 1 = no problem, 2 = minimal problem, 3 = mild problem, 4 = moderate problem, 5 = moderately severe problem, 6 = severe problem, 7 = very severe. Scale used in testing is attached as Appendix B.5 (Van Zanten et al., 2006). * denotes $P < 0.05$ for difference between PLA and BIC at the same timepoint.

4.5 Subgroup analysis

4.5.1 Performance measures

Comparisons between END and SPR showed PPO was significantly higher in SPR at PPO1 (1231 ± 267 SPR vs. 827 ± 201 W END, $P = 0.001$). This initial difference in PPO was due to higher peak torques in SPR at PPO1 (83.4 ± 17.4 SPR vs. 59.6 ± 11.4 $N.m^{-1}$ END, $P = 0.001$) but not peak cadence (190 ± 9 SPR vs. 185 ± 11 $rev.min^{-1}$ END, $P = 0.36$) (Fig. 4.1B & C). However, no significant difference in PPO was seen between subgroups at PPO2 (511 ± 125 SPR vs. 567 ± 100 W END, $P > 0.05$) (Fig. 4.1A). This resulted in a greater percentage decrease of PPO in SPR (51.6 ± 14.8% SPR vs. 37.9 ± 5.5% END, $P = 0.01$). No between-interventions differences were seen in either subgroup concerning PPO ($P > 0.05$).

4.5.2 Physiological measures

Subgroup plasma measures were significantly different over several time points (Table 4.3). Firstly, END had significantly higher plasma [HCO_3^-] at rest ($P = 0.005$). Plasma pH was higher in SPR at PPO2 ($P = 0.002$). Blood [La^-] was higher in END at PPO2 ($P = 0.037$). No other between-subgroup differences were detected in any of the plasma electrolytes or SpO_2 ($P > 0.05$). No between-subgroup/ between-intervention/ within-trials interactions or between-subgroup/ between-intervention interactions were detected ($P > 0.05$).

Table 4. 3. Subgroup blood measures.

PLA	END (*n* = 6)			SPR (*n* = 6)		
	Rest	**PPO1**	**PPO2**	**Rest**	**PPO1**	**PPO2**
$[HCO_3^-]$ (mmol.L^{-1})	30.5 ± 1.0*	27.4 ± 1.8	21.0 ± 2.8	29.6 ± 1.5	26.6 ± 2.6	23.2 ± 2.2
pH (pH units)	7.35 ± 0.02	7.29 ± 0.04	7.05 ± 0.14*	7.35 ± 0.02	7.30 ± 0.03	7.21 ± 0.08
$[La^-]$ (mmol.L^{-1})	1.0 ± 0.3	7.0 ± 1.1	21.7 ± 2.7*	1.3 ± 0.6	7.8 ± 3.8	17.5 ± 5.1
$[K^+]$ (mmol.L^{-1})	4.3 ± 0.4	5.5 ± 0.4	5.5 ± 0.7	4.1 ± 0.2	5.1 ± 0.6	5.3 ± 0.8
$[Na^+]$ (mmol.L^{-1})	142 ± 1	143 ± 2	147 ± 4	142 ± 2	143 ± 2	145 ± 3
$[Ca^{2+}]$ (mmol.L^{-1})	1.27 ± 0.02	1.30 ± 0.02	1.32 ± 0.03	1.29 ± 0.03	1.32 ± 0.05	1.33 ± 0.06
SpO_2 (%)	99 ± 1	99 ± 1	94 ± 1	99 ± 1	99 ± 1	95 ± 4

BIC	END (*n* = 6)			SPR (*n* = 6)		
	Rest	**PPO1**	**PPO2**	**Rest**	**PPO1**	**PPO2**
$[HCO_3^-]$ (mmol.L^{-1})	31.8 ± 1.2*	34.9 ± 2.6	25.3 ± 2.8	29.2 ± 0.8	32.8 ± 2.5	28.2 ± 2.3
pH (pH units)	7.33 ± 0.03	7.37 ± 0.06	7.11 ± 0.11*	7.35 ± 0.02	7.38 ± 0.04	7.28 ± 0.06
$[La^-]$ (mmol.L^{-1})	1.0 ± 0.2	6.7 ± 1.7	24.0 ± 0.9*	1.5 ± 0.2	6.9 ± 2.25	20.2 ± 3.8
$[K^+]$ (mmol.L^{-1})	4.3 ± 0.5	5.2 ± 0.6	5.4 ± 0.6	4.3 ± 0.2	4.9 ± 0.7	5.2 ± 0.8
$[Na^+]$ (mmol.L^{-1})	141 ± 1	145 ± 1	149 ± 2	142 ± 1	146 ± 1	147 ± 1
$[Ca^{2+}]$ (mmol.L^{-1})	1.30 ± 0.02	1.19 ± 0.04	1.20 ± 0.03	1.28 ± 0.04	1.17 ± 0.07	1.19 ± 0.06
SpO_2 (%)	99 ± 1	99 ± 2	93 ± 4	98 ± 1	99 ± 1	97 ± 1

Values are presented as mean ± standard deviation. Abbreviations: END = Endurance subgroup; SPR = Sprint subgroup; PLA = Placebo intervention trial; BIC = Sodium bicarbonate intervention trial; $[HCO_3^-]$ = Plasma bicarbonate concentration; $[La^-]$ = Blood lactate concentration; $[K^+]$ = Plasma potassium concentration; $[Na^+]$ = Plasma sodium concentration; $[Ca^{2+}]$ = Plasma calcium concentration; SpO_2 = peripheral oxygen saturation. * denotes $P < 0.05$ for between subgroup differences at the same timepoints. All *P* values were adjusted post hoc using the Bonferroni method.

4.6 Anaerobic power reserve

The subgroup differences above (Section 4.5) illustrate the recruitment of two distinct elite cohorts. This provides rationale for the analysis of relationships between the APR and performance and physiological variables (significant correlations are shown in Fig. 4.7). A strong positive correlation was found between the APR and the percentage decrease of PPO (Fig. 4.7A). The absolute increase in blood [La^-] from PPO1 to PPO2 (Fig. 4.7B) was found to have a significant but negative moderate correlation with APR. Additionally, a moderate positive correlation was found between APR and the absolute decrease in plasma [HCO_3^-] from PPO1 to PPO2 (Fig. 4.7C). Further analysis also illustrated that the absolute changes from PPO1 to PPO2 in pH ($r = 0.52$, $P = 0.08$) and SpO_2 ($r = 0.50$, $P = 0.098$) were both seen to be approaching significance. However, the absolute changes in plasma [Na^+] ($P = 0.11$), plasma [K^+] ($P = 0.15$) and plasma [Ca^{2+}] ($P > 0.05$) concentrations all failed to significantly correlate with APR.

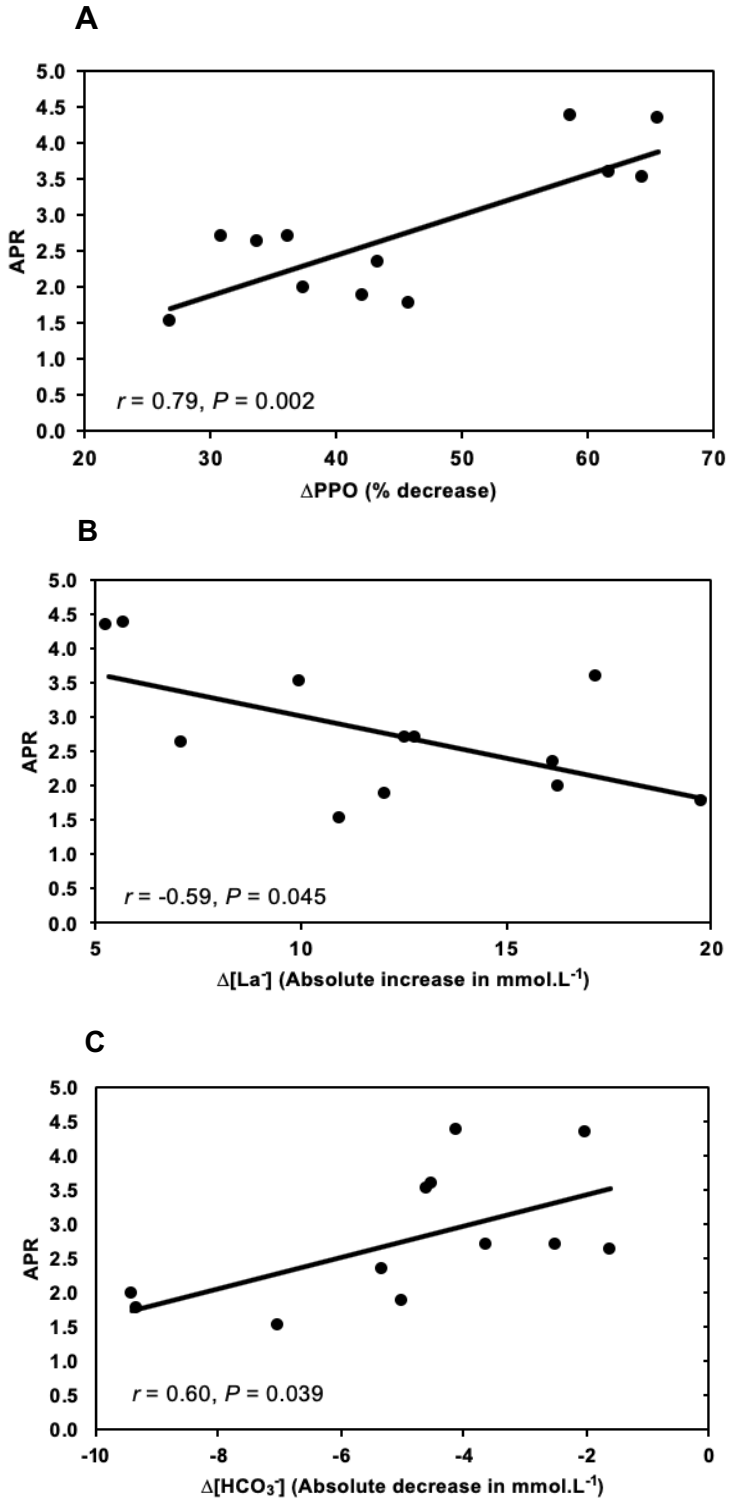

Figure 4. 7. Pearson's correlation – Scatterplot comparing anaerobic power reserve and A) percentage change (Δ) in PPO from initial PPO (PPO1) and final PPO (PPO2) B) absolute Δ in blood lactate concentration ($[La^-]$) from PPO1 to PPO2 and C) absolute Δ in plasma bicarbonate concentration ($[HCO_3^-]$) from PPO1 to PPO2. Strength of association: $0.1 \leq r \leq 0.29$ = weak positive correlation, $-0.1 \leq r \leq -0.29$ = weak negative correlation, $0.3 \leq r \leq 0.59$ = moderate positive correlation $-0.3 \leq r \leq -0.59$ = moderate negative correlation, $r \geq 0.7$ = strong positive correlation, $r \leq -0.7$ = strong negative correlation (Akoglu, 2018).

Chapter 5 – Discussion

5.1 The effect of exercise-induced plasma acidosis on the decrease in peak power output

The primary aim of this study was to determine the effect of exercise-induced plasma acidosis on the percentage decrease of PPO. Results demonstrated that a 3-min fixed-intensity cycling time-trial simulating ~75% of a 4000-m of individual pursuit successfully induced plasma acidosis, with plasma pH being significantly higher at PPO2 in BIC compared with PLA (Table 4.2). However, no significant differences were seen between PLA and BIC in mechanical performance variables such as PPO, peak torque or peak cadence (Fig. 4.1). These findings therefore do not support the notion that exercise-induced plasma acidosis plays a significant role in the ability to produce PPO over the concluding stages of a simulated 4000-m individual pursuit. This conclusion is further strengthened by a lack of a linear relationship between the percentage decrease of PPO and either the absolute end-plasma pH at PPO2 (Fig. 4.2A) or the absolute decrease in plasma pH from PPO1 to PPO2 (Fig. 4.2B). Examining the separate proposed mechanisms of $NaHCO_3$'s ergogenic effects shows that desired plasma changes were achieved, yet these did not enhance performance. Firstly, a significantly higher plasma [HCO_3^-] was achieved in the BIC post-ingestion of $NaHCO_3$ (measured at PPO1) (Table 4.2), illustrating the effectiveness of the loading protocol used. Additionally, it was shown that the use of $NaHCO_3$ also enhanced lactate efflux resulting in a higher absolute blood [La^-] measured at PPO2 in BIC (Table 4.2). An increased blood [La^-] in $NaHCO_3$ conditions has previously been suggested to signify a higher rate of anaerobic metabolism and subsequently improved performance, however the increased blood [La^-] was not found to improve performance in the current investigation (Fig. 4.1) (Bouissou et al., 2003; Hollidge-Horvat et al., 2000).

5.1.1 Mechanical Performance - Changes in peak power output, peak torque and peak cadence

Acidosis is generally believed to cause fatigue via its depressive effects on PPO (Fitts, 2016; Knuth et al., 2006). However, this research has primarily been based on animal models with a lack of human research exploring the effects of acidosis on PPO or more specifically its components, namely force and velocity (Westerblad, 2016). Therefore, results of the current investigation oppose the general belief that acidosis depresses PPO given the primary finding that there was no significant difference between interventions in the in the percentage decrease of PPO (Fig. 4.1A), despite a significantly higher plasma pH at PPO2 in BIC (Table 4.2). Although no significant difference was detected in performance between the two intervention trials, a significant percentage decrease of PPO did occur within-trials in both (Fig. 4.1A), hence considerable fatigue was observed. The decline in PPO seen in both PLA and BIC was found to be due to a significant decrease in peak cadence (Fig. 4.1B), with no difference within-trials seen in peak torques in both PLA and BIC (Fig. 4.1C). Given these findings concerning the reduction in peak cadence after a supramaximal cycling time-trial, it can therefore be postulated that the mechanism of fatigue directly affected shortening velocity or the reduced ability of the muscle to relax (Knicker, Renshaw, Oldham & Cairns, 2011; Fitts, 2016).

Plasma acidosis has been well researched in humans and specifically in supramaximal cycling performance, through the use of $NaHCO_3$ and NH_4Cl supplementation over close-ended time-trials, time-to-exhaustion trials and repeated-sprint protocols. In the majority of these studies performance has been measured using time-to-completion, total work done, total distance covered, mean power output and fatigue index (expressed as the decline in power output divided by the time interval between PPO and minimum power) (Kent, 2006).

Acknowledging these constraints, research using elite and well-trained cohorts over similar durations to the present study have suggested a more consistent ergogenic response of $NaHCO_3$ supplementation (Table 2.1). Even though this evidence exists over such performance measures, the effect of $NaHCO_3$ has not yet been studied using percentage changes in PPO. Instead, prior research measuring PPO in cycling has mainly measured this over the early stages of an exercise protocol (Deb et al., 2017; Driller et al., 2012; McCartney, Heigenhauser & Jones, 1983; McNaughton, 1992; Vanhatalo et al., 2010). In doing so this assesses the ATP-PCr system and neuromuscular capacity over the early stages before an acidosis has occurred. Given the importance of the end-spurt in supramaximal events, it is critical to understand how alactic based activity is influenced by fatigue and therefore changes to acid-base balance (Christensen et al., 2017). Therefore, the results of the current investigation provide novel insights. Firstly, concerning the effect of exercise-induced plasma acidosis and $NaHCO_3$ supplementation on the ability to produce PPO's later into supramaximal exercise and secondly examining this in an elite cohort.

Although no studies have used the change in PPO after a supramaximal exercise bout as a measure of fatigue in the study of plasma acidosis, two studies have examined the effect of exercise-induced metabolic acidosis on the components of PPO, namely force/torque and velocity. This previous research has suggested that $NaHCO_3$ supplementation can reduce the negative effects of acidosis on the rate of force development (Siegler et al., 2013) and peak and total torque production (Verbitsky et al., 1997). Although these studies appear to show complimentary findings regarding the two separate components of power, they also conflicted with regards to the effect of $NaHCO_3$ on peak torque development. In agreement with the present study (Fig. 4.1B), Siegler et al. (2013) found no effect of $NaHCO_3$ on peak torque development, whereas Verbitsky et al. (1997) saw significant increases in both peak and total

torque development. Several methodological differences may explain these conflicting results. Firstly, the repeated-sprint protocol used by Siegler and colleagues differed from Verbitsky and colleagues as well as the current study, which both captured mechanical measures before and after a fixed supramaximal time-trial. Additionally, the contraction types used to measure performance differed across all protocols i.e. maximum voluntary isometric contraction, muscle stimulation and a dynamic sport-specific sprint. Finally, the studies differed regarding the length of performance measurement. In this case Siegler et al. measured their maximal voluntary contraction over a 5-8-s period which was similar to the 6 s used to measure PPO in the current study. Alternatively, the study by Verbitsky et al. measured total torque pre and post-exercise over a 2-min stimulation protocol. In their study Verbitsky et al. saw a higher peak and total torque in the $NaHCO_3$ trial due to a delayed peak torque measured 60 s into the 2-min stimulation. Considering these methodological differences across the three studies it is difficult to draw certain conclusions. However, the delayed peak torque seen in Verbitsky et al. may suggest that the 6 s allotted for the measurement of PPO in the current investigation or 5-8 s Siegler et al. (2013) may not have been long enough to witness differences in peak torques between BIC and PLA. This potentially suggests that the attenuation of plasma acidosis might induce an enhanced performance in end-spurts of greater than 60 s, common in middle-distance running events such as the 1-mile run (Tucker, Lambert & Noakes, 2009). Alternatively, shorter explosive outputs, such as those used in the current study and Siegler et al. suggest that reduced shortening velocity is the primary mechanism of fatigue.

5.1.2 Effectiveness of sodium bicarbonate loading procedure

The ability to conclusively study the effects of exercise-induced plasma acidosis on changes in PPO was dependent on the successful attenuation of plasma acidosis through $NaHCO_3$ supplementation. Given this, the change in plasma [HCO_3^-] from rest to PPO1 highlighted the

success of the supplement loading protocol to effectively augment plasma [HCO_3^-] (Table 4.2). Changes in plasma [HCO_3^-] have been established as the primary marker for measurement of successful $NaHCO_3$ loading due to the high intraindividual reproducibility shown previously (de Araujo Dias et al., 2015; Gough et al. 2017). In the current investigation an increase in plasma [HCO_3^-] from rest to PPO1 of 3.4 mmol.L^{-1} was achieved (Table 4.2). Coupling this increase seen in BIC with the 3.1 mmol.L^{-1} reduction seen over the same time points in PLA, resulted in a difference of 6.5 mmol.L^{-1} between the two interventions at PPO1 ($P < 0.001$). Viewing the results in this way provides a net difference in plasma [HCO_3^-] between PLA and BIC in excess of the 6 mmol.L^{-1} threshold believed to enhance the mechanisms that elicit $NaHCO_3$'s ergogenic effects (Bishop & Claudius, 2005; Carr et al., 2011; McNaughton & Cedaro, 1991; Van Montfoort et al., 2004; Wilkes, Gledhill, & Smyth, 1983). In addition to this, post-trial measures also highlighted desired plasma changes, with plasma pH significantly higher at PPO2 in BIC compared with PLA (7.13 PLA vs. 7.20 pH units BIC, $P < 0.001$). A higher plasma [HCO_3^-] was also seen in BIC at PPO2, despite a greater absolute decrease from PPO1 to PPO2 in BIC (4.9 PLA vs. 7.2 mmol.L^{-1} BIC). Taken together this evidence speaks to the effectiveness of the $NaHCO_3$ loading protocol and its ability to create the required plasma changes needed to assess differences in plasma pH on performance. As the model was successful, it can therefore be inferred that any changes in performance (i.e. PPO, peak torque or peak cadence) between BIC and PLA can be directly attributed to the difference in plasma acidosis.

5.1.3 The effect of gastric discomfort and participant blinding on performance

It should be acknowledged that $NaHCO_3$ supplementation can induce gastric discomfort (Carr et al., 2011; Gough et al., 2017; Jones et al., 2016; Kahle et al., 2013; Mohr, 2017; Saunders et al., 2014). This gastric discomfort has been suggested to help distinguish those who experience

an ergogenic effect from those who do not (Jones et al. 2016; Saunders et al., 2014; Matson & Tran, 1993). Considering this gastric discomfort was successfully minimized throughout testing with no participants reporting anything more than a mild problem (Fig. 4.6). Therefore, efforts made to reduce the occurrence of gastric discomfort, such as the use of pilot testing to assess the best method of administration, the use of gelatine capsules, and consistent monitoring throughout intervention trials, all reduced the potential negative impact of excessive gastric discomfort on participant health and performance.

A further consideration is whether the double-blind condition was upheld throughout the study. Participant blinding was assessed because studies have suggested the ergogenic effects of $NaHCO_3$ could be explained by the expectancy of its erogenicity (Higgins & Shabir, 2016; McClung & Collins, 2007). Although no participant experienced more than a mild gastric disturbance, there were however differences between interventions regarding gastric discomfort (Fig. 4.6). This may have impacted participant blinding, since 92% of participants guessed the correct session for the BIC (Section 4.4). Despite this observation there was no difference between PLA and BIC in terms of performance (Fig. 4.1). In line with these findings Carr, Gore & Dawson (2011) reported that 81% of their participants were able to guess the correct condition, yet no overall ergogenic effect was seen during a 2-km rowing time-trial. Also, Tobias et al. (2013) reported an overall ergogenic effect in repeated 30-s maximal sprints on an upper-body erg with 61% of participants guessing the correct condition. These combined results suggest that an expectancy effect with $NaHCO_3$ supplementation was not present in this model. Despite no apparent effects of expectancy on the results of the present investigation, the inability to completely blind participants is a limitation that should be addressed in future research. Therefore, future blinding methods should employ taste matched placebo ingredients.

A good example of this is the use of NaCl, which alone has been shown to not effect performance (Driller et al., 2012).

5.1.4 Proposals for the lack of ergogenic effect

Considering previous research using $NaHCO_3$ in supramaximal cycling performance several other factors may have contributed to the lack of ergogenic effect seen in the current study. Firstly, the well-trained nature of the participants and their high resting plasma [HCO_3^-] may have offset the augmented extracellular buffer capacity of the $NaHCO_3$. Training has been shown to improve buffer capacity allowing for better control of severe disturbances in acid-base balance (McNaughton et al., 2016). With high resting plasma [HCO_3^-] of ~30 mmol.L^{-1} (Table 4.1) it appears that the participants had a robust extracellular buffer capacity prior to supplementation. This is evident when considering studies in which participants with lower resting plasma [HCO_3^-] (e.g. ~25 mmol.L^{-1}) have experienced increases in plasma [HCO_3^-] to similar average resting concentrations of those seen in the current investigation (Costill et al., 1984; Hobson et al., 2013; Siegler & Hirscher, 2010). Alternatively, several others have shown no ergogenic effect when participants have had similar resting levels to those in the current study (Callahan et al., 2015; de Araujo Dias et al., 2015; Linderman et al., 1992; Raymer et al., 2004). Therefore, by virtue of their trained status participants were potentially able to self-regulate the extreme acid loads without the enhancement of $NaHCO_3$.

Another proposed mechanism of action of $NaHCO_3$ involves the enhanced transmembrane transport of H^+ through a widening of the intracellular and extracellular pH gradient. Best shown in studies inducing metabolic extracellular acidosis via NH_4Cl supplementation, these studies have shown a more pronounced reduction in performance coupled with a reduced pH gradient and lowered peak blood [La^-] at exhaustion (Brien, 1982; Correia-Oliveira et al., 2017; Robergs et al., 2005; Jones, Sutton, Taylor & Toews, 1977; Sutton, Jones & Toews, 1981). In

this case an induced metabolic acidosis could perceivably blunt the regulatory mechanisms that rely on pH gradients to co-transport lactate and H^+ out of the muscle cell (Juel, 1997). It has been shown that MCT proteins are a prominent bidirectional transporter of both lactate and H^+ (Brown & Brooks, 1994). Therefore, it is possible to assume that the increase in peak blood [La^-] seen post-exercise is related to the effect of $NaHCO_3$ on improved lactate and H^+ regulation (Table 4.2). Also considering that the sarcolemma is largely impermeable to $NaHCO_3$ transport, it is therefore conceivable that $NaHCO_3$ could help lower intramuscular pH via this improved pH gradient and upregulation of MCT expression (Mainwood & Cechetto, 1980; Mainwood & Renaud, 1985; Sahlin, 2014). This has been shown by Raymer et al. (2004) who, through continuous monitoring of phosphorus-31 magnetic resonance spectroscopy, found that $NaHCO_3$ lowered intramuscular pH during exercise without a change in total H^+ production. MCTs have also been shown to increase in content with training (Dubouchaud, Butterfield, Wolfel, Bergman, & Brooks, 2000; Thomas, Bishop, Moore-Morris & Mercier, 2007), and their content within muscle has also been negatively correlated with improved exercise performance under alkalosis (Messonnier et al., 2007). Therefore, in this study's cohort of well-trained participants it could be speculated that, due to their training status, participants could have already possessed a robust trans-sarcolemma transport system which could have nullified the ergogenic effects $NaHCO_3$ supplementation.

In summary, despite successful manipulation of plasma [HCO_3^-] (Table 4.2), no significant differences were seen in mechanical performance measures such as PPO, peak torque and peak cadence between PLA and BIC (Fig. 4.1). Several proposals have been put forth to explain why $NaHCO_3$ was not able to elicit an ergogenic effect. Despite these the lack of a relationship between the decrease in PPO and either the absolute plasma pH at PPO2 (Fig. 4.2A) or the absolute change in plasma pH (Fig. 4.2B) suggest that plasma acidosis does not affect the decline in PPO seen as a result of a 3-min fixed-intensity supramaximal cycling time-trial.

5.2 Mechanisms for fatigue (decline in peak power output)

The second aim of the study was to determine whether other plasma based physiological variables, aside from plasma acidosis, could be identified as contributing mechanisms to fatigue. The results of the current investigation suggest that changes in plasma [K^+], [Ca^{2+}] and [Na^+], and SpO_2 were not involved in the fatigue seen as a result of the fixed-intensity simulated 4000-m individual pursuit time-trial. This was evidenced by the lack of significant correlations seen between the percentage decrease of PPO and these physiological variables (Section 4.3.2.4). Given that fatigue in this study was isolated to reductions in peak cadence, and therefore shortening velocity, specific hypothesis can be made relating to the underlying mechanisms of fatigue. It has been suggested that shortening velocity is both peripherally and centrally dependent (Aagaard, Simonsen, Andersen, Magnusson & Dyhre-Poulsen, 2002). However, many of these variables are intracellular and were not measured in the current investigation. Therefore, in order to conclusively comment on these more research is required to fully elucidate the underlying causes of fatigue that occurred during this study.

5.2.1 Peripheral mechanisms underlying fatigue

5.2.1.1 Ionic proposals

From a peripheral perspective several changes to muscle physiology have been shown to inhibit the ability of muscle to rapidly shorten and then relax during exercise. Of these proposals' excessive changes in both intracellular and extracellular ions, namely K^+, Na^+ and Ca^{2+} can act to reduce muscle excitability and action potentials (Cairns & Lindinger, 2008). A prominent ionic hypothesis for fatigue is the rise in extracellular K^+, which has been shown to be directly proportional to exercise intensity (Sjogaard, Adams, & Saltin, 1985; Vøllestad, Hallen & Sejersted, 1994). In the present study using supramaximal intensity exercise plasma [K^+] rose

in both PLA and BIC, between rest and PPO1 (Table 4.2). However, several elements perceivably question if plasma K^+ had a role in fatigue. Firstly, there was no further significant increase between PPO1 and PPO2 in intervention trials, implying that a greater increase of plasma [K^+] was not the cause of the reduced PPO. Secondly, with a mean concentration at PPO2 of 5.4 mmol.L^{-1} in PLA and 5.3 mmol.L^{-1} in BIC, plasma changes did not appear large enough to elicit fatigue (Table 3). Similar concentrations have been seen by others who have studied plasma K^+ kinetics at 110% and 140% $\dot{V}O_2$peak (Vøllestad, Hallen & Sejersted, 1994). Several researchers have illustrated that plasma changes during exercise ranging from 7-14 mmol.L^{-1} are required to reduce the trans-sarcolemma K^+ gradient that negatively impacts resting membrane potentials (Cairns, Flatman & Clausen, 1995; Cairns, Hing, Slack, Mills & Loiselle, 1997; Juel, 1988; Renaud & Light, 1992; Overgaard, Nielsen & Clausen, 1997). Therefore, even taking individual responses into account, of which the highest individual measurement was 6.7 mmol.L^{-1}, concentrations were not high enough to elicit fatigue.

Peripheral fatigue is often attributed to impaired Ca^{2+} handling by the sarcoplasmic reticulum (reduced release and rate of uptake) and impaired myofilament function (reduced Ca^{2+} sensitivity or maximal force) (Westerbald & Allen, 2002). Although the present study was not able to measure intramuscular [Ca^{2+}], extracellular measures were taken (Table 4.2). Despite a significant reduction in plasma [Ca^{2+}] from rest to PPO1 in BIC (with no significant changes seen in PLA over the same timepoints), concentrations did not change in either BIC or PLA from PPO1 to PPO2. Although this provides evidence against plasma [Ca^{2+}]'s role in fatigue, it does show some interaction between Ca^{2+} and $NaHCO_3$ with exercise (measured from rest to PPO1 in BIC). It is speculated that in this case excess HCO_3^- simply interacted with Ca^{2+} to form $CaCO_3$, however no firm conclusions can be made (Hughes, Aurbach, Sharp & Marx, 1984). It must be highlighted that a potential interaction between K^+ and Ca^{2+} has been shown

in previous work concerning fatigue (Cairns et al., 2015). This proposal outlines that increases in [Ca^{2+}] have been shown to reinstate muscle force in K^+ depressed fibres. However, as there were no significant increases in plasma [Ca^{2+}] throughout exercise (Table 4.2), it therefore it is unlikely that this interaction had any effect on performance. Future research should look to expand on this finding by also examining changes in intramuscular [Ca^{2+}] which will give direct insights in to de-sensitivity to Ca^{2+} within the sarcoplasmic reticulum.

The final ion measured in this study was plasma Na^+. Plasma [Na^+] was seen to increase in both PLA and BIC in response to exercise (Table 4.2). Ingestion of Na^+ has been proposed to provide its own ergogenic benefits through increases in intravascular volume (Kozak-Collins et al., 1994; Saltin, 1964) or by assisting the efflux of H^+ from working muscle during exercise via the Na^+-H^+ exchange (McKenna, Bangsbo, & Renaud, 1985). However, despite an increased plasma [Na^+] throughout the present protocol (Table 4.2), the significant differences seen between BIC and PLA at both PPO1 and PPO2 were not accompanied by performance changes. To add to this was the lack of significant correlation seen between the change in plasma [Na^+] and the percentage decrease of PPO (Section 4.3.2.4). Therefore, it appears that increased plasma [Na^+] neither augmented performance nor was a cause of fatigue, as represented by the decline in PPO seen as a result of a 3-min fixed-intensity supramaximal cycling time-trial.

Other ionic factors could also be proposed to explain the fatigue that occurred. Given the low membrane permeability of HCO_3^-, it is likely that intracellular pH was largely unaffected during the BIC, aside from potential improvements to pH gradient. Therefore, in this investigation intracellular acidosis cannot be ruled out as a cause of fatigue (Mainwood & Cechetto, 1980; Mainwood & Renaud, 1985; Sahlin, 2014). It is believed that intracellular acidosis specifically reduces myosin ATPase and hence lowers shortening velocity (Fitts,

2016). It is well understood that pH drops in both muscle and plasma as a result of supramaximal exercise (Hermansen & Osnes, 1972). Therefore, the extreme reductions in plasma pH (reaching as low as 6.83 pH units in the PLA condition and 6.93 pH units in BIC in some individuals (Section 4.3.1.2)) suggest that a severe intracellular acidosis also occurred as a result of exercise in the current study. Another prominent ionic proposal, also known for its interaction with acidosis, is increased intracellular inorganic phosphate (P_i). P_i has been shown to diminish contractile function through reduced cross-bridge force production and myofibrillar Ca^{2+} de-sensitivity (Dahlstedt, Katz & Westerblad, 2001), the inhibition of energy-driven Ca^{2+} release, or Ca^{2+} reuptake in the sarcoplasmic reticulum (Dahlstedt, Westerblad , 2001; Duke & Steele, 2000; Giannesini et al., 2000; Kabbara & Allen, 2001). A synergistic interaction between acidosis and P_i has also been proposed to cause greater fatigue (Karatzaferi, Franks-Skiba & Cooke, 2008; Jarvis et al., 2018; Nelson, Debold & Fitts, 2014; Sundberg, Hunter & Bundle, 2016; Woodward & Debold, 2018). Predominantly shown in animal models, this interaction was recently observed in isolated human muscle fibres from the vastus lateralis (Sundberg et al., 2018). In this case researchers showed that the additive effects of these ions inhibited cross-bridge cycling and PPO. Unfortunately, no further speculation can be made due to the lack of intracellular measurements in the present study. However, this provides firm rationale for future research required to explore intracellular acidosis, P_i, or their interactive roles on the ability to sprint or produce PPO after supramaximal exercise.

In summary, although the ions measured throughout the study were not found to influence the decrease in PPO through interactions with changes in plasma acidosis, or association with the decrease in PPO, it must be noted that measures taken were only within plasma. Due to the role of intramuscular Ca^{2+} within the sarcoplasmic reticulum and the flux of Na^{+} and efflux of K^{+} in muscle contraction, future research will need to add to the plasma findings of the current

study with intramuscular measures in order to understand the complex changes that occur within muscle during contraction.

5.2.1.2 Metabolic proposals

From a metabolic perspective lactate has historically been linked to fatigue through its association (and dissociation) with rising H^+ in both muscle and plasma as a result of anaerobic glycolysis and ATP hydrolysis (Brooks, 2010; Robergs, Ghiasvand & Parker, 2004). However, these proposals have been mostly debunked. Research debunking the commonly held lactic acid hypothesis of fatigue have shown that the accumulation of lactate in blood and tissue is not dependent on states of anoxia (Connett, Gayeski & Honig, 1986; Linnarsson et al., 1974; Richardson et al., 1998). Additionally, the temporal relationship between lactate, acidosis and fatigue has also been questioned, as force depression and acid-base balance has been shown to deviate during recovery (Costill et al., 1983; Degroot et al., 1993; Hermansen & Osnes, 1972; Juel et al., 2004; Sahlin & Ren 1989; Saugen et al., 1997; Street, Bangsbo & Juel, 2001; Vøllestad et al., 1988; Westerblad & Allen, 1992). Finally, researchers have shown that lactate and H^+ separate at physiologically relevant temperatures and therefore must be viewed separately in the context of their roles in fatigue (Toffaletti, 1991), with lactate now viewed as a metabolic substrate instead of an agent in fatigue (Brooks, 2018). The current investigation has also shown no association between lactate and fatigue. Despite significant rises in blood [La^-] from PPO1 to PPO2 (Table 4.2) aligning with reductions in PPO in both intervention trials, such changes in blood [La^-] were not correlated with the decrease PPO from PPO1 to PPO2 (Section 4.3.1.4). Significant differences were also seen between intervention trials with the absolute blood [La^-] at PPO2 higher in BIC compared to PLA without any difference in the reduction of PPO from PPO1 to PPO2.

Two other potential metabolic factors not measured in the study but worth mentioning are intramuscular ATP and reactive oxygen species (ROS). Firstly, freely available ATP is critical for the continued function of muscle contraction. With reduced intramuscular ATP Na^+-K^+ pump activity is compromised, exacerbating membrane depolarization through greater K^+ efflux (Clausen, 2003; Dutka & Lamb, 2007b; Fink & Luttgau, 1976). However, a synergistic effect between acidosis and lowered intracellular ATP availability has also been shown to alter the voltage dependence of sarcolemma ClC-1 chloride channels which may potentially oppose fatigue (Bennetts, Parker & Cromer, 2007). Aside from this synergistic relationship with acidosis, the energetic requirements of the 6-s PPO sprint allow it to be postulated that the reduced intracellular availability of ATP may have been an agent of fatigue (Gaitanos, Williams, Boobis & Brooks, 1993). Secondly, ROS has been shown to increase with high-intensity exercise and an increased reliance on mitochondrial activity (Powers & Jackson, 2008). Such high levels of accumulated ROS have been suggested to elicit fatigue through several mechanisms. These include impaired muscle function through diminishing the Na^+-K^+ pump which enhances the negative effects of K^+ efflux, or through the stimulation of muscle afferents reducing motor drive (Allen, Lamb & Westerblad, 2008; Delliaux et al., 2009a; Delliaux et al., 2009b; McKenna et al., 2006; Reardon & Allen, 2009). Although the findings of the current study may suggest that K^+ played less of a role in the fatigue seen, the supra-spinal effects of either K^+ or ROS on muscle afferents cannot be ruled out. Given the large aerobic contribution required over supramaximal efforts >1-min it can be postulated that a large degree of oxidative stress was placed on participants in the current investigation (Craig & Norton, 2001). Therefore, although research using $NaHCO_3$ has shown little effect on oxidative stress during repeated-sprint exercise, ROS should be considered as an isolated fatigue agent in future research (Peart et al., 2013).

5.2.2 Central mechanisms underlying fatigue

Despite the suggestion that peripheral fatigue may have caused the reduction in peak cadence in the present study (Fig. 4.1C), several central mechanisms may have also contributed to this fatigue. One such proposal is the decrease in peripheral arterial oxygen saturation (Fig. 4.3). It is proposed that large decreases in oxygen saturation of ~10% induce cerebral hypoxia via the Bohr effect in which H^+ causes oxygen to offload from haemoglobin, and in turn cause a reduced central drive (Amann et al., 2006; Romer et al., 2006). A key study exploring the role of $NaHCO_3$ in the maintenance of oxygen saturation is that by Nielsen et al. (2002). In this study researchers administered conditions via a constant rate intravenous infusion of either 200-350mL of $NaHCO_3$ (1mM) or an equal volume isotonic saline. During a 2000-m maximal ergometer row a significant difference was seen in both the lowest plasma pH (7.0 placebo vs. 7.20 pH units $NaHCO_3$) and arterial oxygen saturation recorded during testing. In this case large reductions in SaO_2 were seen in the placebo trial (89%) while only moderate reductions were seen in the $NaHCO_3$ trial (~94%). These changes were accompanied by a significantly lower RPE and significantly faster time to competition of the 2000-m row in the $NaHCO_3$ trial. In accordance with the findings of Nielsen et al. oxygen saturations below 90% have also been associated with reduced performance capacity in hypoxia (Chapman, Stager, Tanner, Stray-Gundersen & Levine, 2011; Deb et al., 2017). Given this evidence, it appears that the levels of SpO_2 seen throughout the course of the fixed-intensity time-trial in the current study (Fig. 4.3) may not have been excessive enough to suggest it played a significant role in fatigue.

Despite not achieving the low levels shown to significantly impair exercise performance, the lack of a significant difference in SpO_2 between interventions may have been a factor in the lack of performance difference seen between-interventions. Given this lack of difference in the levels of SpO_2 achieved during exercise (Fig. 4.3), despite significant differences in pH at

PPO2 between PLA and BIC (Table 4.2), one could speculate that a key reason for the lack of an ergogenic effect seen in the BIC could have been due to the inability of $NaHCO_3$ to effectively attenuate the drop in SpO_2. This is strengthened by research conducted by Thomas et al. (2016) which observed a significant difference in mean power output over 70 s of maximal cycling, despite not achieving the low levels of oxygen saturation highlighted by previous investigations (Thomas et al., 2016). Thomas and colleagues saw similar levels seen in the current study, however, this study design was able to achieve significant differences between the levels of SpO_2 achieved during exercise (94 ± 1.3 placebo vs. 96.9 ± 0.8% $NaHCO_3$). Despite no differences seen between-interventions in SpO_2, which aligned with a lack of significant difference in performance measures in the current study, there is still suggestion that SpO_2 was not a factor in fatigue. This is evidenced by the moderate negative relationship found between levels of SpO_2 and the decrease in PPO (r = -0.442, P = 0.03, Fig. 4.4). This seemingly counterintuitive relationship implied that those who experienced greater levels of fatigue also maintained higher levels of SpO_2 throughout exercise. Although unlikely to be the cause of fatigue, no definitive answers can be reached regarding SpO_2 due to the nature of correlations, the lack of excessive oxygen desaturation, or because no significant difference was seen between interventions in the study.

Changes in the central drive to muscle during supramaximal cycling have been studied and attributed to changes in anaerobic contribution to exercise (Bundle, Ernst, Bellizzi, Wright & Weyand, 2006; Sundberg & Bundle, 2015; Sundberg, Hunter & Bundle, 2016). However, none have studied it in the context of changes to acid-base balance via $NaHCO_3$. Measured via surface electromyography, it has been shown that as power outputs increase above $p\dot{V}O_2peak$ the rate of neuromuscular recruitment also increases from the start of exercise until exhaustion (Bundle, Ernst, Bellizzi, Wright & Weyand, 2006; Sundberg & Bundle, 2015; Sundberg, Hunter & Bundle, 2016). Researchers have speculated that this rise in compensatory

neuromuscular recruitment is due to an increased reliance on anaerobic energy, irrespective of the absolute or relative amount of force generated by the muscle. Given this it would appear that central drive would be a key area of study using $NaHCO_3$ loading. However, evidence is lacking in modalities such as cycling, running, swimming and rowing. Instead studies have looked at the effect of $NaHCO_3$ and central drive in isometric contractions using small muscle groups (Siegler, 2015; Siegler et al., 2018). Siegler et al. (2018) measured the decline in the rate of torque development as a surrogate for central motor output, finding that the decline in rate of torque development was attenuated in the $NaHCO_3$ condition. Unfortunately, due to the PPO measurement being taken at maximal voluntary cadence in the current study, rate of force development was not able to be measured as a surrogate of central drive. However, the study by Siegler and colleagues is promising and provides rationale for further analysis in whole-body modalities such as cycling.

In summary, several mechanisms were explored in an attempt to explain the fatigue seen during the study. However, none of the measures taken during the study provided any evidence for their role in fatigue. Instead, further research is required to examine intramuscular mechanisms such as increased acidosis, increased P_i, decreased ATP and increased ROS.

5.3 Anaerobic power reserve

The final aim of the present study was to assess whether the APR could help characterize both supramaximal exercise performance and the physiological changes underpinning anaerobic energy production. From a performance perspective the findings of the current study align with original researchers who have shown differences in performance decrement between sprinters with a larger ASR, and endurance athletes with a smaller ASR (Weyand & Bundle, 2005). In the current investigation a positive linear relationship was found between APR and the decrease

in PPO (Fig. 4.7A) showcasing a steeper decrement in performance amongst those with larger APR's. Additionally, a significantly greater decrease in PPO was identified in SPR participants (n = 6) vs. END (n = 6) during subgroup analysis (Section 4.5.1). Alternatively, limited research has sought to characterize the ASR/APR by the physiological changes that occur at such supramaximal intensities. Despite this lack of physiological context, some researchers have proceeded to endorse the percentage of ASR/APR as a useful tool for measuring relative exercise intensity (Buchheit & Laursen, 2013a; Buchheit & Laursen, 2013b; Sandford & Maunder, 2018). However, to date only one study has effectively explored the ASR and it's use as a relative exercise intensity measure, with changes in physiological variables i.e. blood lactate (Julio et al., 2019). Therefore, a novel finding of the current study was that the APR does not effectively characterize the physiological changes commonly associated with high rates of anaerobic ATP production. Indeed, there was a negative correlation between APR and the absolute increase in blood [La^-] (r = -0.59, P = 0.045, Fig. 4.7C), as well as a lack of significant linear relationships between APR and either the absolute changes in pH (P = 0.08) or SpO_2 (P = 0.098) from PPO1 to PPO2 (Section 4.6). This evidence disputing the APR's characterization of anaerobic glycolytic ATP production instead implies, by elimination, that the ATP-PCr required by the initial sprint is a major determinate of the APR (Bogdanis et al., 1998; Hirvonen, Rehunen, Rusko & Härkönen, 1987). Subgroup differences in PPO also support this finding with significant differences in PPO only seen at PPO1 (Fig. 4.1A), highlighting the advanced sprinting capacity of the SPR participants. Alternatively, the lack of a significant difference in PPO at PPO2 showed that the larger APR of SPR participants was not a defining factor in the ability to sprint later into the protocol with high rates of anaerobic glycolysis was required.

5.3.1 Relationship between anaerobic power reserve and the decrease in peak power output

The percentage decrease of PPO was positively correlated with APR (Fig. 4.7A), showing that a higher APR is associated with a greater fatigue, represented by a greater percentage decrease of PPO. Additionally, a significantly greater decrease in PPO was identified in SPR participants during subgroup analysis (Section 4.5.1). These results align with previous research showing that sprint athletes, with larger ASRs, exhibit greater exponential declines in absolute performance compared to endurance athletes, with smaller ASRs, over supramaximal exercise bouts of 3-300 s (Weyand & Bundle, 2005). Researchers have attempted to explain this increased exponential decline in performance by attributing it to the higher percentages of the APR/ASR (as a measure of exercise intensity) and assuming that this represents an increased rate of anaerobic metabolism (Bundle, Hoyt & Weyand, 2003; Sundberg, Hunter & Bundle, 2016; Weyand & Bundle, 2005; Weyand, Lin & Bundle, 2006). However, the results of the current investigation question the proposed metabolic basis proposed to explain the exponential decrement in fatigue (Blondel et al., 2001; Bundle, Hoyt & Weyand, 2003; Weyand & Bundle, 2005; Weyand, Lin & Bundle, 2006). In the current study differences in the percentage decrease of PPO seen between SPR and END came as a result of a higher PPO at PPO1, with no difference in PPO at PPO2 (Fig. 4.1A and Section 4.5.1). Such evidence could suggest that the steeper drop in PPO (representing greater fatigue) seen in SPR, with higher APR's (Table 4.1), relies more on the superior ability of those with larger APR to sprint while unfatigued. This counters previous hypothesis that such fatigue is due to the accumulated effects of metabolites throughout supramaximal exercise. However, another view suggests that the smaller percentage decrease of PPO seen in END participants, who possess significantly lower APR's (51.6% SPR vs. 37.9% END, $P = 0.01$) (Table 4.1), provides evidence that they are instead able to resist higher metabolic disturbance while still producing similar final PPO's

(PPO2) to SPR participants (Fig. 4.1A). This ability to withstand metabolic disturbance is evidenced by the significantly lower plasma pH and higher blood [La^-] measured in END at PPO2 (Table 4.3). However, given the only performance difference between subgroups came at PPO1 it is instead proposed that the APR is defined more so by the initial ability to produce PPO, and therefore the capacity of ATP/PCr system, than the fatiguing effects of accumulated metabolites on the ability to sprint later into supramaximal exercise (PPO2) (Bogdanis et al., 1998; Hirvonen et al., 1987).

5.3.2 Application of the anaerobic power reserve to the characterisation of anaerobic energy production and alterations in plasma acid-base balance

Despite being used to imply anaerobic energy contribution the physiological underpinnings of the APR are still yet to be truly identified. A key finding questioning the validity of the model's measurement of anaerobic glycolysis is the moderate negative correlation that was found between APR and the change in blood [La^-] from PPO1 to PPO2 (Fig. 4.7B). Lactate is used as a common measure of anaerobic metabolism, accounting for ~90% of all metabolites generated via the anaerobic catabolism of carbohydrate (Bangsbo et al. 1990; Green & Dawson, 1993). Given that the APR has been promoted as an alternative to other measures of anaerobic capacity and contribution, a positive relationship was expected if an increased APR correspond with a larger anaerobic capacity (Blondel et al., 2001). Previous research has shown mixed findings regarding the relationship between ASR/APR and lactate. With some in agreement with the findings of the present investigation (Buchheit, Hader, & Mendez-Villanueva, 2012), while others have found a positive relationship between blood lactate accumulation and ASR/APR (Dardouri et al., 2014; Panissa et al., 2016). Acknowledging the flaws concerning net blood [La^-] as a measure of anaerobic energy production, the negative relationship between the APR and the change in blood [La^-] from

PPO1 to PPO2 still questions the validity of the model to accurately account for the prominent source of anaerobic energy production.

Other findings further question the validity of the model's use in characterising anaerobic capacity and contribution. Firstly, a lack of linear relationship was found between APR and the resultant changes in plasma acid-base balance commonly seen in highly anaerobic exercise. Examples in the current investigation of such changes include plasma pH and SpO_2 (Section 4.6). Although seen to be approaching significance correlations between APR and the absolute decrease from PPO1 to PPO2 in either pH and SpO_2 failed to do so. Additionally, although significant metabolic differences were observed between subgroups (i.e. higher plasma pH seen in SPR at PPO2 and the higher blood [La^-] in END at PPO2 (Table 4.3)) no significant differences in PPO were observed between subgroups at PPO2 (Section 4.5). Instead, as highlighted previously, the performance difference between subgroups was seen at PPO1 due to the superior sprint capabilities of SPR participants (Fig. 4.1A). Although these findings provide evidence questioning the validity of the APR to quantify the glycolytic component of the anaerobic pathway an opposing explanation could exist for lack of associations seen in the present investigation.

5.3.3 An alternate view – Suggestion for the use of percentage of anaerobic power reserve as a measure of relative exercise intensity

Evidence questioning the validity of the APR as a measure of anaerobic glycolytic contribution may instead be explained by differences in the relative workloads assigned during the study. Although the 3-min fixed-intensity time-trial was conducted at the same relative intensity as a percentage of $p\dot{V}O_2peak$, the END participants were not only required to complete significantly higher absolute workloads, due to their higher $p\dot{V}O_2peak$, but also a relatively higher

percentage of their APR. More specifically, END participants were forced to complete the 3-min fixed-intensity time-trial at 6% of their APR while SPR participants rode at 2% (Table 4.1). This difference may have placed END participants under greater anaerobic strain during the 3-min of supramaximal exercise, resulting in lowered plasma pH and higher blood [La^-] at PPO2 (Table 4.3). To the researcher's knowledge, only one study to date has made a comparison between the percentage of APR/ASR and other relative prescriptive protocols i.e. percentage of $v\dot{V}O_2peak$. In this case greater variability in blood lactate and performance time-to-exhaustion was present between subgroups of rugby players and long-distance runners when exercise intensity was based off percentages of $v\dot{V}O_2peak$ (Julio et al., 2019). However, this physiological difference in response to exercise was eliminated when exercise was prescribed as a percentage of ASR. Although the study by Julio et al. (2019) differs from the current investigation, which used one prescriptive tool (105% of $p\dot{V}O_2peak$) to assess relationships between the varying APR and either performance or physiological variables, their findings could explain the differences seen in plasma pH and blood lactate seen between subgroups at PPO2. Despite the recent findings of Julio et al. (2019) supporting the alternative hypothesis that subgroup differences could be explained by the measure of relative exercise intensity used, prescription of exercise intensity as a percentage of APR remains a debated theory (Boullosa & Abreu, 2014; Buchheit & Laursen, 2013a; Buchheit & Laursen, 2013b; Sandford & Maunder, 2018). Therefore, results of the current study may suggest that some value could be placed on the use of the APR as a measure of relative exercise intensity. However, this is only suggestive. Instead, further research is required to continue to explore the role of the percentage of APR as a measure of relative exercise intensity, particularly through examination of physiological changes (similar to those measured in this study) across a wider range of intensities.

In summary, although it was found that greater decreases in PPO were correlated with larger APR the inverse correlation with changes in blood [La^-] and the non-significant relationship with changes in plasma pH suggest that the APR does not accurately account for the contribution of anaerobic glycolysis (Bangsbo et al. 1990; Green & Dawson, 1993). Instead, because the APR/ASR utilizes PPO or maximal velocity as its upper capacity measure it appears that the APR is only suggestive of the capabilities of a well-developed ATP/PCr system (Bogdanis et al., 1998; Hirvonen et al., 1987). Taken together these question the metabolic basis of fatigue put forward by early researchers of the APR/ASR. It also questions the use of the ASR/APR to accurately represent the anaerobic ATP contribution to supramaximal exercise.

Chapter 6 – Conclusion, limitations and areas for future research

6.1 Conclusion

This study provides strong evidence that an exercise-induced reduction in plasma acidosis does not affect the decrease in PPO after supramaximal cycling. No differences were seen in PPO between intervention trials (PLA vs. BIC) despite significant differences in plasma pH and [HCO_3^-], measured after PPO2. Further supporting this finding, no significant linear correlation was found between the decrease in plasma pH and the decrease in PPO. Secondly, it was shown that increased plasma [K^+], plasma [Na^+] and blood [La^-], a decreased plasma [Ca^{2+}], and reductions in SpO_2 were also not independently associated with the reduction in PPO. This suggests that although the current investigation was not able to conclusively elucidate the primary cause of fatigue, represented by a decrease in PPO, it did however allow for the several variables to be eliminated as causes in the current model of exercise. Taken together, these results provide novel findings concerning plasma acidosis' role in limiting the ability to sprint over the concluding stages of a supramaximal event, which is similar in intensity and duration to several other events that may also benefit from these findings. Finally, the present investigation also questions the ability of the APR to represent the anaerobic capabilities above $\dot{V}O_2$peak. Evidence of a negative relationship between changes in blood [La^-] suggest that a higher APR does not imply an increased contribution of the anaerobic glycolytic pathway. Furthermore, a non-significant relationship with plasma pH shows an inability of the APR to accurately predict the changes in acid-base balance that occur over supramaximal exercise. Therefore due to the use of PPO as a capacity measure it appears that the ATP-PCr pathway is a major determinate of the APR as opposed to the contribution of the anaerobic glycolytic pathway.

6.2 Limitations

6.2.1 Sodium bicarbonate loading strategy

Although researchers have recently called for the use of individualized time to peak [HCO_3^-] protocols when using $NaHCO_3$ supplementation it was decided not to use such a strategy in the present study (McNaughton et al., 2016; Siegler et al., 2016). Several constraints around data collection were present restricting the utilization of an individualized strategy. It has been shown that such a protocol can add multiple sessions per participant to the study design (Deb et al., 2018; Gough et al., 2018; Miller et al., 2016). Given the time requirements of each testing session and the intensity of the exercise protocol, it was decided that a standardized dosing protocol would be more appropriate to improve recruitment and execution of data collection. Also, research using individualized strategies is currently in its infancy. Although the few studies using such a strategy have led to more ergogenic results across group data, closer examination of individual responses has shown that inconsistency of erogenicity as well as plasma [HCO_3^-] and pH response is still present (Deb et al., 2018; Miller et al., 2016). Clearly further research is required to elucidate whether a certain strategy is superior across both performance and physiological outcomes.

A second consideration regarding the dosing strategy was validation of its efficacy through pre-exercise measurement following ingestion. Traditionally, a plasma sample is taken prior to exercise, to avoid the confounding effects of exercise, and gain the best representation of changes to plasma pH and [HCO_3^-] as a result of supplementation. This did not occur in the current investigation for two reasons. Firstly, the aim of the study was to compare differences that occurred between the two PPO (PPO1 and PPO2) in the intervention trial as a result of the fixed-intensity time-trial. Secondly, the cost of testing cartridges meant that the addition of pre-exercise measures would fall outside of the available budget. However, through pilot

testing it was determined that adequate changes to plasma [HCO_3^-] and pH could be achieved even if measurements were taken post-exercise at PPO1. To do this plasma changes between BIC and PLA would be compared at PPO1 instead of rest to standardize the effect of exercise. Yet, because of the interference of exercise it can only be assumed that the desired 6 $mmol.L^{-1}$ change in plasma [HCO_3^-] outlined by Carr et al. (2011) was achieved prior to exercise.

A third consideration was the absence of an equimolar, taste matched placebo and its effect on the blinding of participants. Given the inability to completely blind the majority of participants (Section 4.4), the use of a non-flavour matched placebo is a limitation. Although it allowed for the speculation of the proposed expectancy hypothesis for $NaHCO_3$ ergogenic effect, it did limit the double-blinded study design. Similar issues concerning blinding to those found in this investigation have been noted previously when cornflour has been used (Carr, Gore & Dawson, 2011). However, it has also been successfully used on other occasions when assessing $NaHCO_3$ during supramaximal exercise performance (Kupcis et al., 2012; Kilding, Overton & Gleave, 2012).

6.2.2 Plasma measures

Several limitations must also be acknowledged in the selection of blood draw sites and the timing between when measures were collected. Due to the rapid recovery of plasma electrolytes, some urgency was placed on the collection of blood post-exercise (Vøllestad, Hallen & Sejersted, 1994). It was decided that a venepuncture from the arm was the most practical given the timing requirement for collection and also considering the comfort of the participant. However, this is a limitation of the study due to the proximity of the blood draw site to the working muscle. With plasma concentrations post-exercise shown to be vastly

different when compared to samples from the working muscle (i.e. femoral artery or vein vs. interstitial), the data gathered in the current investigation must be interpreted with this context in mind (Green, Langberg, Skovgaard, Bülow & Kjær, 2000; Sjogaard, Adams & Saltin, 1985; Vøllestad, Hallen & Sejersted, 1994). Although the results of this study have been shown to be similar to other studies, it still must be assumed that the data collected underestimates [K^+] nearer to the working muscle (Vøllestad, Hallen & Sejersted, 1994). Additionally, with the continued accumulation of blood lactate within the first few minutes post-exercise, lactate levels may have changed with time (Freund et al., 1986). Additional considerations concerning lactate also include the draw site away from working muscle and the significant differences between plasma concentrations to that seen in working muscle (Freund et al., 1986; Sahlin, Harris, Nylind & Hultman, 1976).

6.2.3 Intramuscular measures

Several notable measures were not present in the current study meaning that definitive conclusions regarding intracellular measures (particularly acidosis) and acid-base balance could not be reached. Although changes in plasma acidosis were the focus of the present investigation due to the perceived benefit of $NaHCO_3$ and the consistent negative effects of NH_4Cl, the lack of measurement within the muscle limits the ability to rule out intramuscular acidosis as a fatigue mechanism. Many of the past fatigue proposals concerning acidosis are associated to intramuscular changes as opposed to plasma changes (i.e. sarcoplasmic reticulum changes in Ca^{2+} sensitivity and release, effects of acidosis on enzymes and myofilaments, etc.) (Hollidge-Horvat et al., 1999; Lännergren & Westerblad, 1991; Pate et al., 1995; Westerblad & Allen, 1991). The lack of intramuscular measures also impacted interpretation of several other proposed fatigue measures, such as intramuscular ATP, PCr, P_i, Ca^{2+} and ROS. Therefore,

although several plasma variables were able to be directly discussed in addition to plasma acidosis, other mechanisms were only able to be postulated upon. A final plasma measure that was notably missing from the present study is plasma [Cl^-]. Inclusion of plasma [Cl^-] would have allowed for the calculation of strong ion difference and therefore better speculation of changes to plasma acid-base balance (Gough, 2018; Lindinger, Kowalchuk & Heigenhauser, 2005; Stewart, 1981; Van Slyke & Cullen, 1917). Additionally, intramuscular measurements of components of the strong ion difference will have allowed for better understanding of the effects intramuscularly, but also how the interaction between intra and extracellular changes impact fatigue (Gough, 2018; Lückher et al., 2017).

6.2.4 Relative exercise intensity

Although the percentage of $\dot{V}O_2$peak better reflected the intensities required in a 4000-m individual pursuit the subgroup results may have questioned whether this was an appropriate measure of exercise intensity (Table 4.1). Instead some have suggested percentage of APR/ASR as a more appropriate measure of supramaximal intensity, better accounting for differences in anaerobic capacity (Buchheit & Laursen, 2013a; Buchheit & Laursen, 2013b; Sandford & Maunder, 2018). This belief has arisen due to research showing that performance in supramaximal efforts (120% and 140% of velocity at $\dot{V}O_2$peak) correlated more highly with anaerobic capabilities above aerobic capacity than v$\dot{V}O_2$peak (Blondel et al., 2001). However, research is particularly limited concerning the use of the percentage of APR/ASR as a measure of relative exercise intensity. Currently one published study has compared the use of the percentage of ASR to the more commonly used percentage of v$\dot{V}O_2$peak. Combining this lack of research using the percentage of APR/ASR with the established research defining the

intensity of a 4000-m individual pursuit by percentage of p$\dot{V}O_2$peak there was more rationale for the use of percentage of p$\dot{V}O_2$peak in this study. However, it was noted that subgroups did ride at significantly different percentages of their APR (Table 2) and absolute workloads during the fixed-intensity time-trial in the current study. From this it could be hypothesised that the significant differences seen in pH at PPO2 could be attributed to the absolute difference in exercise workload and relative percentage of APR. This is supported by recent research showing that supramaximal exercise prescribed as a percentage of v$\dot{V}O_2$peak showed more performance and physiological variability compared to exercise prescribed as a percentage of ASR (Julio et al., 2019). Therefore, the significant difference between subgroups highlights a potential limitation in the relative exercise intensity tool used. Calling for further research to be conducted on quantifying the physiological load when using varying percentages of APR.

6.3 Future research

Due to the success of the exercise protocol used in this study it is proposed that further research conducted using this protocol should focus on other potential mechanisms of fatigue. These include not only plasma measures, potentially taken closer to the working muscle, but also various intramuscular measures. In addition to this plasma acidosis could also be re-examined using an individualized time to peak plasma [HCO_3^-] strategy or with the use of a taste matched equimolar placebo such as NaCl. It may also be valuable to gather intramuscular data relating to transmembrane transportation via MCT proteins. Given that research has correlated the ergogenic effects of endogenously induced alkalosis to the content of these proteins within the muscle, this could be valuable information to help not only elucidate the role of pH regulation via transport mechanisms in fatigue, but also help clarify some of the responder/non-responder debate concerning $NaHCO_3$ as an ergogenic aid (Messonnier et al., 2007).

Additionally, this is the first study to examine the APR in the context of changes to plasma acid-base balance over a sustained supramaximal bout. Considering the results and limitations of the present study several areas for future study arise. Firstly, furthering the current investigation by relating intramuscular mechanisms to the APR. Secondly, assess changes in muscle activation via electromyography (as a surrogate of central drive) to metabolic measures (i.e. ATP, PCr, P_i and acidosis). Given that muscle activation has been the primary physiological measure used in the study of the exponential decrements seen in the intensity/duration relationship of supramaximal exercise within ones APR, studying this in the context of changes to metabolic factors via interventions such as $NaHCO_3$ or NH_4Cl supplementation or the use of lumbar intrathecal fentanyl to manipulate group III and IV muscle afferents, could help associate instead of speculate the influence of a metabolic variable in the exponential relationship identified in research.

Finally, research should continue to assess the credibility of the percentage of APR as a measure of relative exercise intensity. In order to do this research can continue to map the physiological changes over supramaximal exercise bouts across a range of intensities relative to different percentages of the APR. This is critical before widespread use of the measure to prescribe intensities can be confidently advocated.

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Appendices

Appendix A: Ethics Approval

A.1. Ethics Approval

AUTEC Secretariat

Auckland University of Technology
D-88, WU406 Level 4 WU Building City Campus
T: +64 9 921 9999 ext. 8316
E: ethics@aut.ac.nz
www.aut.ac.nz/researchethics

1 December 2017

Simeon Cairns
Faculty of Health and Environmental Sciences

Dear Simeon

Re Ethics Application: **17/386 Examination of the effect of exercise induced plasma acidosis on peak power after a simulated 4000 m individual pursuit on a bicycle ergometer**

Thank you for providing evidence as requested, which satisfies the points raised by the Auckland University of Technology Ethics Committee (AUTEC).

Your ethics application has been approved for three years until 1 December 2020.

Standard Conditions of Approval

1. A progress report is due annually on the anniversary of the approval date, using form EA2, which is available online through http://www.aut.ac.nz/researchethics.
2. A final report is due at the expiration of the approval period, or, upon completion of project, using form EA3, which is available online through http://www.aut.ac.nz/researchethics.
3. Any amendments to the project must be approved by AUTEC prior to being implemented. Amendments can be requested using the EA2 form: http://www.aut.ac.nz/researchethics.
4. Any serious or unexpected adverse events must be reported to AUTEC Secretariat as a matter of priority.
5. Any unforeseen events that might affect continued ethical acceptability of the project should also be reported to the AUTEC Secretariat as a matter of priority.

Please quote the application number and title on all future correspondence related to this project.

AUTEC grants ethical approval only. If you require management approval for access for your research from another institution or organisation then you are responsible for obtaining it. You are reminded that it is your responsibility to ensure that the spelling and grammar of documents being provided to participants or external organisations is of a high standard.

For any enquiries, please contact ethics@aut.ac.nz

Yours sincerely,

Kate O'Connor
Executive Manager
Auckland University of Technology Ethics Committee

Cc: mathew.mildenhall@gmail.com; plews@plewsandprof.com

Appendix B: Tools

B.1 Participant Information Sheet

AUT
TE WĀNANGA ARONUI O TĀMAKI MAKAU RAU

Participant Information Sheet

Date Information Sheet Produced:

12 April 2018

Project Title

The effect of exercise induced acidosis on peak power after simulated 4000 m Individual Pursuit on a bicycle ergometer.

An Invitation to part take in this study

Firstly, a massive thank you for considering this study. My name is Mathew Mildenhall and I am a master's student, researcher, and endurance enthusiast, currently studying at AUT. I am wanting to dive deeper into the current beliefs around what causes fatigue, especially the acidosis that occurs during longer high intensity cycling (i.e. 4000 m Individual Pursuit). With your help as a potential participant this research will enhance our understanding of how the body works during intense cycling and will also provide information that could help substantiate a newly proposed performance prediction tool, the Anaerobic Power Reserve (APR) model. Please feel free to read on for further details about the project and the requirements of participants.

What is the purpose of this research?

The purpose of this study is to improve scientific understanding in two major areas, which are likely to benefit athletes, researchers and the wider cycling community. Firstly, it is a common belief that acidosis (specifically lactic acid) causes muscle fatigue which negatively impacts cycling performance. However, new research has questioned this belief. Given this and New Zealand's rich history in track cycling, our research team believe it would help to better understand the role acidosis plays in determining performance. Therefore, we will firstly study processes linked with blood acidosis (and lactate), relating them back to a key determinant of cycling performance, peak power output. Secondly, the study will strengthen understanding of a newly proposed model for performance prediction, the APR model. The APR model, offers a simplistic way to predict performance over durations of 5-350 s. However, this model is currently not linked to any of the body processes that could limit performance (e.g. acidosis). By analysing relationships between an athlete's APR and blood acidosis levels during cycling we will attempt to expand the model's application power as a non-invasive performance/training tool. The findings could significantly advance understanding for both the academic and athletic communities.

How was I identified and why am I being invited to participate in this research?

You have been identified as a potential participant through your Cycling New Zealand affiliated club or through the researcher's personal network. After initially reaching out to your club, whose contact details were listed on the Cycling New Zealand website, we were put in contact with the coaching staff who allowed us to attend one of your training sessions.

Inclusion and exclusion criteria

Each prospective participant's eligibility is dependent on their age (18-40 yr), competitive cycling background, and health status. We require participants to have trained consistently over the past 12 months with no injuries during the past 6 months. If you have experienced some form of cycling related injury in this time you will have to be excluded from the study as a precaution to help avoid re-injury. Additionally, if you fail to meet the health standards required to participate, specifically concerning renal conditions which may put you at risk of adverse reaction to the sodium bicarbonate supplement used during testing (purpose outlined below), you will also be discouraged from participating in the study. This will be assessed via a medical questionnaire completed at the beginning of the initial testing and familiarization session, which includes questions concerning medical history and medication use prior to inclusion in the study. Finally, in an effort to target the APR, you are also required to reach powers over and above those achieved during peak oxygen consumption (VO_2peak) testing, otherwise you will be excluded from the remainder of the testing.

How do I agree to participate in this research?

Your participation in this research is voluntary (i.e. it is your choice) and whether or not you choose to participate will neither advantage nor disadvantage you. You are able to withdraw from the study at any time. If you choose to withdraw, then you will be offered the choice of having any data, which is associated to your specific participation, removed. However, once all findings have been obtained removal of your data may not be possible.

What will happen in this research?

The study will occur at the SPRINZ laboratory based at AUT Millennium, 17 Antares Pl, Rosedale, Auckland. It will be conducted over three separate sessions using a bicycle ergometer for each session. These testing sessions will be separated by at least 48 hr and each session is anticipated to require about 2-3 hr of your time. You should avoid strenuous exercise for the 48 hr leading up to testing, along with avoiding the consumption of caffeine/ alcohol/ergogenic aids within 24 hr of testing. You are asked to arrive at testing in a rested and well hydrated state and having not eaten (fasted) for at least 2 hr prior to arrival. Finally, in an effort to ensure that your nutrition status is consistent across testing sessions you will be required to keep a food and beverage diary the day of initial testing and asked to repeat this eating/drinking leading into each intervention session.

Outline of each session

A standardized warm-up will be used prior to each testing session. This protocol consists of 10 min at 100 W followed by 3 high intensity 6 s sprints separated by 1 min of cycling at 100 W. These sprints are to be conducted at 80%, 90% and 100% of perceived maximal effort, after which a 5 min stand down period will occur prior to the start of testing. An outline of each test session is listed below:

- ***Initial testing and familiarization*** - This first session will establish your APR and familiarize you (or practice) with the requirements of the two intervention sessions to follow. Establishing your APR will require you to complete two bouts of exercise to achieve different power levels. Firstly, an all-out 6 s sprint will be used to determine your peak power output, followed by a 10 min period to allow for your recovery before commencing the next bout. Secondly, an incremental exercise test will be used to establish your power at VO_2peak (pVO_2peak). The difference between these two power levels will determine your APR. After a further 20 min rest period you will complete a short familiarization trial to prepare you for the intensity required during the following two intervention trials. During this familiarization you will cycle for ~2 min at 105% of pVO_2peak.
- ***Intervention sessions*** - There will be two separate intervention sessions. When you arrive (2 hr before the exercise trial) resting blood measures will be obtained from a finger prick and venous sample. You will then be randomly assigned into either a placebo group (using corn flour in gelatine capsules) or a treatment group (using sodium bicarbonate in gelatine capsules), which you will be given 30 min to swallow with water. After the supplement has been consumed you will rest for 70 mins before the warm up will commence. Once warmed up you will undergo a fresh peak power output assessment which will be followed directly by a second blood draw and a 10 min rest period. Following this rest, you will move on to a cycling time trial. This time trial involves two parts: (i) 3 min at a fixed power output of 105% pVO_2peak (based on the results of the earlier test). (ii) at the 3 min mark participants will then be instructed to conduct a final all-out peak power output assessment. Blood measures will then be taken again to capture post cycling trial measures.

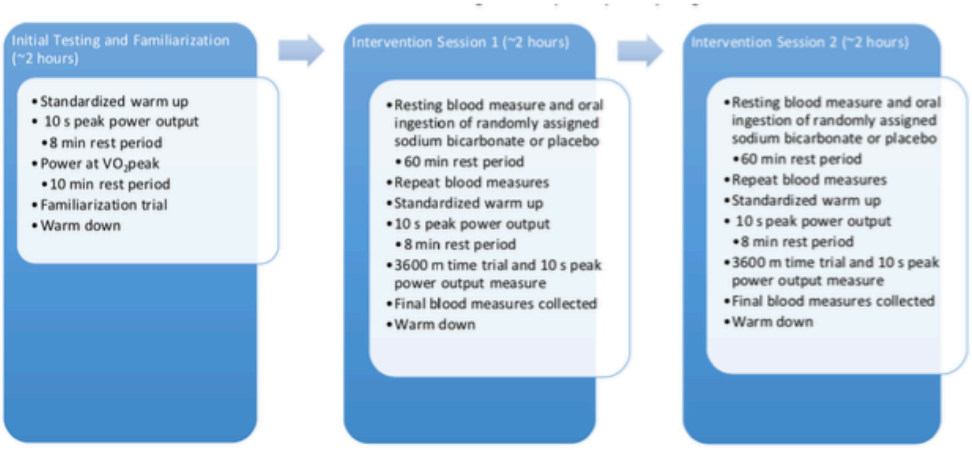

Figure 1: Flow chart of the three required sessions for the study

What are the discomforts and risks?

During each exercise bout you may experience some discomfort and fatigue since it involves strenuous exercise (i.e. peak power test, VO_2peak test, familiarization and exercise trial). However, this discomfort will be similar to what you would normally experience during intensive training and racing. Also, sodium bicarbonate can produce minor gastric discomfort in some participants. The research team will monitor your level of gastric comfort throughout the rest period and testing to ensure you're comfortable throughout the testing process. Finally, the study will require some blood to be drawn on several occasions across the two intervention sessions. This blood will be drawn via two means, small finger prick and venous/vein blood. There is a small risk and mild soreness associated with blood collection. Additionally, if you suffer from renal conditions it is stressed that you make the research team aware of this. This is done in an effort to help avoid potential adverse reactions to the sodium bicarbonate supplementation if you possess such conditions. Only qualified, experienced phlebotomists will draw venous blood throughout the study. Adherence to correct techniques will ensure minimal risk and discomfort.

What are the benefits?

We believe that this research provides enormous benefit to various stakeholders. Firstly, for the athlete/participant, coaches and practitioners this research will provide a better understanding of how changes that occur in the body (especially relating to acidosis) during a simulated 4000 m individual pursuit time trial, and how these influence peak power output. By capturing various measures using a blood draw we will be able to track this response at a point in a 4000 m individual pursuit where one would prepare for the final surge to the line. In addition to this you as the participant will understand whether you respond positively to sodium bicarbonate. Believed to reduce the exercise-induced blood acidosis it has become a well-regarded supplement, to aid performance at higher intensities. Finally concerning the APR, from the athlete/participants perspective, by establishing your APR we will be able to help you understand the energy reserve above VO_2peak that you have access to. This prediction tool has been used in research studies to predict performances within 6.6% for efforts lasting from 5-350 s. Furthering understanding of this APR could be of benefit for athletes and coaches by helping to better assess the performance fitness of the athlete without having to produce multiple race performances across various durations. It will also aid in deciding what event the athletes are best suited for or whether their current fitness level aligns with that of previous results.

From the perspective of the research team, this study offers an opportunity to explore blood acidosis in great depth and how it effects peak power output over longer duration events (i.e. 60 s – 8 min), currently has limited scientific understanding behind it. Furthermore, we will be able to associate blood acidosis levels with the APR model. This will be a novel endeavour as currently no research has related blood acidosis with the APR model. Based on two power data points (power at VO_2peak and peak power output) the model benefits from its simplicity for application, however this simplicity also currently limits it's understanding. Therefore, creating relationships between the APR and acidosis could have progress this new model another step forward. Finally, this project provides the primary researcher, Mathew Mildenhall with a basis for his master's thesis project.

What compensation is available for injury or negligence?

In the unlikely event of a physical injury as a result of your participation in this study, rehabilitation and compensation for injury by accident may be available from the Accident Compensation Corporation, providing the incident details satisfy the requirements of the law and the Corporation's regulations.

How will my privacy be protected?

Your privacy as a participant will be maintained at all times. Once your consent form and personal descriptive information has been gathered, we (the research team) will assign you a unique participant code which will mean that we no longer need to have your personal data on hand. This will allow us to safely store your data, and the results gained from this research, in a secure facility within SPRINZ at AUT Millennium. In addition, there will be no identifiable data used in any research publications or distributed to any third party.

Blood samples will be stored in accordance to best practices outlined in the *Code of Health and Disability Services Consumers' Rights*. Each blood sample will be assigned its participants unique participant code during storage. You will also be given the opportunity to have your blood samples returned at the conclusion of the study. This will be identified in the written consent form completed prior to this study. However, you are able to change your selection any time before the completion of the study. If you choose to have your samples returned this will be done in accordance with right 7 (9) of the *Code of Health and Disability Services Consumers' Rights*. Alternatively, if you select to not have your blood samples returned they will be de-identified and disposed of in accordance to SPRINZ laboratory protocols.

What are the costs of participating in this research?

Participation is at no financial expense to you. The only requirements will be your time, which is about 2 hr per session for three sessions. Therefore, the total time required will be ~6 hr (over about one week given the 48 hr periods required between sessions). This study will require high intensity cycling and there is a requirement to refrain from intense exercise within the 48 hr prior to testing. With this being said, in return for your commitment to the study you will be granted the benefits outlined above. These include determining your APR which can be applied to help predict performances over durations of 5-350 s and assess current fitness. The findings from the research will also allow you to gain a better understanding of your body responses to high intensity work in conditions that are similar to a simulated 4000 m Individual Pursuit. Finally, findings will also help you establish whether your dose response to the ergogenic aid, sodium bicarbonate, is effective in buffering against acidosis.

What opportunity do I have to consider this invitation?

You will have up to two weeks to decide on your participation. In order to participate written consent will be required within this time frame. This will be evidenced by the competition of a written consent form which is included below. This will be required to be returned to the primary researcher in person prior to testing.

Will I receive feedback on the results of this research?

At the completion of the study, a summary of the research can be provided via email to you as the participant. This summary will include your personal results and the pooled averages for all participants. No data will be given to participants coaches or others, unless specifically requested by the participant.

What do I do if I have concerns about this research?

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor, Associate Professor Simeon Cairns:

- *Email:* simeon.cairns@aut.ac.nz
- *Work phone:* + 64 9 921 9999 ext. 7125.

Concerns regarding the conduct of the research should be notified to the Executive Secretary of AUTEC, Kate O'Connor:

- *Email:* ethics@aut.ac.nz
- *Work phone:* + 64 9 921 9999 ext. 6038.

Whom do I contact for further information about this research?

Please keep this information sheet and a copy of the consent form for your future reference. You are also able to contact the research team as follows:

Researcher Contact Details: Mathew Mildenhall can be contacted at:

- *Email:* mathew.mildenhall@gmail.com
- *Mobile phone:* 0273522550

Project Supervisor Contact Details: Simeon Cairns can be contacted via email or work phone at:

- *Email:* simeon.cairns@aut.ac.nz
- *Work phone:* + 64 9 921 9999 ext. 7125

Approved by the Auckland University of Technology Ethics Committee on *type the date final ethics approval was granted*, AUTEC Reference number *type the reference number*.

B.2 Consent form

Consent Form

Project title: The effect of acidosis on peak power after a simulated 4000 m Individual Pursuit on a bicycle ergometer

Project Supervisors: Assoc. Prof. Simeon Cairns & Dr. Daniel Plews

Researcher: Mathew Mildenhall, Masters student

Date form produced: 12 April 2018

Please tick the following:

- ○ I have read and understood the information provided about this research project in the Information Sheet.
- ○ I agree to take part in this research.
- ○ I agree to refrain from strenuous exercise within 48 hr of each of the three sessions required for the study.
- ○ I agree to avoid the consumption of caffeine, alcohol and any other ergogenic substances within 24 hr of each of the three sessions required for the study.
- ○ I agree to fasting for the two hours prior to each of the three sessions required for the study.
- ○ I agree to provide blood samples.
- ○ I agree to orally ingest one of the two supplements (sodium bicarbonate or cornflour) allocated randomly during the two intervention trials.
- ○ I have had an opportunity to ask questions and to have them answered.
- ○ I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time without being disadvantaged in any way.
- ○ I understand that if I withdraw from the study then I will be offered the choice between having any data or tissue that is identifiable as belonging to me removed or allowing it to continue to be used. However, once the findings have been produced, removal of my data may not be possible.
- ○ I am not suffering from heart disease, high blood pressure, any respiratory condition (mild asthma excluded), renal condition, any illness or injury that impairs my physical performance.
- ○ I agree to my de-identified performance data being kept indefinitely in storage for future analysis/research purposes, and my personal details being stored in a locked cabinet for a period of 6 yr before being destroyed.
- ○ I wish to receive a summary of the research findings (please tick one): Yes○ No○
- ○ I wish to have my blood samples returned to me in accordance with right 7 (9) of the *Code of Health and Disability Services Consumers' Rights* (please tick one): Yes○ No○

(If you select *"No"* your blood will be disposed of in accordance to SPRINZ laboratory protocols).

Participant's signature: .....

Participant's name: .....

Participant's Contact Details (if appropriate):

.....

.....

.....

Date:

Approved by the Auckland University of Technology Ethics Committee on type the date on which the final approval was granted AUTEC Reference number type the AUTEC reference number

Note: The Participant should retain a copy of this form.

B.3 Participant recruitment advertisement

CYCLISTS WANTED!

WHAT IS THIS STUDY ABOUT?

- Looking at the effect of lactate acidosis, that occurs during racing, on peak power.
- This will be achieved by looking at differences in blood markers and peak power, before and after a simulated 4000 Individual Pursuit, using a sodium bicarbonate and placebo supplement.
- We want to understand if there is a link between these blood markers and a new performance predictor – the Anaerobic Power Reserve, and potentially highlight what makes some better at dealing with high intensity racing than others.

WHAT IS IN IT FOR YOU?

You will receive a customized performance report outlining your:

- ***VO_2peak*** - Establishing training zones and your peak oxygen carrying capacity
- ***Peak power output*** - One of the critical determinants of cycling performance
- ***Performance prediction*** - for durations from 5 s – 5 min.
- Overview of your **body's blood response to exercise**

INCLUSION CRETERIA:

- You are aged 18 to 40 yr, with a competitive cycling background.
- You have trained consistently over the past 12 months.

EXCLUSION CRITERIA:

- If you are unable to reach speeds above that at VO_2peak.
- If you have been injured in the past 6 months.
- If you do not meet the health standards required for such exercise e.g. some renal conditions.

IF THIS INTERESTS YOU, PLEASE CONTACT:

Primary researcher: Mathew Mildenhall
Email: mathew.mildenhall@gmail.com
Phone: 027 352 2550

Supervisor: Assoc. Prof. Simeon Cairns
Email: simeon.cairns@aut.ac.nz
Phone: 09 921 9999 ext. 7125

AUT

B.4 Health questionnaire

SPORTS PERFORMANCE
RESEARCH INSTITUTE, NEW ZEALAND
AN INSTITUTE OF AUT UNIVERSITY

AUT

Pre-test medical questionnaire

Surname: _____

First name/s: _____

Date of Birth: _____ Gender: Male ☐ Female ☐

Please answer the following questions by ticking the appropriate box, or filling in the blank.

1. **How would you describe your present level of activity?**

   Sedentary ☐ Moderately active ☐ Active ☐ Highly active ☐

2. **How would you describe you present level of fitness?**

   Unfit ☐ Moderately fit ☐ Trained ☐ Highly trained ☐

3. **How would you consider your present body weight?**

   Underweight ☐ Ideal ☐ Slightly over ☐ Very overweight ☐

4. **Smoking Habits:** Are you currently a smoker? Yes ☐ No ☐
   How many do you smoke .....per day

   Were you a previous smoker? Yes ☐ No ☐
   How long is it since you stopped? .....years
   Were you an occasional smoker? Yes ☐ No ☐
   Were you a regular smoker Yes ☐ No ☐
   How many did you smoke .....per day

5. **Do you drink alcohol?** Yes ☐ No ☐
   If you answered **Yes**, do you have?

   An occasional drink ☐ A drink every day ☐ More than one drink a day ☐

6. **Have you had to consult your doctor within the last six months?** Yes ☐ No ☐
   If you answered **Yes**, please give details.....
   .....
   .....

7. **Are you presently taking any form of medication?** Yes ☐ No ☐
   If you answered **Yes**, please give details.....
   .....
   .....

Pre-test medical questionnaire continued next page....

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8. **As far as you are aware, do you suffer or have you ever suffered from:**

a) Diabetes?	Yes ☐ No ☐	**b)** Asthma?	Yes ☐ No ☐
c) Epilepsy?	Yes ☐ No ☐	**d)** Bronchitis?	Yes ☐ No ☐
e) *Any form of heart complaint?	Yes ☐ No ☐	**f)** Raynaud's Disease?	Yes ☐ No ☐
g) *Marfan's Syndrome?	Yes ☐ No ☐	**h)** *Aneurysm/embolism?	Yes ☐ No ☐
I) Aneamia	Yes ☐ No ☐		

9. ***Is there a history of heart disease in your family?** Yes ☐ No ☐

10. ***Do you currently have any form of muscle or joint injury?** Yes ☐ No ☐
If you answered **Yes**, please give details.....
.....
.....

11. **Have you had to suspend you normal training in the last two weeks?**
Yes ☐ No ☐
If the answer is **Yes** please give details.....
.....
.....

12. **Please read the following questions**:

a)	Are you suffering from any known serious infection?	Yes ☐ No ☐
b)	Have you had jaundice within the previous year?	Yes ☐ No ☐
c)	Have you ever had any form of hepatitis?	Yes ☐ No ☐
d)	Are you HIV antibody positive	Yes ☐ No ☐
e)	Have you ever been involved in intravenous drug use?	Yes ☐ No ☐
f)	Are you haemophiliac?	Yes ☐ No ☐

13. **As far as you are aware, is there anything that might prevent you from successfully completing the tests that have been outlined to you?**
Yes ☐ No ☐

If the answer to any of the above is yes then:

a) Discuss with the Sports Performance Research Institute New Zealand (SPRINZ) clinician the nature of the problem.
b) Questions indicated by (*) Allow your Doctor to fill out the 'Doctors Consent Form' to be provided by the SPRINZ clinician.

Signed: _____ Date:_____

Signature of guardian / parent (if under 18) _____

Signature of tester: _____ Date:_____

B.5 Gastric discomfort scale

Gastric Distress Scale

Please rate your overall stomach distress over the last 10 minutes:

No Problem	**Minimal problem**	**Mild problem**	**Moderate problem**	**Moderately severe problem**	**Severe problem**	**Very severe**
I don't notice anything	*...but I can ignore it*	*Can be ignored but takes effort.*	*Ignorable but may influence my testing*	*Cannot be ignored and will influence my testing*	*Cannot be ignored and is concerning me*	*Worst stomach pain I've ever experienced!*
1	2	3	4	5	6	7

(Cite: Carr et al., 2011, Zanten, et al., 2006).

B.6 Food diary

Food Diary

Participant name:

INITIAL TESTING

Date:

Meal 1	Meal 2	Meal 3	Snacks	Other Meals	Beverages
Outline the food and beverages consumed over the 24hours prior to INITIAL TESTING					

INTERVENTION SESSION 1

Date:

Meal 1	Meal 2	Meal 3	Snacks	Other Meals	Beverages
Outline the food and beverages consumed over the 24hours prior to INTERVENTION SESSION 1					

INTERVENTION SESSION 2

Date:

Meal 1	Meal 2	Meal 3	Snacks	Other Meals	Beverages
Outline the food and beverages consumed over the 24hours prior to INTERVENTION SESSION 2					

B.7 Lode instruction manual – Explanation of the linear mode

Linear factor

The formula for linear ergometry is:

P = linear factor x RPM2

The factor has been made adjustable because then the dependency between the RPM and the load can be set to individual norms, which is a prerequisite for accurate measurements in for example a sprint test. As the RPM that is feasible for a person depends on personal characteristics like weight, size, biomechanical factors and technique it is useful to adjust the factor for each individual.
By executing a number of tests, the optimal linear factor can be assessed for a person.
The following example illustrates the effects of changing the Linear Factor:
We have two persons: A and B. The highest RPM that A can achieve is 80.
Because of personal characteristics B can achieve a RPM of 90. This difference in "top RPM„ influences the workload they will achieve in a RPM dependent test.
If both A and B perform a test at the standard linear factor of 0.04, the results will be as follows:

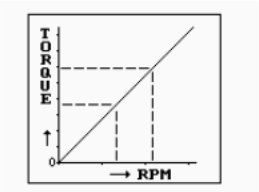

Fig. 4-18 Comparison Graph
A: 80 RPM x 80 RPM x 0.04 = 256 Watts
B: 90 RPM x 90 RPM x 0.04 = 324 Watts

However, there is a possibility that A is able to achieve a higher workload if the linear factor is changed. The linear factor has to be changed into:
324 : 80 RPM x 80 RPM = 0.051
By means of several tests a person(s highest RPM has to be measured before the linear factor can be adapted on a basis of personal characteristics like power and technique.

B.8 Communication with Lode – Calculation of peak power output

From: **Lode for Life | Understanding Movement & Performance**
Subject: RE: Lode question - Max Power
Date: 1 May 2018 at 8:49 PM
To:
Cc: Lode for Life | Understanding Movement & Performance

Dear Mathew

About your new questions:
Max power output - *From the output produced by the LEM software I am trying to determine the best measure of maximal power. However, I am a little confused as the peak power number given in the PDF output (attached) seems very high compared to elite cyclists (article attached). Because of this I have turned to the excel PFM output and see that there is an average power across one complex. I am assuming that a complex is a complete rotation of the crank. Would you suggest this as the most accurate measure of a peak max power?*

Somewhere during the forwarding the attachments have disappeared, can you please mail them to me? Unless you have enough on my answer:
The accuracy of the pedal force measurement during the total revolution is obtained by the placement of highly specific strain gauges in the crank axis making it possible to measure the pedal Torque for each pedal every 2 degrees during each revolution during the exercise test .

So the Torque is measured 180 times * 2 pedals in a single revolution. The power P is calculated 180 times per revolution by multiplying the sum of both pedal torques with the current rotational speed (in radians per second). The derived data 'average power' is the mean value of the 180 P values.

Kind regards,

B.9 Communication with Lode – Establishing appropriate torque factor

Torque factor

First I'll explain what our Wingate software is doing with the breaking Torque of the ergometer. Because Power is the product of Torque (Nm) and velocity, scoring in the Wingate test depends on the braking torque (in Nm) selected for each test. When the Torque is low, the athlete can pedal fast. If the torque is higher the pedalling rate becomes slower. For every person there is an optimal braking torque that yields the highest possible power. Ideally one would like to identify such an optimum for each subject. However to assess this, the test would have to be repeated a number of times against various braking torques. Like you can easily do with the LEM Wingate module *plus*. General guidelines on braking torques are Torque factors which you can multiply with the bodyweight to receive an estimated optimum for the breaking Torque. So the default Torque factor 0.7 times the bodyweight (0.7 Nm/kgBM) will give a Torque for an 'average' test subject. For top athletes the torque factor can be changed to receive a better fitting breaking Torque.

Normally an athlete will need a higher Torque factor between 0.8 and 1 for males and between 0.75-0.9 for females but it is totally dependent on the kind and level of sport activity.
When you do have the time to let them perform a multiple wingate test in advance, I would recommend that.
A commonly used multiple test is 4 trials of 5 seconds with recovery in between each trial of 2 minutes. First Torque factor 0.8, second one 0.75, third one 0.85 and last one 0.9. In the results you can easily see what has been the best Torque setting without exhausting the test subject too much. See below for the programming.