Enhancing athletic performance through high-intensity interval training and sodium bicarbonate supplementation

Matthew W. Driller
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A thesis submitted in fulfilment of the requirements for the degree of
Doctor of Philosophy

School of Human Life Sciences, University of Tasmania, Australia.

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Primary Supervisor: Dr. James Fell
Statement of Originality and Ethical Conduct

I, Matthew Driller certify that this work is entirely my own effort except where otherwise acknowledged. I also certify that, to the best of my knowledge and belief, the work is original and has not been previously submitted for any other award, nor does the thesis contain any material that infringes copyright. This thesis may be made available for loan and limited copying in accordance with the Copyright Act 1968.

The research associated with this thesis abides by the international and Australian codes on human and animal experimentation, the guidelines by the Australian Government's Office of the Gene Technology Regulator and the rulings of the Safety, Ethics and Institutional Biosafety Committees of the University.

Matthew Driller

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SUPERVISOR ENDORSEMENT

Date: 27/02/2012
Abstract

Introduction: Metabolic acidosis is a by-product of the energy production process required during high-intensity exercise, and it is thought to play a part in influencing muscle function and fatigue. Consequently, the efficacy of an athlete’s intra- and extracellular buffering systems may influence their performance during an exercise task. These buffering systems can be enhanced through exercise training and nutritional supplementation. Therefore, the purpose of this series of studies was to investigate combined training and sodium bicarbonate (NaHCO₃) supplementation techniques for enhancing performance in well-trained athletes.

Study 1. The aim of this study was to evaluate high-intensity interval training (HIT) for improving performance in already well-trained athletes. To achieve this we compared traditional rowing training (CT) to HIT in state-representative rowers. Following baseline testing (2000 m rowing test, incremental rowing test) 10 rowers were randomly allocated to HIT or CT, which they performed seven times over a 4-week period, after post-treatment testing the rowers were allocated to the alternative training method, completing a cross-over design. The HIT produced significantly greater improvements in 2000 m time, 2000 m power and relative \( \dot{V}O_2^{\text{peak}} \) when compared to CT \((P < 0.05)\). It was concluded that four weeks of HIT improves 2000 m time-trial performance and relative \( \dot{V}O_2^{\text{peak}} \) in competitive rowers, more than CT.

Study 2. After establishing that HIT was effective in improving rowing performance the next step was to investigate if the combination of HIT and NaHCO₃ supplementation could further enhance performance. However, the research literature was still equivocal as to the most effective method of NaHCO₃ supplementation. Consequently, the aim of Study 2 was
to compare acute NaHCO₃ loading with serial NaHCO₃ loading (split doses over three days) in well-trained cyclists to establish which method was best for producing performance improvements and enhanced acid-base balance with minimal side effects.

Eight male cyclists completed three tests in a double blind, randomised design over a three week timeframe: acute NaHCO₃ loading (AL), serial NaHCO₃ loading (SL) and a placebo loading condition (P). Following each loading protocol, cyclists completed a 4-min performance test on a cycling ergometer. Both the AL and SL trials produced a significantly higher average power in the 4-min test when compared to the P trial ($P < 0.05$), with no significant difference between AL and SL trials ($P = 0.29$). The improvements in performance associated with the SL trial were despite any changes to the measure blood-gas variables (pH and HCO₃⁻). It was concluded that SL may provide a convenient and practical alternative approach for athletes preparing for competition; however, AL was the most effective for altering acid-base balance as well as improving performance with minimal negative side-effects, and was deemed the most appropriate method to use when combing HIT and NaHCO₃.

**Study 3.** With appropriate protocols for both HIT and NaHCO₃ loading in well-trained athletes confirmed, the aim of Study 3 was to combine these two strategies and investigate whether there was any additive benefit when used in a chronic training setting. Subjects were 12 elite rowers preparing for international competition. Following baseline testing, rowers were allocated to either NaHCO₃ (ALK) or a placebo (PLA) group (sodium chloride matched for equimolar sodium content). Both groups performed 8 HIT sessions over a 4-week period. Prior to each HIT session, subjects were required to ingest NaHCO₃ or a placebo substance. The 2000 m time-trial performance improved after 4 weeks of HIT; however, there were no statistically significant performance improvements ($P > 0.05$).
attributable to the NaHCO$_3$ supplementation during HIT training of fixed volume and intensity.

**Study 4.** Due to the results from Study 2 and 3, along with some inconsistencies in the literature regarding the influence of NaHCO$_3$ loading on athletic performance, it was hypothesised that a possible reason for lack of performance improvements after NaHCO$_3$ supplementation was the use of sodium chloride (NaCl) as a placebo. The sodium content has been proposed to provide some performance benefits, possibly through blood volume shifts, obscuring some of the benefits associated with NaHCO$_3$ supplementation, limiting its use as a valid placebo substance. Therefore the aim of Study 4 was to compare NaHCO$_3$ and NaCl to a physically inert substance by evaluating the haematocrit changes and their influence on high-intensity cycling performance. Subjects undertook three tests in a random, double-blind design over a one week timeframe: NaHCO$_3$ loading (SB), NaCl loading (SC) and dextrose loading (D). Following each loading protocol, subjects completed a 2-min performance test on a cycling ergometer. The SB trial produced a significantly higher ($P < 0.01$) mean power (W) in the 2-min test when compared to the SC and D trial with no significant difference between SC and D trials ($P > 0.05$). It was concluded that the HCO$_3^-$ not the Na$^+$ was primarily responsible for providing any ergogenic benefit during high-intensity exercise performance.

**Conclusions:** The findings from these studies suggest that independently, both HIT and NaHCO$_3$ supplementation can improve high-intensity exercise performance in well-trained athletes. However, this thesis provides the first study to investigate the combination of these two techniques in highly-trained athletes and provides evidence that such an approach does not lead to additional performance gains in this population; however, further research is warranted. The findings from the final study of the thesis suggest that it is the HCO$_3^-$ content in NaHCO$_3$ which is likely to facilitate performance benefits more so than
the Na⁺ content. The findings of the studies included in this thesis are applicable to high-intensity exercise performance in the context of high-level athletic competition. The research adds to the knowledge base regarding practical information for athletes and coaches in terms of novel NaHCO₃ loading and interval training protocols while providing likely performance outcomes.
Publications Arising From This Thesis

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Peer Reviewed Conference Proceedings


Awards/Grants


Statement of Candidate Contribution

This thesis comprises four research investigations which have been completed almost entirely by Matthew Driller (the candidate). The candidate designed the studies, coordinated and supervised all data collection, analysed the data, and prepared all manuscripts. The contributions of all parties to each of the four studies are detailed below.

Study one: The effects of high-intensity interval training in well-trained rowers

- Mr Matthew Driller: lead role in study design, data collection, statistical analysis and first author on manuscript (70%)
- Dr James Fell: assisted with study design, data collection and manuscript revision (20%)
- Dr Andrew Williams: assisted with study design, data collection, statistical analysis and manuscript revision (5%)
- Mr John Gregory: assisted with data collection (2.5%)
- Dr Cecilia Shing: assisted with data collection and manuscript revision (2.5%)

Study two: The effects of serial and acute NaHCO₃ loading in well-trained cyclists

- Mr Matthew Driller: lead role in study design, data collection, statistical analysis and first author on manuscript (80%)
- Dr James Fell: assisted with study design and manuscript revision (10%)
- Mr John Gregory: assisted with data collection (5%)
- Dr Andrew Williams: assisted with statistical analysis and manuscript revision (5%)
Study three: The effects of chronic sodium bicarbonate loading and interval training in highly-trained rowers

- Mr Matthew Driller: lead role in study design, data collection, statistical analysis and first author on manuscript (80%)
- Dr James Fell: assisted with study design, statistical analysis and manuscript revision (10%)
- Mr John Gregory: assisted with data collection (5%)
- Dr Andrew Williams: assisted with statistical analysis (5%)

Study four: The effects of NaHCO₃ and NaCl loading on performance

- Mr Matthew Driller: lead role in study design, data collection, statistical analysis and first author on manuscript (65%)
- Dr James Fell: assisted with study design, statistical analysis and manuscript revision (10%)
- Mr Sam Howe: assisted with data collection and manuscript revision (10%)
- Mr Phillip Bellinger: assisted with data collection and manuscript revision (10%)
- Dr Andrew Williams: assisted with statistical analysis and manuscript revision (5%)
There was one further study that was directly related to this thesis and it appears in the appendices (Appendix I). The study was derived from blood collected during the conduct of study one. Therefore, the candidate completed all data collection but did not perform the first draft of the final manuscript and as such has not been included as part of the body of the thesis. The contribution to the study is listed below:

**Study five: The effects of high-intensity interval training on plasma adiponectin in well-trained rowers**

- Dr Cecilia Shing: data collection, statistical analysis and first author (40%)

- Mr Matthew Driller: assisted with data collection and manuscript revision (30%)

- Dr James Fell: assisted with data collection and manuscript revision (15%)

- Ms Jess Webb: assisted with data collection, analysis of blood, and manuscript revision (10%)

- Dr Andrew Williams: assisted with data collection and manuscript revision (5%)

We the undersigned agree with the above stated “proportion of work undertaken” for each of the above published (or submitted) peer-reviewed manuscripts contributing to this thesis:

_Signed:_

Candidate __________________________

Signed: ______________________

Dr. James Fell
Supervisor
School Of Human Life Sciences
University of Tasmania

Professor Madeleine Ball
Head of School
School of Human Life Sciences
University of Tasmania

Date: 27/02/2012
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To my parents, thank you for supporting me in pursuing my career in sports physiology, even if you didn’t know there was such a thing and would prefer I got a “real job”. Thank you also for instilling in me the importance of hard work.
Lastly, I would like to thank my wife, Kirsty. It’s been a trying journey over the last 5 years - but we made it! Thank you for encouraging me to finish this thing and for letting me follow my dreams. Hopefully, we can now spend some more weekends together!
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## Abbreviations

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<th>Abbreviation</th>
<th>Definition</th>
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<tbody>
<tr>
<td>ADP</td>
<td>adenosine di-phosphate</td>
</tr>
<tr>
<td>ATP</td>
<td>adenosine tri-phosphate</td>
</tr>
<tr>
<td>°C</td>
<td>degrees celcius</td>
</tr>
<tr>
<td>CI</td>
<td>confidence intervals</td>
</tr>
<tr>
<td>CL</td>
<td>confidence limits</td>
</tr>
<tr>
<td>Ca²⁺</td>
<td>calcium ions</td>
</tr>
<tr>
<td>CV</td>
<td>coefficient of variation</td>
</tr>
<tr>
<td>GI</td>
<td>gastrointestinal</td>
</tr>
<tr>
<td>g·kg⁻¹·BM</td>
<td>grams per kilograms of body mass</td>
</tr>
<tr>
<td>[H⁺]</td>
<td>hydrogen ion concentration</td>
</tr>
<tr>
<td>H⁺</td>
<td>hydrogen ions</td>
</tr>
<tr>
<td>Hct</td>
<td>haematocrit</td>
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<tr>
<td>HIT</td>
<td>high-intensity interval training</td>
</tr>
<tr>
<td>[HCO₃⁻]</td>
<td>bicarbonate ion concentration</td>
</tr>
<tr>
<td>Pᵢ</td>
<td>inorganic phosphate</td>
</tr>
<tr>
<td>kg</td>
<td>kilograms</td>
</tr>
<tr>
<td>kJ</td>
<td>kilojoules</td>
</tr>
<tr>
<td>[K⁺]</td>
<td>potassium ion concentration</td>
</tr>
<tr>
<td>[La⁻]</td>
<td>lactate ion concentration</td>
</tr>
<tr>
<td>m</td>
<td>metres</td>
</tr>
<tr>
<td>min</td>
<td>minutes</td>
</tr>
<tr>
<td>mL</td>
<td>millilitres</td>
</tr>
<tr>
<td>mL·kg⁻¹·min⁻¹</td>
<td>millilitres per kilogram of body mass per minute</td>
</tr>
<tr>
<td>mmol·L⁻¹</td>
<td>millimoles per litre</td>
</tr>
<tr>
<td>mmol·kg⁻¹·BM</td>
<td>millimoles per kilogram of body mass</td>
</tr>
</tbody>
</table>
\( \beta_m \) muscle buffer capacity
\( \text{Na}^+ \) Sodium
\( \text{NaCl} \) sodium chloride
\( \text{NaHCO}_3 \) sodium bicarbonate
\( \text{NH}_4\text{Cl} \) ammonium chloride
\( \text{PPO} \) peak power output
\( \text{PV} \) plasma volume
\( \text{RPE} \) rating of perceived exertion
\( \text{SD} \) standard deviation
\( \text{SEM} \) standard error of measurement
\( \text{TEM} \) typical error of measurement
\( \dot{\text{VO}}_2_{\text{max}} \) maximal oxygen uptake
\( \dot{\text{VO}}_2_{\text{peak}} \) peak oxygen uptake
\( W \) watts
\( \text{Watts kg}^{-1}\text{BM} \) watts per kilogram of body mass
\( \mu\text{L} \) microlitres
Overview

The adaptation of skeletal muscles in response to exercise has been a focus of decades of research. Advances in the sport sciences have resulted in the ability of humans to become faster, stronger, more powerful and have enhanced endurance capabilities, as evidenced by world-record breaking performances that continue to occur in most sports. The beneficial effects of exercise on health and sports performance highlight the extraordinary ability of the human body to adapt to external stimuli. In particular, the ability of skeletal muscle to adapt in response to exercise has become a major focus for sports scientists and medical professionals worldwide. While advances in exercise physiology have greatly enhanced our understanding of skeletal muscle adaptation to exercise, many questions remain unanswered. Sport scientists have become interested in the manipulation of skeletal muscle fibres in athletes, aiming to gain advantage over their opponents. The search for any improvement, even as small as 1%, in elite athletes has caused sports scientists and coaches to look at optimal training methods and nutritional supplementation to enhance skeletal muscle adaptation. Therefore, the main purpose of this thesis is to examine the optimal application of some of these training methods and nutritional supplementation techniques in an athletic population.

Physical exercise causes a number of changes within the body. These changes include both metabolic and ionic changes within both the intracellular and extracellular environment. The demand placed on the body during high-intensity exercise elicits an increase in energy production for the working muscles. The production of this energy during high-intensity exercise is largely achieved through the resynthesis of a metabolite known as adenosine triphosphate (ATP) (Marieb & Hoehn, 2010). The resynthesis of ATP during exercise
causes changes to the ionic environment and contributes to an increase in hydrogen ion concentration ([H⁺]). While the effects of changes to [H⁺] on muscular fatigue remain controversial, a number of studies (Parkhouse, McKenzie, Hochachka, & Ovalle, 1985; Sahlin & Henriksson, 1984) show the athletic benefits of an improved ability to remove hydrogen ions (H⁺), and it has been suggested that the accumulation of H⁺ within the muscle cell may inhibit the muscle contraction process (Linderman & Gosselink, 1994). The ability to reduce the negative effects associated with the accumulation of H⁺ can be achieved through a number of methods. The methods employed tend to focus on improving the intracellular buffering capacity and thereby reducing the impact that the [H⁺] has on muscle function during exercise. Improved muscle buffer capacity (βm) can be achieved through exercise training-induced adaptation to muscles (Edge, 2006a). However, while exercise training is known to improve muscle buffer capacity, the optimal methods and protocols to bring about these benefits are still largely unknown. Improving the extracellular buffering capacity has also become a focus and can be achieved through nutritional supplementation.

Researchers have identified the role of the body’s extracellular buffering mechanisms as being a key component in reducing the consequences associated with an increase in [H⁺] (Linderman & Gosselink, 1994; Sahlin, 1978). One of the most effective extracellular buffering mechanisms in the body is the bicarbonate ion concentration ([HCO₃⁻]), accounting for some 15-18% of total buffer capacity (Sahlin, 1978). Therefore, the ingestion of sodium bicarbonate (NaHCO₃) has proven to be an effective means for enhancing the muscle buffering systems during exercise. The mechanism by which NaHCO₃ loading exerts its influence may be through the elevation of the extracellular [HCO₃⁻], which then increases rate of efflux of H⁺ from the intracellular space. The acute
effects of NaHCO$_3$ on exercise performance are well known; however, there is limited research examining the application of NaHCO$_3$ in a chronic setting. Furthermore, alternative protocols of NaHCO$_3$ supplementation need to be examined to provide a more practical method for athletes when preparing for competition and to reduce the negative side effects associated with traditional methods of NaHCO$_3$ loading (Burke & Pyne, 2007).

While there is a plethora of research investigating both training methods and acute NaHCO$_3$ supplementation to improve muscle function and consequently, exercise performance (Burke & Pyne, 2007; Laursen & Jenkins, 2002a), there remain unanswered questions pertaining to their combined effects, and their effects in a highly-trained athletic population. The purpose of this series of studies is to answer some of the questions related to the optimal application of NaHCO$_3$ supplementation and high-intensity interval training to highly-trained athletes in both an acute and chronic training setting.
Thesis Organisation

This thesis consists of seven chapters (Figure 1). Chapter one provides a general introduction to the thesis. Chapter two outlines a review of the literature and is separated into two main sections: A) the effect of high-intensity interval training (HIT) on physiology and performance, and B) the effect of NaHCO₃ supplementation on physiology and performance. Chapters three, four, five and six are all experimental studies, which are described below. Finally, chapter seven consists of the thesis summary and also discusses practical applications, limitations and future directions from the thesis.

Each experimental chapter is presented in paper format with its own introduction, methodology, results and discussion section. Experimental chapters appear in the format required by the individual journal guidelines that they have either been published or are currently under review in; however; slight formatting changes have been made to assist with the thesis flow. Additionally, for the ease of the reader, a single reference style was used throughout the thesis and all references have been placed together at the end of the thesis rather than at the end of each chapter.

The overall aim of this thesis was to investigate the performance effect of combining HIT and NaHCO₃ supplementation in highly-trained athletes. Given the physiological demands of both track-cycling and rowing are very similar, coupled with the fact that it is often difficult to access the same group of athletes from the same sport for multiple studies, the thesis alternated between cyclists and rowers with the intention that the results achieved for both modes could be relevant to the other population.

The progression of experimental studies is outlined below:
Study One (chapter three):

“The effects of high-intensity interval training in well-trained rowers”

The purpose of chapter three was to firstly establish an effective interval training protocol for improving performance in well-trained rowers. The study implemented an interval training protocol similar to ones used in cycling studies and compared it to a more traditional method of rowing training.

Study Two (chapter four):

“The effects of serial and acute NaHCO₃ loading in well-trained cyclists”

With an effective interval training protocol determined in study one, study two aimed to establish an effective method of NaHCO₃ supplementation. The study compared acute NaHCO₃ loading with serial NaHCO₃ loading (split doses over three days) in well-trained athletes.

Study Three (chapter five):

“The effects of chronic sodium bicarbonate loading and interval training in highly-trained rowers”

With effective protocols for both HIT and NaHCO₃ loading now established, the aim of study three was to combine these two strategies in a chronic training setting and investigate whether there was any additional benefit in highly-trained rowers.

Study Four (chapter six):

“The effects of NaHCO₃ and NaCl loading on haematocrit and high-intensity cycling performance”
Given the results from our previous studies, along with the inconsistencies in the literature regarding the success of NaHCO₃ loading and athletic performance, it was hypothesised that a possible reason for conflicting results was the use of NaCl as a placebo substance. Study four compared NaHCO₃ and NaCl to a physically inert substance on high-intensity cycling performance.
Figure 1 – Schematic of the thesis structure.
Chapter One:

Introduction
General Introduction

During exercise, the skeletal muscles transfer from a resting to a contracted state, resulting in a number of changes to both the intracellular and extracellular environment. These changes are dependent on the intensity of the exercise and the physiology of the individual. The cellular energy required to produce the excitation-contraction process of the skeletal muscles can be achieved through the breakdown of glycogen, protein and fat stored within the muscle, blood, liver and adipose tissue. During intense exercise, the primary source of energy for muscle contraction is derived from the breakdown of muscle glycogen and uptake of plasma glucose (Hargreaves, 1995). The process that derives energy from the breakdown of glucose and glycogen in the muscle cell, called glycolysis, results in the production of pyruvate and resynthesis of adenosine tri-phospate (ATP). Every time ATP is broken down to adenosine di-phosphate (ADP) and inorganic phosphate (P$_i$), a hydrogen ion (H$^+$) is released. The increase in glycolytic rate required to match ATP demand during high-intensity exercise results in an accumulation of pyruvate, which is readily altered to lactate by the lactate dehydrogenase enzyme. The formation of lactic acid has long been suggested to be the main cause of H$^+$ release during muscle contraction. In support of this hypothesis, both pyruvate and lactate have been identified as acids that have a low pK$_a$ and therefore, become fully dissociated/ionised and result in the release of H$^+$ (Hultman & Sahlin, 1980). When the ATP demand of muscle contraction is met by mitochondrial respiration (during lower intensity exercise), a portion of pyruvate is converted to acetyl coenzyme A and enters the Krebs cycle for further production of ATP (Figure 2). During mitochondrial respiration, the H$^+$ that are produced during metabolism are utilised (in the electron transport chain) to further produce ATP for exercise (Robergs, Ghiasvand, & Parker, 2004). The metabolic by-products of these reactions include carbon dioxide (CO$_2$) and water (H$_2$O). When exercise intensity increases beyond that which can be supplied
aerobically, H⁺ production exceeds the rate of use by the mitochondria, resulting in H⁺ accumulation within the cell.

**Figure 2** - Diagram of the glycolysis process that occurs during exercise. The diagram outlines three phases that depict the final outcome of the pyruvic acid. Phase 1: two ATP molecules activate glucose into fructose-1, 6-bisphosphate. Phase 2: fructose-1,6-bisphosphate is cleaved into two 3-carbon isomers called dihydroxyacetone phosphate and glyceraldehyde phosphate. Phase 3: the 3-carbon sugars are oxidised (reducing NAD+), inorganic phosphate groups (Pi) are attached to each oxidized fragment and the phosphates are cleaved by ADP to form four ATP molecules. The final products are two pyruvic acid molecules, two NADH + H⁺ molecules and a gain of two ATP molecules. Adapted from Benjamin Cummings Publishing, 2006. (Marieb & Hoehn, 2010).
As depicted in Figure 2, pyruvic acid, the end result of glycolysis, may proceed in one of two directions; 1) it can be shuttled into the mitochondria (Krebs cycle); or 2) it can be converted to lactate. When pyruvate is converted to lactate, ATP resynthesis occurs at a faster rate, but is limited in duration. This process is sometimes called anaerobic glycolysis (or fast glycolysis). However, when pyruvate is shuttled into the mitochondria to enter the Krebs cycle, the ATP resynthesis rate is slower, but can occur for a longer duration if the exercise intensity is low enough. This process is often referred to as aerobic glycolysis (or slow glycolysis) (Marieb & Hoehn, 2010).

While not necessarily being the direct cause (Robergs, Ghiasvand, & Parker, 2005), the accumulation of lactate produced from glycolysis is associated with the accumulation of H\(^+\) (Sahlin, Harris, Nylind, & Hultman, 1976). The increase in lactate and [H\(^+\)] coincides with cellular acidosis. Associated with these metabolic changes is a decrease in muscle pH. The literature suggests that muscular fatigue resulting from high-intensity exercise is due, partly, to this decrease in intramuscular pH (Costill, Verstappen, Kuipers, Janssen, & Fink, 1984). Furthermore, recovery has been associated with the rapid removal of lactate and H\(^+\) from muscle cells (Costill, et al., 1984). Support for the contention that decreased pH (whether intra- or extracellular) may be performance-limiting in high-intensity exercise comes from many sources. The lowering of pH levels is thought to hinder sarcoplasmic reticulum function (Nakamura & Schwartz, 1970), glycolytic flux (Newsholme & Start, 1973), myofilament interaction and force generation (Chase & Kushmerick, 1988). More specifically, the ability of troponin (a myofibrillar protein) to bind to calcium is inhibited as pH decreases, resulting in a reduced ability for actin to bind to myosin which is ultimately responsible for muscle contraction (Fuchs, Reddy, & Briggs, 1970). Phosphofructokinase, a key enzyme in the process of glycolysis, is also impaired by a
decrease in pH (Trivedi & Danforth, 1966). Therefore, while it is not fully understood how an increase in [H+] contributes to peripheral fatigue, there are many likely contributing factors as discussed above.

**Importance of muscle buffer capacity**

The buffering capacity of skeletal muscle (βm) appears to depend on the metabolic demands placed on the tissue, with extensive variation across different animal species (Figure 3) (Harris, Marlin, Dunnett, Snow, & Hultman, 1990). Animals that are regularly exposed to hypoxia and/or involved in very intense physical activity have a higher βm (Abe, Dobson, Hoeger, & Parkhouse, 1985). High-speed land animals, including the horse and dog, have been reported to have elevated βm above that of foraging animals (e.g. sheep and cows) (Harris, et al., 1990). The greater βm is suggested to be due to the intense physical efforts performed by these animals, often to escape from predators or capture prey (Harris, et al., 1990). Humans have a low βm compared to a number of animal species that are designed for fast locomotion (e.g. dogs, horses), but have a greater βm than some foraging animals such as cows and pigs (Abe, 2000; Harris, et al., 1990). Even within species, there appears to be subtle differences in the buffering capacity (Harris, et al., 1990). Differences in βm have been reported in studies comparing trained and untrained human subjects (Parkhouse, et al., 1985). Furthermore, there seems to be a difference in βm amongst athletes from different sports, with rowers and sprinters having significantly enhanced βm when compared to endurance runners (Figure 4) (Parkhouse, et al., 1985). Moreover, fast-twitch muscle fibres have an elevated βm compared to slow-twitch muscles fibres (Abe, et al., 1985).
**Figure 3** – In-vitro muscle buffer capacity ($\beta_{m\text{-vitro}}$) in major muscles of the thoroughbred horse, greyhound dog and man (adapted from Harris et al., 1990).
Figure 4 - In-vitro muscle buffering capacity ($\beta_{\text{min vitro}}$) in the vastus lateralis of untrained subjects, rowers, sprinters and marathon runners (Adapted from Parkhouse et al., 1985).
Muscle buffer capacity in response to exercise

Perhaps one of the most important peripheral adaptations associated with HIT, is the effect that this kind of training has on muscle buffer capacity. A number of studies have compared the $\beta m$ between different types of athletes, as well as the comparison of $\beta m$ between trained and untrained subjects. Furthermore, several studies have investigated both the acute and chronic changes to $\beta m$ following exercise bouts and periods of training, respectively. An elevated $\beta m$ in humans has been associated with better performance in a number of athletic settings. Recreational runners with the highest $\beta m_{\text{in-vitro}}$ had the fastest 200 m times, highest peak power output and largest increases in muscle lactate during a treadmill sprint ($P < 0.05$) (Nevill, Boobis, Brooks, & Williams, 1989). Enhanced $\beta m$ has also been associated with endurance performance and recovery between repeated sprints. Bishop et al. (2004) showed that $\beta m_{\text{in-vitro}}$ was related to work decrement during repeated sprints ($P < 0.05$). Superior $\beta m_{\text{in-vitro}}$ has also been associated with enhanced 40-km time-trial cycling performance in well-trained subjects ($P < 0.05$) (Weston, et al., 1997). This would suggest that $\beta m$ may not only be important for short-duration exercise bouts, but also for exercise that requires sustained periods of high-intensity exercise. Furthermore, athletes in sports involving short sprints with little recovery time (such as most team-sports), may also benefit from having an enhanced $\beta m$. Some of the aforementioned studies evaluating changes in $\beta m$ after a period of high-intensity training also showed performance improvements in the experimental group when compared to the control group (Bell & Wenger, 1988; Christensen, Bagger, Juel, Hojlund, & Pedersen, 2000; Troup, Metzger, & Fitts, 1986; Weston, et al., 1997). The corresponding peripheral adaptations to $\beta m$ following HIT will be explored further in Part A of the literature review.
**Extracellular buffering**

A popular method of improving buffer capacity in sports performance, is supplementation with NaHCO₃, which increases extracellular pH and HCO₃⁻ levels (Burke & Pyne, 2007). The increase in these extracellular concentrations act to enhance the bodies buffering systems during exercise by increasing the rate of efflux of H⁺ from inside the muscle cell (Linderman & Gosselink, 1994). However, not all studies on NaHCO₃ and sports performance have reported successful applications in the athletic arena and these inconsistencies may be due to the different loading/dosage strategies employed and also the training status of the subjects used.

There is some recent evidence to suggest that a combination of HIIT and NaHCO₃ supplementation may produce positive performance results in recreationally-trained subjects through enhancing muscle buffering capacity. However, it is unknown whether these same benefits could be applied to their highly-trained counterparts. The effects of extra-cellular buffering will be further examined in Part B of the literature review.

**Summary**

The tolerance of high-intensity exercise may be limited by the ability of the body to counteract decreases in intracellular (muscle) and extracellular (blood) pH through its intrinsic buffering systems. Thus, it has been hypothesised that increasing the body’s buffering capacity would protect against acidosis and thereby delay the onset of muscle fatigue during exercise (Costill, et al., 1984). The intracellular content of protein, HCO₃⁻, P, and carnosine have been identified as important physical/chemical buffers within humans and have been shown to be affected by training status (Edge, 2006a). Exercise training has
also been shown to improve $\beta$m and $[H^+]$ regulation (Bell & Wenger, 1988). However, not all training studies have reported improved $\beta$m (Harmer, et al., 2000; Pilegaard, et al., 1999). These findings may be the result of the subjects and/or the training methods employed. It seems that HIT sessions are the most beneficial for $\beta$m adaptation, when compared to either low-moderate endurance training or resistance training.

The purpose of the following review of literature is to outline the aforementioned strategies in improving intra- and extracellular buffering capacity during exercise and to outline the effects these strategies have on both the physiology and performance in highly-trained athletes. Accordingly, the review will be divided into two sections A) The effect of high-intensity interval training on physiology and performance, and; B) The effect of NaHCO$_3$ supplementation on physiology and performance. Conclusions will then be made with regard to the possibility of combining these two strategies to further enhance performance in well-trained athletes.
Chapter Two:

Literature Review:

PART A - The effect of high-intensity interval training on physiology and performance
High-intensity interval training (HIT)

A number of definitions exist for HIT. According to Daniels & Scardina (1984), HIT involves repeated bouts of exercise (~95-100% of $\dot{V}O_2$\textsubscript{max}) lasting between 30 s-5 min, with rest periods the same length or slightly shorter than the work period (Daniels & Scardina, 1984). Billat (2001a) stated that HIT is repeated short to long bouts of high-intensity exercise (equal or superior to the maximal lactate steady-state velocity) interspersed with recovery periods of light exercise or even rest (Billat, 2001a). Similarly, Laursen & Jenkins have defined HIT as repeated bouts of short-moderate duration exercise lasting between 10-s and 5-min completed at an intensity that is greater than anaerobic threshold with the rest period allowing for a small amount of recovery prior to the next repetition (Laursen & Jenkins, 2002a). The only point of agreement among scientists, coaches and athletes regarding the definition of HIT is that it includes alternate periods of exercise and recovery. The early work of Åstrand et al. (1960) and Christensen et al. (1960) began investigation into what has become a popular research topic within the sports sciences. The important findings from those early studies showed that the length of the work bouts is the critical factor in determining the body’s adaptation to exercise. Additionally, they found that intermittent exercise allowed much more high-intensity work to be performed when compared to continuous exercise. By exposing the body to high amounts of stress in short bursts, researchers found that similar or even greater gains in fitness could be achieved through lower volumes of training. Indeed, this needed to be balanced by appropriate periods of recovery, allowing some level of waste removal, and fuel replenishment in order to perform the next high-intensity bout (Daniels & Scardina, 1984). Since the early research on HIT, numerous studies have shown that varying the intensity of exercise and the work to rest ratio during HIT can result in different degrees of stress on both aerobic and anaerobic systems. It was also established early on in HIT
research that there was a need for a high degree of specificity, emphasising the importance of the development of both peripheral and central adaptations to exercise training (Saltin, 1976).

**Physiological rationale for HIT**

The primary purpose of HIT is to repeatedly stress the physiological systems involved during exercise. This form of training is commonly used to load the training stimulus by using maximal or supra-maximal sport specific activity to induce a training response. The benefits associated with HIT can be attributed to both peripheral and central adaptations. The magnitudes of peripheral and central adaptations are highly dependent on the intensity, duration and frequency of the training bout (Laursen, 2010; Laursen & Jenkins, 2002a).

**Peripheral adaptations to HIT**

Peripheral adaptations refer to the ability of the skeletal muscles to produce and use ATP during exercise. Additionally, as alluded to in the introduction, a major peripheral adaptation to HIT is the $\beta_m$ capacity, and therefore, will be the main focus for this section of the literature review.

**HIT induced changes to muscle buffer capacity**

Limited evidence suggests that intense exercise may initially result in a decrease in $\beta_m$. The $\beta_m\text{in-vitro}$ of the gastrocnemius muscle was found to be significantly decreased two days following stimulated eccentric exercise in rats (Pilegaard & Asp, 1998). This can
possibly be attributed to the loss of carnosine from the contracting muscle during exercise (Dunnett, Harris, Dunnett, & Harris, 2002). Carnosine is a dipeptide and it has proven to be a powerful buffering agent to maintain acid-base balance during high-intensity exercise (Suzuki, Ito, Takahashi, & Takamatsu, 2004). Therefore, it seems plausible that the metabolic acidosis associated with intense exercise may be a contributing factor in reducing $\beta m$ after an acute exercise bout.

In contrast to the observed changes in $\beta m$ after an acute exercise bout, chronic training appears to increase $\beta m$. However, research relating to the changes in $\beta m$ following a period of training have highlighted that it is likely to be the training intensity, not the volume that is the most important factor in increasing $\beta m$. The findings from Parkhouse et al. (1985) support this theory (Figure 4). Parkhouse et al. (1985) reported that sprint and rowing athletes were more likely to have enhanced $\beta m$ when compared to the endurance or untrained subjects because of the regular, intense interval-type nature of training. Furthermore, there was no difference in $\beta m$ between the endurance-trained and untrained subjects, suggesting that high-volume, moderate-intensity training does not improve $\beta m$ above that of untrained subjects (Parkhouse, et al., 1985). Sahlin and Henriksson (1984) evaluated the $\beta m$ in team-sport athletes (ice-hockey, soccer, basketball) and found that the athletes had significantly elevated $\beta m$ when compared to untrained subjects (Sahlin & Henriksson, 1984). These results suggest that athletes involved in sports of an intense nature, are likely to have a greater $\beta m$ due to type of training they perform; however, rather than studying the $\beta m$ in a cross-section of different athletes, longitudinal training studies are required in order to truly evaluate training-induced changes in $\beta m$.

Bell and Wenger (1988) investigated the effects of sprint-cycling training (15-20 x 20 s sprints at 150% of power output at $\dot{V}O_2_{peak}$ with 60 s rest), four days per week for seven
weeks on $\beta m_{\text{in-vitro}}$ in untrained subjects. The results showed significant improvements in $\beta m_{\text{in-vitro}}$ of around 15% (Bell & Wenger, 1988). Sharp et al. (1986) showed even greater improvements in $\beta m$ following eight weeks of sprint-interval training on a cycle ergometer. The previously untrained group performed 8 x 30-s sprints, separated by 4-min rest (4 days a week) and increased their $\beta m_{\text{in-vitro}}$ by 37% at the end of the training intervention (Sharp, Costill, Fink, & King, 1986). Improvements have also been seen in trained athletes after increasing the intensity of their training. Christensen et al. (2000) had well-trained runners increase their training intensity by 30%, 60% or no change (control) from their normal training and matched groups on training volume. The researchers found that after 4-weeks of training both the control group and the 30% group had no significant improvements in $\beta m_{\text{in-vitro}}$; however, the 60% group showed a significant improvement of 24% ($P < 0.05$) in $\beta m_{\text{in-vitro}}$ after the training intervention when compared to the control (Christensen, et al., 2000). Similarly, Weston et al. (1997) recruited well-trained cyclists and replaced some regular endurance training sessions with high-intensity intervals at 80% of peak-power (60 s rest). After just six interval-sessions over a four-week period, they reported significant improvements in $\beta m_{\text{in-vitro}}$ (~16%) (Weston, et al., 1997). This suggests that well-trained endurance athletes may also be able to improve $\beta m$ by increasing the intensity of training.

While there is limited research on the effect of training on $\beta m$ in humans, animal studies have given further insight into $\beta m$ changes with exercise. For example, Troup et al. (1986) showed an improved $\beta m_{\text{in-vivo}}$ of ~10% following six weeks of sprint interval training in rats (Troup, et al., 1986). The improved $\beta m_{\text{in-vivo}}$ was seen in all muscle groups including the soleus, extensor digitorium longus and the vastus lateralis muscle groups following sprint treadmill running (4.5 min x 6, separated by 2.5 min rest).

The effects of other training methods on $\beta m$ have also been investigated; however, it seems that high-intensity interval training sessions are the most beneficial for $\beta m$ adaptation.
Mannion et al. (1994) studied the effects of resistance training (25 max reps x 6 sets separated by 30 s, 3 x a week for 16 weeks) and found no significant change in $\beta_m$ in-vitro (Mannion, Jakeman, & Willan, 1994). Pilegaard et al. (1999) also investigated the use of resistance training (leg extensions, 3-5 x per week for 8 weeks) and found no change in $\beta_m$. Additionally, studies evaluating the effect of low-moderate intensity training have also found negligible changes to $\beta_m$ (Weston, Wilson, Noakes, & Myburgh, 1996). Therefore, it can be assumed that the key variable in increasing $\beta_m$ is the exposure to repeated high-intensity exercise training. Furthermore, athletes involved in high-intensity sports, with already well-developed $\beta_m$ may find it harder to attain any further increases in $\beta_m$ through high-intensity training alone (Edge, Bishop, & Goodman, 2006b). Edge et al. (2006b) evaluated changes in $\beta_m$ after 8-weeks of high-intensity interval training in recreational subjects. They found a significant correlation between pre-training $\beta_m$ and percent change in $\beta_m$ after training ($r = -0.70; P < 0.05$). Because of this relationship between pre-training $\beta_m$ and percent change in $\beta_m$, they then placed subjects into either a high ($\geq 160 \mu \text{mol H}^+ \cdot \text{g muscle dry wt}^{-1} \cdot \text{pH}^{-1}$) or low ($\leq 140 \mu \text{mol H}^+ \cdot \text{g muscle dry wt}^{-1} \cdot \text{pH}^{-1}$) pre-training $\beta_m$ group in order to analyse the changes. There was a significant improvement in $\beta_m$ for the low-$\beta_m$ group (31%), with no-change in the high-$\beta_m$ group (4%). These results support the notion that athletes with less-developed $\beta_m$ systems can gain further improvements in $\beta_m$ when adding HIT to their program. While various methods of HIT seem to be effective in improving $\beta_m$ when compared to continuous low-moderate intensity exercise; there are also many other peripheral adaptations that occur in response to HIT, of which only a few will be mentioned in this review.
Other peripheral adaptations to HIT

A large focus in the literature on the peripheral adaptations to HIT in athletic populations has investigated the effect that this type of training has on $\beta$m. While somewhat less focus has been given to other peripheral adaptations, especially amongst trained human subjects, there are still a number of peripheral mechanisms that lead to performance improvements in an athletic setting. HIT has been proven to increase both oxidative and glycolytic enzyme activity coupled with an improved exercise capacity in untrained and recreationally active individuals (Linossier, Denis, Dormois, Geyssant, & Lacour, 1993; MacDougall, et al., 1998; Rodas, Ventura, Cadefau, Cussa, & Parra, 2000). More specifically, training intensities above $\dot{V}O_2\max$ have shown to increase both glycolytic and oxidative enzymes (MacDougall, et al., 1998). Much of the focus on peripheral HIT adaptation has been on the changes associated with phosphocreatine, adenosine triphosphatase and mitochondrial enzymes such as succinate dehydrogenase, citrate synthase and malate dehydrogenase as well as glycolytic enzymes such as hexokinase and phosphofructokinase following exercise (Bogdanis, Nevill, Boobis, Lakomy, & Nevill, 1995; Burgomaster, Heigenhauser, & Gibala, 2006; Gaitanos, Williams, Boobis, & Brooks, 1993; MacDougall, et al., 1998). Some of the other peripheral adaptations following HIT are thought to be greater skeletal muscle oxidative capacity, greater use of fatty acids as a fuel during exercise (Shepley, et al., 1992), the improved efficiency of Na$^+$-K$^+$ cation pumps (Laursen & Jenkins, 2002a), the increases in myoglobin (Green, 2000), capillary density (Bishop, Jenkins, McEniery, & Carey, 2000) and changes in fibre type characteristics (Esbjörnsson, Hellsten Westing, Balsom, Sjödin, & Jansson, 1993; Linossier, et al., 1993).
The ability of HIT to elicit rapid changes in skeletal muscle oxidative capacity is likely to be related to its potential to stress type II fibres in particular, but the mechanisms are still unclear. From a cell-signalling perspective, exercise is typically classified as either strength or endurance, with short-duration high-intensity work usually associated with increased skeletal muscle mass and prolonged low intensity work associated with increased mitochondrial mass and oxidative enzyme activity (Baar, 2006). Relatively little is known regarding the intracellular signalling that elicits skeletal muscle changes in response to HIT. It seems likely that metabolic adaptations to this type of exercise could be mediated through pathways normally associated with endurance training (Gibala & McGee, 2008).

A key regulator of oxidative enzyme expression in a number of cell types including skeletal muscle, is peroxisome proliferator-activated receptor-γ coactivator 1 alpha (PGC-1α). PGC-1α is partly responsible for inducing a fast to slow fibre-type conversion (Lin, et al., 2002). This change in phenotype is accompanied by enhanced mitochondrial enzyme expression and an increase in time to fatigue when electrically stimulated (Lin, et al., 2002). Endurance exercise increases PGC-1α activity and expression, suggesting that PGC-1α could be a critical component of the adaptive response to low-intensity endurance training (Gibala & McGee, 2008). However, Burgomaster et al. (2008) found that six weeks of low-volume HIT (4-6 x 30 s intervals with ~4 min recovery, 3 times a week) increased PGC-1α protein content in human skeletal muscle similar to high-volume endurance training (40-60 min at 65% \( \dot{VO}_2 \) peak, 5 times a week), suggesting that the same muscular adaptations can occur with a lower volume of training when implementing HIT.
Central adaptations to HIT

As mentioned, there are many different peripheral adaptations that occur in response to HIT, one of the major ones being the changes to $\beta_m$. However, there are also a number of central adaptations that occur with HIT that are worth investigating. Central adaptations to HIT include the ability to deliver oxygen to working skeletal muscles. Improvements in oxygen delivery to exercising muscles during high-intensity exercise can be attributed to an increase in stroke volume (Rowell, 1993). The increase in stroke volume following HIT has also been shown to increase $\dot{VO}_2_{max}$ in 69 healthy males (Fox, et al., 1975). The authors suggested that 13-wk of HIT led to an improved $\dot{VO}_2_{max}$, stroke volume, and lower circulatory stress as evidenced by decreased heart rate during sub-maximal exercise. Furthermore, Wisløff et al. (2007), found superior cardiovascular effects following HIT when compared to continuous training in heart failure patients. The study showed that HIT aided in the improvement of left ventricular function, endothelial function and $\dot{VO}_2_{max}$ in post-infarction heart failure patients (Wisloff, et al., 2007). There is a plethora of research supporting the claim that HIT improves $\dot{VO}_2_{max}$ in trained athletes (Table 1), which of course, may be attributed to a number of both central and peripheral adaptations. The table presents the variety of HIT studies that have shown a benefit to $\dot{VO}_2_{max}$ with improvements of 0.7-7.1% after 2-14 weeks of HIT.

Plasma volume expansion has been regarded as the single most important event in promoting cardiovascular stability and improving thermoregulation during prolonged exercise (Convertino, 1991). A possible mechanism by which HIT may improve central adaptations is by improving the cutaneous blood flow and sweat rate during exercise (Pandolf, 1979). HIT can elicit improved work-heat tolerance in physically active individuals (Gisolfi, 1973), but this has yet to be investigated in athletes.
For the purpose of this review and indeed, this thesis, the numerous benefits that HIT has on both the peripheral and central adaptations is only discussed briefly, as the main focus of HIT in this thesis will be on athletic performance. However, even though these areas could have been explored in much more detail, they need to be acknowledged as they play a major role in the understanding of the physiological mechanisms behind the performance improvements.
Table 1 – Effects of high-intensity interval training on maximum oxygen consumption (\(\dot{\text{VO}}_2\text{max}\)) in competitive athletes (Adapted from Paton & Hopkins, 2004).

<table>
<thead>
<tr>
<th>Performance test</th>
<th>Change in (\dot{\text{VO}}_2\text{max}) (%)</th>
<th>Experimental training</th>
<th>Duration of training</th>
<th>Subjects(^a)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cycling</td>
<td>9.3</td>
<td>Max intervals</td>
<td>3.wk(^{-1}) for 2 wk</td>
<td>35 M, 13 F trained</td>
<td>Hazell et al. (2010)</td>
</tr>
<tr>
<td>Rowing</td>
<td>7.0</td>
<td>Submax intervals</td>
<td>2.wk(^{-1}) for 4 wk</td>
<td>5 M, 5 F rowers(^b)</td>
<td>Driller et al. (2009)</td>
</tr>
<tr>
<td>Running</td>
<td>6.9</td>
<td>Submax intervals</td>
<td>2.wk(^{-1}) for 7 wk</td>
<td>21 M footballers</td>
<td>Bravo et al. (2008)</td>
</tr>
<tr>
<td>Cycling</td>
<td>2.6 at 2 wk; 7.1 at 4 wk</td>
<td>Max intervals (short recovery)</td>
<td>2.wk(^{-1}) for 4 wk</td>
<td>8+11 M cyclists</td>
<td>Laursen et al. (2002c)</td>
</tr>
<tr>
<td></td>
<td>2.0 at 2 wk; 4.4 at 4 wk</td>
<td>Max intervals (long recovery)</td>
<td>2.wk(^{-1}) for 4 wk</td>
<td>8+11 M cyclists</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3.5</td>
<td>Supramax intervals</td>
<td>2.wk(^{-1}) for 2 wk</td>
<td>7+7 M cyclists</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.8 at 2 wk; 2.2 at 4 wk</td>
<td>Supramax intervals</td>
<td>2.wk(^{-1}) for 4 wk</td>
<td>8+11 M cyclists</td>
<td></td>
</tr>
<tr>
<td>Cycling</td>
<td>4.6 – 8.5</td>
<td>Submax intervals</td>
<td>2.wk(^{-1}) for 7 wk</td>
<td>29 M, 6 F cyclists</td>
<td>Seiler et al. (2011)</td>
</tr>
<tr>
<td>Running</td>
<td>4.9</td>
<td>Max intervals</td>
<td>2.wk(^{-1}) for 4 wk</td>
<td>5 M runners</td>
<td>Smith et al. (1999)</td>
</tr>
<tr>
<td>Cycling</td>
<td>2.3</td>
<td>Max intervals</td>
<td>2.wk(^{-1}) for 3 wk</td>
<td>12 M cyclists</td>
<td>Stepto et al. (1999)</td>
</tr>
<tr>
<td>Running</td>
<td>2.2</td>
<td>Submax intervals</td>
<td>1.wk(^{-1}) for 14 wk</td>
<td>8 M runners</td>
<td>Sjodin et al. (1981)</td>
</tr>
<tr>
<td>Running</td>
<td>2.1</td>
<td>Submax and max intervals</td>
<td>2.wk(^{-1}) for 4 wk</td>
<td>8 M runners</td>
<td>Billat et al. (1999)</td>
</tr>
<tr>
<td>Skiing</td>
<td>2.0</td>
<td>Explosive sport-specific movements</td>
<td>6.wk</td>
<td>7+8 M cross-country skiers</td>
<td>Paavolainen et al. (1991)</td>
</tr>
<tr>
<td>Skiing</td>
<td>3.4 to -3.9(^b)</td>
<td>Explosive sport-specific movements</td>
<td>3.wk(^{-1}) for 9 wk</td>
<td>8+7 F cross-country skiers</td>
<td>Hoff et al. (1999)</td>
</tr>
<tr>
<td>Skiing</td>
<td>1.4(^c)</td>
<td>Explosive sport-specific movements</td>
<td>3.wk(^{-1}) for 8 wk</td>
<td>9+10 M cross-country skiers</td>
<td>Hoff et al. (2002)</td>
</tr>
<tr>
<td>Running</td>
<td>0.7</td>
<td>Max intervals</td>
<td>3.wk(^{-1}) for 8 wk</td>
<td>7 M runners</td>
<td>Acevedo and Goldfarb (1989)</td>
</tr>
</tbody>
</table>

Supramax, supraximal; max, maximal; submax, submaximal; M, male; F, female; \(\dot{\text{VO}}_2\), oxygen consumption.

Changes based on \(\dot{\text{VO}}_2\) in L.min\(^{-1}\).

\(^a\)Numbers are experimental + control.

\(^b\)Indicates a cross-over design.

\(^c\)Wide inconsistency between \(\dot{\text{VO}}_2\) in L.min\(^{-1}\), ml.min\(^{-1}\).kg\(^{-1}\) and ml.min\(^{-1}\).kg\(^{0.67}\) probably due to \(~3\%\) increase in body mass in resistance group.

\(^d\)Estimated from \(\dot{\text{VO}}_2\) in ml.min\(^{-1}\).kg\(^{-1}\) by adding 0.2% change in body mass.
The influence of HIT in trained and untrained populations

For already well-trained athletes, improvements in performance become difficult to attain and increases in training volume can potentially yield no improvements. Consequently, athletes and coaches must find alternative approaches to achieve greater physiological and performance gains (Billat, 2001a). Previous research would suggest that, for athletes who are already trained, improvements in endurance performance can be achieved through HIT (Laursen, 2010; Mujika, 2010). HIT studies performed on cyclists, swimmers and runners, have shown improvements in variables such as \( \dot{\text{VO}}_2 \text{max} \) (Costill & Winrow, 1970), peak-power output (Hawley & Noakes, 1992), lactate threshold (Farrell, Wilmore, Coyle, Billing, & Costill, 1979), utilization of \( \dot{\text{VO}}_2 \text{max} \) at the same power outputs (Costill, 1972) and time-trial performance (Lindsay, et al., 1996). In the majority of these studies, the prescription of intensity for the intervals has been based on power output, speed or velocity at \( \dot{\text{VO}}_2 \text{max} \). Furthermore, performance gains following HIT seem to be more easily achieved in untrained or sedentary subjects when compared to more highly-trained populations.

Untrained

While it is interesting to note the large changes in physiology and performance following the abundance of HIT studies in untrained populations, the focus of this review and indeed this thesis is aimed more at trained athletes. However, it is worth noting that the implementation of HIT in untrained/recreational or even sedentary subjects, has resulted in significant improvements in performance over a range of sports (Laursen & Jenkins, 2002a). Laursen & Jenkins (2002a) suggested that in sedentary (\( \dot{\text{VO}}_2 \text{max} \leq 45 \, \text{mL-kg}^{-1} \cdot \text{min}^{-1} \)) and recreationally active individuals (\( \dot{\text{VO}}_2 \text{max} \text{ 45-55 mL-kg}^{-1} \cdot \text{min}^{-1} \)) several years
of training may be required to achieve a $\dot{V}O_2_{\text{max}}$ comparable to highly-trained athletes ($\dot{V}O_2_{\text{max}} \geq 60 \text{ mL.kg}^{-1}.\text{min}^{-1}$) (Ekblom, 1968; Rowell, 1993). However, Hickson et al. (1977) showed significant improvements in $\dot{V}O_2_{\text{max}}$ ($P < 0.05$) of 16.8 mL.kg$^{-1}$.min$^{-1}$ (44%) in eight sedentary and recreational subjects after 10 weeks of cycling and running HIT (40 min per day, 6 days/week) (Hickson, Bomze, & Holloszy, 1977). Furthermore, 50% of the subjects approached or exceeded a $\dot{V}O_2_{\text{max}}$ of 60 mL.kg$^{-1}$.min$^{-1}$. In another study using untrained individuals (n=21), HIT (5 x 4 min running at 100% $\dot{V}O_2_{\text{max}}$ with 2 min recovery, 3 times a week for 7 weeks) was found to significantly enhance the oxidative capacity of type II fibres ($P < 0.05$) when compared to a continuous exercise group performing the same duration and average intensity (79% $\dot{V}O_2_{\text{max}}$) (Henriksson & Reitman, 1976). Similar outcomes have also been supported in a study on untrained rats, where mitochondrial fatty acid oxidation rates have been shown to increase after 6 sessions of HIT per week for 12 weeks (Chilibeck, Bell, Farrar, & Martin, 1998).

**Trained**

Compared to the volume of research that describes the physiological adaptations to traditional endurance exercise training in sedentary to moderately-trained individuals, relatively little work has examined the physiological and performance responses of already well-trained athletes to HIT. One of the earliest and most cited scientific studies examining HIT in well-trained athletes was conducted by Acevedo and Goldfarb in the late 1980’s (Acevedo & Goldfarb, 1989). The study investigated seven competitive long-distance runners who underwent 8 weeks of HIT at 90 to 95% of peak heart rate. The HIT significantly improved their 10km running performances by ~3% (35:27 to 34:24 min:s)
and increased their supra-maximal endurance by ~20%. A study that was written up for numerous publications evaluating different aspects of the results (Lindsay, et al., 1996; Westgarth-Taylor, et al., 1997; Weston, et al., 1997) examined the effects of HIT in male competitive cyclists (\(\dot{V}O_2\max \geq 65\ \text{mL-kg}^{-1}\text{-min}^{-1})\). Following baseline testing, the cyclists replaced ~15% of their ~300 km/week endurance training with 6 to 12 sessions of HIT. The HIT sessions took place twice a week and consisted of 6-9 intervals at 80% of peak power separated by 1-min rest periods. The study reported improvements in 40 km time-trial performance of 3-3.5% after 6-12 HIT sessions (Lindsay, et al., 1996; Westgarth-Taylor, et al., 1997). In a review article on training techniques, Hawley and colleagues concluded that the 4-5% improvements in peak power values (not time-trial performance) after 4-6 HIT sessions in the study by Lindsay et al. and Westgarth-Taylor et al. were not further improved when up to 12 sessions were implemented (Hawley, Myburgh, Noakes, & Dennis, 1997). It should be noted, however, that some of these studies did not include a control group in their design (Acevedo & Goldfarb, 1989; Lindsay, et al., 1996; Stepto, et al., 1999; Westgarth-Taylor, et al., 1997; Weston, et al., 1997). Therefore, some of the performance improvements may be, in-part, related to psychological factors or the phase of training.

Interval training at intensities around the velocity or power achieved at \(\dot{V}O_2\max\) (lasting from 2-10 min), improves mainly sub-maximal endurance performance (by ~6%) (Paton & Hopkins, 2004). These performance improvements can be largely attributed to improvements in all three components of the aerobic system (\(\dot{V}O_2\max\), anaerobic threshold, economy) (Paton & Hopkins, 2004). Table 2 summarises the effects of HIT on different types of performance tests. The studies have been divided into different intensities of performance tests comprising of; sub-maximal, maximal and supra-maximal tests.
Table 2 – Effects of high-intensity training on measures of endurance performance in competitive athletes. Performance is expressed as change in mean power in a sport-specific time-trial or its equivalent (Adapted from Paton & Hopkins, 2004).

<table>
<thead>
<tr>
<th>Performance test</th>
<th>Change in power (%)</th>
<th>Experimental training</th>
<th>Duration of training</th>
<th>Subjects</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Submaximal Tests</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-h 40-km cycling</td>
<td>12.4&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Max intervals (short recovery)</td>
<td>12 sessions over 6-7 wk</td>
<td>8 M cyclists</td>
<td>Westgarth-Taylor et al. (1997)</td>
</tr>
<tr>
<td>1-h 40-km cycling</td>
<td>~8.3</td>
<td>Max intervals (short recovery)</td>
<td>6 sessions over 4 wk</td>
<td>8 M cyclists</td>
<td>Lindsay et al. (1996)</td>
</tr>
<tr>
<td>1-h 40-km cycling</td>
<td>4.6 at 2 wk; 6.6 at 4 wk&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Max intervals (short recovery)</td>
<td>2.wk&lt;sup&gt;−1&lt;/sup&gt; for 4 wk</td>
<td>8+11 M cyclists</td>
<td>Laursen et al. (2002c)</td>
</tr>
<tr>
<td>1-h 40-km cycling</td>
<td>3.2 at 2 wk; 6.2 at 4 wk&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Max intervals (long recovery)</td>
<td>2.wk&lt;sup&gt;−1&lt;/sup&gt; for 4 wk</td>
<td>8+11 M cyclists</td>
<td>Laursen et al. (2002c)</td>
</tr>
<tr>
<td>1-h 40-km cycling</td>
<td>2.7 at 2 wk; 5.3 at 4 wk&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Supramax intervals</td>
<td>2.wk&lt;sup&gt;−1&lt;/sup&gt; for 4 wk</td>
<td>8+11 M cyclists</td>
<td>Laursen et al. (2002c)</td>
</tr>
<tr>
<td>Cycling at ventilatory threshold</td>
<td></td>
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<tr>
<td>1-h 40-km cycling</td>
<td>~4.6</td>
<td>Max intervals (short recovery)</td>
<td>6 sessions over 4 wk</td>
<td>6 M cyclists</td>
<td>Weston et al. (1997)</td>
</tr>
<tr>
<td>18-min 5-km running</td>
<td>0.3 at 6 wk; 4.0 at 9 wk</td>
<td>Explosive sport-specific movements</td>
<td>9 wk</td>
<td>12+10 M elite runners</td>
<td>Paavolainen et al. (1991)</td>
</tr>
<tr>
<td>1-h 40-km cycling</td>
<td>3.4</td>
<td>Supramax intervals</td>
<td>2.wk&lt;sup&gt;−1&lt;/sup&gt; for 3 wk</td>
<td>7 M cyclists</td>
<td>Stepto et al. (1999)</td>
</tr>
<tr>
<td>1-h 40-km cycling</td>
<td>3.0</td>
<td>Max intervals</td>
<td>2.wk&lt;sup&gt;−1&lt;/sup&gt; for 3 wk</td>
<td>12 M cyclists</td>
<td>Stepto et al. (1999)</td>
</tr>
<tr>
<td>10-km running</td>
<td>3.0</td>
<td>Max intervals</td>
<td>3.wk&lt;sup&gt;−1&lt;/sup&gt; for 8 wk</td>
<td>7 M runners</td>
<td>Acevedo and Goldfarb (1989)</td>
</tr>
<tr>
<td>1-h cycling</td>
<td>1.0 at 4 wk; 2.9 at 9 wk</td>
<td>Explosive weights</td>
<td>9 wk</td>
<td>6+8 M cyclists</td>
<td>Bastiaans et al. (2001)</td>
</tr>
<tr>
<td>Cycling at D&lt;sub&gt;max&lt;/sub&gt; lactate</td>
<td>2.6</td>
<td>Usual weights</td>
<td>2.wk&lt;sup&gt;−1&lt;/sup&gt; for 12 wk</td>
<td>14+7 F cyclists</td>
<td>Bishop et al. (1999)</td>
</tr>
<tr>
<td>20-min running to exhaustion</td>
<td>~1.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Max intervals</td>
<td>3.wk&lt;sup&gt;−1&lt;/sup&gt; for 8 wk</td>
<td>7 M runners</td>
<td>Acevedo and Goldfarb (1989)</td>
</tr>
<tr>
<td>1-h cycling</td>
<td>0.6 at 6 wk; 1.8 at 12 wk</td>
<td>Usual weights</td>
<td>2.wk&lt;sup&gt;−1&lt;/sup&gt; for 12 wk</td>
<td>14+7 F cyclists</td>
<td>Bishop et al. (1999)</td>
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<tr>
<td><strong>Maximal Tests</strong></td>
<td></td>
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<tr>
<td>5-min skiing to exhaustion</td>
<td>~5.2&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Explosive sport-specific movements</td>
<td>3.wk&lt;sup&gt;−1&lt;/sup&gt; for 9 wk</td>
<td>10+9 M cross-country skiers</td>
<td>Osteras et al. (2002)</td>
</tr>
<tr>
<td>5-min skiing to exhaustion</td>
<td>~5.1&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Explosive sport-specific movements</td>
<td>3.wk&lt;sup&gt;−1&lt;/sup&gt; for 9 wk</td>
<td>8+7 F cross-country skiers</td>
<td>Hoff et al. (1999)</td>
</tr>
<tr>
<td>10-min 3-km running</td>
<td>2.8%</td>
<td>Max intervals</td>
<td>2.wk&lt;sup&gt;−1&lt;/sup&gt; for 4 wk</td>
<td>5 M runners</td>
<td>Smith et al. (1999)</td>
</tr>
<tr>
<td>7-min skiing to exhaustion</td>
<td>~1.9&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Explosive sport-specific movements</td>
<td>3.wk&lt;sup&gt;−1&lt;/sup&gt; for 8 wk</td>
<td>9+10 M cross-country skiers</td>
<td>Hoff et al. (2002)</td>
</tr>
<tr>
<td>10-min 3-km running</td>
<td>1.2</td>
<td>Plyometrics</td>
<td>2.3-wk&lt;sup&gt;−1&lt;/sup&gt; for 6 wk</td>
<td>8+9 M runners</td>
<td>Spurrs et al. (2003)</td>
</tr>
<tr>
<td><strong>Supramaximal Tests</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30-s cycling</td>
<td>10 at 4 wk; 11 at 9 wk</td>
<td>Explosive weights</td>
<td>9 wk</td>
<td>6+8 M cyclists</td>
<td>Bastiaans et al. (2001)</td>
</tr>
<tr>
<td>45-s cycling</td>
<td>4.6</td>
<td>Supramax intervals</td>
<td>2.wk&lt;sup&gt;−1&lt;/sup&gt; for 3 wk</td>
<td>7 M cyclists</td>
<td>Stepto et al. (1999)</td>
</tr>
<tr>
<td>30-s cycling</td>
<td>3.0</td>
<td>Supramax intervals</td>
<td>2.wk&lt;sup&gt;−1&lt;/sup&gt; for 4 wk</td>
<td>10+7 M cyclists</td>
<td>Creer et al. (2004)</td>
</tr>
<tr>
<td>30-s to 2-min 50- to 200- m swimming</td>
<td>~3.0&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Sport-specific resistance</td>
<td>3.wk&lt;sup&gt;−1&lt;/sup&gt; for 10 wk</td>
<td>11+11 M &amp; F swimmers</td>
<td>Toussaint and Vervoorn (1990)</td>
</tr>
<tr>
<td>45-s cycling</td>
<td>0.4</td>
<td>Max intervals</td>
<td>2.wk&lt;sup&gt;−1&lt;/sup&gt; for 3 wk</td>
<td>12 M cyclists</td>
<td>Stepto et al. (1999)</td>
</tr>
</tbody>
</table>

Supramax, supralaxal; max, maximal; submax, submaximal; M, male; F, female; VO<sub>2</sub>, oxygen consumption. Wk, weeks.

<sup>a</sup>Numbers are experimental + control.

<sup>b</sup>The value of 12% in the paper appears to be an unrealistic increase (should probably be ~5.3%).

<sup>c</sup>These changes in performance time on the Cateye ergometer need to be inflated by an unknown factor (perhaps 1.5x) to convert them to changes in mean power.

<sup>d</sup>Estimated from 51% increase in time to exhaustion using methods of Hopkins et al. (2001).

<sup*e</sup>Estimated from 50% increase in time to exhaustion using methods of Hopkins et al. (2001).

<sup>f</sup>Estimated from a 0.8- 1.1% decrease in swim time using methods of Hopkins et al. (2001).
Summary

The physiological and performance effects of HIT, particularly in untrained individuals has been well researched. Part A of the literature review sought to provide some physiological rationale and more specifically, some key peripheral and central adaptations responsible for the performance improvements often demonstrated following a period of HIT. As discussed, one of the key peripheral adaptations associated with HIT is the improvement in $\beta_m$. This adaptation occurs in response to the repeated stress on the bodies’ physiological systems during HIT. Given the main purpose of this thesis, Part A of the review was largely focussed on the performance effects in already trained athletes, with minimal exploration of the effects seen in untrained populations. Subsequently, the implementation of HIT in already trained individuals is an area that requires further investigation and still poses many questions. For example there is evidence to suggest that many different protocols of HIT can elicit significant performance improvements (Table 2), however, further research into the optimisation of both HIT itself (repetitions, intensity, duration) and the integration of HIT into the training plan (time of season) is warranted.
Literature Review:

**PART B** - The effect of NaHCO₃ supplementation on physiology and performance
Background

Sodium bicarbonate has been investigated over the past century in an attempt to determine its potential as an ergogenic supplement in the pursuit of athletic success (Carr, Hopkins, & Gore, 2011a; McNaughton, Siegler, & Midgley, 2008). In general, performance enhancements during high-intensity exercise of ~1-7 min have commonly been demonstrated (Gao, Costill, Horswill, & Park, 1988; Goldfinch, McNaughton, & Davies, 1988; McNaughton & Cedaro, 1991), often associated with pre-exercise increases in \([\text{HCO}_3^-]\) and pH (Matson & Tran, 1993). Matson & Tran conducted an early meta-analysis of the performance benefits associated with sodium bicarbonate ingestion and reported an average standardised effect size of 0.44 (Matson & Tran, 1993) which is regarded as small (Cohen, 1988). A more recent meta-analysis (Carr, et al., 2011a), examined 38 studies on \(\text{NaHCO}_3\) loading and exercise performance and suggested a dose of 0.3-0.5 g kg\(^{-1}\) BM could elicit mean power improvements of 1.7% (90% confidence limits ±2.0%) in high-intensity exercise of short duration, which was suggested to be a moderate effect (Cohen, 1988). According to the meta-analysis, the effectiveness of sodium bicarbonate was enhanced with an increased dose when performing repeated sprints, and there was a reduction in benefit with non-athletes and when increasing the test duration to 10 min or longer (Carr, et al., 2011a). Despite the wide use and evidence for the potential use of \(\text{NaHCO}_3\) as a supplement, it may not always be suitable due to the possible side effects or interfering with competition preparation. Furthermore, there are still many unanswered questions about the optimal dosing/loading protocols as well as the exact mechanisms by which it exerts any ergogenic benefit.
Physiology of NaHCO₃ supplementation

It has been hypothesised that increasing the body’s buffering capacity through increasing the amount of circulating HCO₃⁻ would protect against acidosis and thereby delay the onset of muscle fatigue during exercise (Matson & Tran, 1993). It is this hypothesis that is the basis of the use of exogenous NaHCO₃ as a method of increasing buffering capacity, delaying the onset of fatigue, and increasing exercise performance.

During exercise, when intracellular buffering capacity is exceeded, both H⁺ and lactate diffuse into the blood, causing a drop in the extracellular pH. This stimulates extracellular buffering mechanisms of which the [HCO₃⁻] is one of the most effective, accounting for 15-18% of total buffer capacity (Sahlin, 1978). There is increasing evidence to suggest that the extracellular pH and HCO₃⁻ concentration plays a key role in determining the intracellular pH even though it has been shown that the sarcolemma is relatively impermeable to HCO₃⁻ (Costill, et al., 1984; Hood, Schubert, Keller, & Muller, 1988; Mainwood & Renaud, 1985). Several investigators have observed a positive relationship between the rate of efflux of H⁺ and lactate from the cell and extracellular pH and [HCO₃⁻] (Hirche, Hombach, Langohr, Wacker, & Busse, 1975; Jones, Sutton, Taylor, & Toews, 1977; Mainwood & Worsley-Brown, 1975). These studies have confirmed that increased extracellular pH and higher [HCO₃⁻] raise H⁺ and lactate efflux from active muscles (Bouissou, Defer, Guezennec, Estrade, & Serrurier, 1988; Hirche, et al., 1975; Hood, et al., 1988). This is due to an increase in the activity of the lactate/H⁺ co-transporter, which becomes more active as the intra/extracellular H⁺ gradient increases, during contraction as well as during recovery (Roth, 1991). The precise mechanism by which increasing acidosis causes muscular fatigue is not clear, but it has been suggested that the accumulation of H⁺ may directly hinder the contractile process by inhibiting the release of Ca²⁺ from the
sarcoplasmic reticulum (Nakamura & Schwartz, 1970), by impairing the propagation of neural impulses (Hultman, Del Canale, & Sjoholm, 1985), reducing glycolytic flux (Newsholme & Start, 1973) and myofilament interaction and thereby force generation (Chase & Kushmerick, 1988). It is argued that these factors may ultimately contribute to muscular fatigue (Spriet, Lindinger, Heigenhauser, & Jones, 1986).

During high-intensity exercise, the onset of intracellular acidosis is delayed via intra- and extracellular buffering mechanisms. Intracellular buffers are the first and most rapid line of buffering defence; however, as high-intensity exercise continues, H⁺ produced by anaerobic glycolysis soon exceed intracellular buffering capacity. The second line of defence against intracellular acidosis is extracellular buffering that involves the transport of H⁺ out of the muscle cell. Extracellular buffers include haemoglobin, plasma proteins and HCO₃⁻. Haemoglobin and plasma proteins play a minor role in extracellular buffering, whereas HCO₃⁻ is one of the key constituents (Beaver, Wasserman, & Whipp, 1986; Parkhouse & McKenzie, 1984; Sahlin, 1978). Although HCO₃⁻ provides a major buffering mechanism during high-intensity exercise, the natural stores within the body are quite small and are soon exceeded during high-intensity exercise.

Many researchers have shown that the ingestion of NaHCO₃ results in a significant elevation of the pH and [HCO₃⁻] in the blood (Carr, et al., 2011a; Matson & Tran, 1993). However, there appears to be a threshold elevation in [HCO₃⁻] and/or pH required (6 mmolL⁻¹ and 0.05 respectively) before ergogenic potential is evident (Bishop & Claudius, 2005; McNaughton & Cedaro, 1991; Van Montfoort, Van Dieren, Hopkins, & Shearman, 2004; Wilkes, Gledhill, & Smyth, 1983). It has been hypothesised that the effect of such induced alkalosis would be to allow more lactic acid to be produced as more H⁺ are buffered. More lactic acid means more energy produced from anaerobic glycolysis, thus performance at higher intensities should be enhanced (Matson & Tran, 1993). Post-
exercise blood lactate values can be 1-2 mmol L\(^{-1}\) higher after NaHCO\(_3\) ingestion compared with placebo or control trials (McNaughton, Dalton, & Palmer, 1999; Wilkes, et al., 1983).

**NaHCO\(_3\) and Exercise Performance**

Numerous studies have examined the exogenous administration of alkalotic agents in an attempt to enhance human performance. However, the results of previous studies show considerable discrepancies. Studies both support and refute the efficacy of alkalotic agents as a way to enhance sport performance. It has been suggested that sports that are high-intensity in nature and involve fast motor-unit activity and large muscle mass recruitment (athletics events, cycling, rowing, swimming and many team sports) stand to gain the most benefit from NaHCO\(_3\) loading (Requena, Zabalza, Padial, & Feriche, 2005). Furthermore, it has also been suggested that NaHCO\(_3\) loading is the most successful in sporting bouts lasting from 1-7 min, when compared to events of shorter or longer durations (Linderman & Gosselink, 1994).

Table 3 outlines some of the key methods and protocols and their effect on sport performance from 44 studies examining acute NaHCO\(_3\) loading. Some of the studies included multiple exercise bouts, giving a total 86 bouts to report. From the 86 bouts evaluated, exactly half (43) produced a statistically significant performance result (\(P < 0.05\)). Table 3 also highlights the studies that used trained or un-trained subjects and reports whether or not each study used a double blind design. The table indicates a few inconsistencies in the literature with regard to the methodology used in NaHCO\(_3\) research. The research is difficult to interpret given the differences in doses, exercise protocols, training level of subjects and placebo substances (or lack of) implemented in the studies. Different aspects of these performance improvements in relation to loading methodologies
and effect on blood-gas variables as well as the associated side-effects will be explored further in subsequent sections of this review.
Table 3 – Summary of the acute NaHCO₃ literature with regard to methods and protocols used and the effect on exercise performance.

<table>
<thead>
<tr>
<th>Authors</th>
<th>No. of subjects</th>
<th>Trained athletes?</th>
<th>Double blind?</th>
<th>Placebo substance</th>
<th>Exercise mode</th>
<th>Exercise protocol</th>
<th>Dosage (g·kg⁻¹ BM)</th>
<th>Exercise bout</th>
<th>Effect on performance?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Artioli et al. (2007)</td>
<td>14</td>
<td>Yes</td>
<td>Yes</td>
<td>CaCO₃</td>
<td>Arm Cycling</td>
<td>30s Wingate test</td>
<td>0.3</td>
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<td>Artioli et al. (2007)</td>
<td>9</td>
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<td>Yes</td>
<td>CaCO₃</td>
<td>Judo</td>
<td>Total judo throws in 15s</td>
<td>0.3</td>
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<td>↑</td>
</tr>
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<td>Total judo throws in 30s</td>
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<td>1</td>
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<td>Total judo throws in 30s</td>
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<td>Total judo throws in 30s</td>
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<td>3</td>
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</tr>
<tr>
<td>Aschenbach et al. (2000)</td>
<td>8</td>
<td>Yes</td>
<td>Yes</td>
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? - Authors have not specified
↑ - Significantly better (P < 0.05)
↔ - No difference (P ≥ 0.05)
↓ - Significantly worse (P < 0.05)

% VO₂max – Intensity of exercise in relation to power/velocity achieved at maximum oxygen consumption identified in an incremental test
TTE – Time to exhaustion test
PPO – Peak power output
1RM – One repetition maximum

All studies implemented a cross-over design, therefore No. of subjects refers to the number of subjects that completed the cross-over.
From the literature analysed in Table 3, 56% of the studies that did not match sodium content in the placebo substance, had a significantly positive result on performance when using NaHCO₃. In contrast, when the sodium content was matched in the placebo group (usually with NaCl), 49% of the studies showed a positive result for the NaHCO₃ group. Many previous studies have used NaCl as a placebo or control substance to match the Na⁺ content in NaHCO₃, although very few have considered the potential effects of Na⁺ by providing an alternate control (Aschenbach, et al., 2000; Kozak-Collins, et al., 1994; Mitchell, et al., 1990). Ingestion of Na⁺ is thought to cause increases in intravascular volume (Kozak-Collins, et al., 1994; Saltin, 1964), and subsequently, enhance exercise performance (Saltin, 1964; Sawka, Convertino, Eichner, Schnieder, & Young, 2000).

Mitchell et al. (1990) observed that intravenous infusion of both NaHCO₃ and NaCl significantly improved cycling endurance in a 30-min time to fatigue test when compared to a control (no infusion), despite the fact that only NaHCO₃ prevented acidosis during exercise (Mitchell, et al., 1990). It was concluded that this result was likely to be caused by an increased intravascular volume from the Na⁺ infusion which would result in better perfusion of exercising skeletal muscle. Hinchcliff et al. (1993) reported that oral administration of both NaHCO₃ and NaCl increased peak speed and performance time in equines during progressive treadmill running, despite the fact that blood pH was significantly more acidic in the NaCl trial (Hinchcliff, McKeever, Muir, & Sams, 1993). Similarly, Kozak-Collins et al. found no significant difference between equal oral doses of NaHCO₃ and NaCl with respect to the number of bouts completed during exhaustive leg ergometry, notwithstanding acid-base changes during the protocol (Kozak-Collins, et al., 1994). These findings suggest that NaCl may not be an adequate physiologically inert substance to use as a placebo, possibly because of the Na⁺ concentration. However, the lack of a control trial using another substance makes interpretation of these results difficult.
**NaHCO₃ dosing and loading regimens**

Historically, an acute dose of sodium bicarbonate of 0.2-0.4 grams per kilogram of body mass (g·kg⁻¹ BM) taken 1-3 h prior to exercise (Katz, et al., 1984; Tiryaki & Attebom, 1995) has been implemented. Higher doses taken over a shorter time-frame can often result in gastro-intestinal upset (Burke & Pyne, 2007; Carr, Slater, Gore, Dawson, & Burke, 2011b). Serial doses of 0.3-0.6 g·kg⁻¹ BM per day over several days (Douroudos, et al., 2006; McNaughton & Thompson, 2001), have also been tested as a loading strategy. A loading protocol that is less common is the chronic application of NaHCO₃ loading before training over several weeks (Edge, et al., 2006b). However, experimental research has not systematically investigated the effect of variations in the ingestion protocol (e.g. the comparison of different loading techniques) on the magnitude or duration of induced blood alkalosis and/or performance in trained athletes.

**Acute NaHCO₃ loading**

The acute method of NaHCO₃ loading is the most extensively researched and practiced form of loading, however, there remain inconsistencies regarding the optimal dosage regimens and the administration protocol of NaHCO₃, including the form (capsules or solution), ingestion period (from the beginning to conclusion of ingestion and the timing of the subsequent exercise task) and the volume of co-ingested fluid (Burke & Pyne, 2007; Matson & Tran, 1993; McNaughton, Siegler, & Midgley, 2008).

The dosage of acute NaHCO₃ loading used most commonly is 0.3 g·kg⁻¹ BM (Burke & Pyne, 2007; Matson & Tran, 1993); and this has been derived from several dose-response studies (Douroudos, et al., 2006; Horswill, et al., 1988; McNaughton, 1992). Commercially
available capsules (Sodibic, Aspen Pharmacare, Australia) typically consist of \( \sim 0.8 \) g of bicarbonate, which translates to a total of \( \sim 25 \) capsules required for a 70 kg athlete. This volume of capsules, powder and water are quite large, and many athletes experience discomfort taking in this amount of NaHCO\(_3\). For this reason, the capsules and drink are often consumed in 3 or 4 smaller doses, typically over a 60-90 min time-frame (Carr, et al., 2011b). The meta-analyses conducted by both Carr et al. (2011a) and Matson & Tran (1993) revealed that the majority of NaHCO\(_3\) studies investigated used an acute dose of 0.3 g \(\text{kg}^{-1}\) BM. The Matson & Tran meta-analysis found that in the 29 studies examined, the method of ingestion was equally divided between capsules and solutions, 43% each, with the remaining 14% using either intravenous injection or not stated.

In the meta-analysis by Matson & Tran (1993), the time interval for ingestion ranged from all at once to over a 3-hour period, while the time between completion of ingestion and initiation of exercise ranged from immediately prior to exercise to 3 hours before. In 60% of studies, ingestion was completed between 1 and 2 hours before the exercise test. There was no difference in performance outcomes between capsular and solution administration methods (Matson & Tran, 1993). To experience the full ergogenic effect of NaHCO\(_3\) loading, it has been suggested that the exercise event should take place at the time of peak plasma alkalosis (Carr, et al., 2011a). A recent study by Price and Singh (2008) investigated the time course changes in blood pH and HCO\(_3^-\) concentration following acute-loading of NaHCO\(_3\). Peak values for blood pH and HCO\(_3^-\) concentration were achieved 60-90 min after ingestion (Price & Singh, 2008). However, the authors did not provide any information on the exact loading method (e.g., how many doses the supplement was taken in). A more recent study by Carr et al. (2011b), also evaluated the differences in HCO\(_3^-\) and pH following eight different methods of acute NaHCO\(_3\) loading (Carr, et al.,
The authors suggested that peak alkalosis can be expected ~120 min after commencing ingestion of various protocols involving NaHCO₃ at 0.3 g kg⁻¹ BM.

The occurrence of gastro-intestinal side-effects has been commonly reported in NaHCO₃ literature (Carr, et al., 2011b). While the gastro-intestinal side-effects themselves will be addressed in more detail in a later section of this review, it has become well accepted that the preferential method for acute NaHCO₃ loading is to ingest the supplement in capsule form as to minimise these associated gastro-intestinal side effects (Carr, et al., 2011b). While intravenous delivery of NaHCO₃ may also prevent side-effects, it is not a practical, nor legal method of supplementation before competition. There is some evidence to suggest that co-ingestion with a high-carbohydrate meal and ingestion periods of 30-60 min may further prevent any gastro-intestinal symptoms (Carr, et al., 2011b). Therefore, 0.3 g kg⁻¹ BM of NaHCO₃ taken in capsule form, in split doses over ~60 min with a co-ingested snack (commencing ~120 min before exercise) seems to be ‘best practise’ based on the current research. However, an alternative method to acute loading – serial loading, may provide an equally effective protocol for NaHCO₃ loading while reducing the distraction involved on competition or race day.

**Serial NaHCO₃ loading**

A variation of the acute loading protocol is to ingest NaHCO₃ in split doses over a few days leading up to competition. The implementation of serial loading may help to avoid some of the negative side-effects associated with acute NaHCO₃ loading. Furthermore, serial loading also helps to eliminate the distraction of having to ingest a large amount of capsules or fluid in the hours leading up to a competition. Typically, a slightly larger dose than the acute dose is used (~0.3-0.5 g kg⁻¹ BM) split into 3 or 4 smaller doses spread over
the day for 3 to 5 days before the competition or event (Burke & Pyne, 2007; McNaughton, Backx, Palmer, & Strange, 1999a; McNaughton, et al., 1997). There have been a few scientific studies that have investigated the application of serial loading, along with a number of anecdotal reports of athletes trialling this technique in an uncontrolled setting in the field (McNaughton, et al., 1999a; McNaughton, et al., 1997). Studies have shown that several days of split NaHCO₃ doses increase the blood buffer levels, which can remain elevated for at least 24 hours after the last dose (Burke & Pyne, 2007). One study compared a medium and a high serial dose of NaHCO₃ (0.3 and 0.5 g kg⁻¹ BM, respectively) to a control substance on performance and acid-base variables in 24 men after 5 days of supplementation (Douroudos, et al., 2006). The authors reported that HCO₃⁻ increased proportionately to the dosage level. Supplementation increased mean power (Watts·kg⁻¹) in the medium and high dose trials compared to the control, with the high dose trial being more effective than the medium dose trial (control = 6.69 ± 0.6, high = 7.72 ± 0.9 medium = 7.36 ± 0.7 Watts·kg⁻¹). NaHCO₃ ingestion resulted in higher pH and HCO₃⁻ following exercise, when compared to the control trial, with no difference between medium and high-dose trials ($P > 0.05$).

Given serial-loading has been demonstrated to improve performance on the following day after NaHCO₃ loading has ceased (McNaughton, 1992a), it may be a preferred loading method for athletes involved in competitions that require maximal performance in a heat or semi-final before performing in the final event on the subsequent day. The serial loading approach could be a much more practical solution to acute NaHCO₃ loading assuming it has the same performance benefits. To our knowledge, there have not been any studies that have compared serial and acute loading using a placebo-controlled cross-over design in well-trained athletes.
Chronic NaHCO₃ loading

Recent research has suggested that chronic exposure to NaHCO₃ supplementation during training (not competition) may be beneficial to training adaptation, and subsequently, performance (Edge, et al., 2006b). Edge et al., (2006b) suggested that reducing H⁺ accumulation during training (via pre-training ingestion of NaHCO₃), may lead to further improvements in mitochondrial function and therefore, exercise performance. Subjects were matched on their lactate threshold and were randomly placed into the NaHCO₃ or placebo groups, and performed 8 weeks (3 days per week) of 6-12 x 2-min cycle intervals at 140-170% of their lactate threshold, ingesting NaHCO₃ or a placebo before each training session. The study found a significantly greater improvement in lactate threshold and time to fatigue during a constant load cycling test after HIT in those supplementing with NaHCO₃ compared with a placebo group (164% vs 123%, P < 0.05). While this was a novel finding, the study investigated only moderately-trained participants and the already well-adapted lactate threshold of highly-trained athletes may negate the likelihood of a similar response in such a population. Although there is evidence for benefits from chronic supplementation, to our knowledge, the Edge et al. (2006b) study is the only investigation to date, that looks at chronic NaHCO₃ ingestion in combination with a period of exercise training in humans. Bishop et al. (2010) found significant improvements in mitochondrial mass and mitochondrial respiration in the soleus muscle of male Wistar rats after a period of five weeks of HIT combined with NaHCO₃ supplementation. The improvement in mitochondrial function in the rats also translated to a significant improvement in a time to fatigue test. Whether these types of responses could occur in humans, or more specifically - highly-trained athletes, remains unknown. Burke & Pyne (2009) posed some important questions related to chronic loading – they questioned whether this method would allow athletes to train harder and therefore obtain greater training adaptations, or would athletes
in fact achieve the same level of adaptation with less training. Further investigation into this method is clearly warranted.

**Effects on blood variables**

Previous research and reviews have reported that ingestion of NaHCO3 results in a significant elevation of pH and HCO3\(^{-}\) in the blood (Carr, et al., 2011a; Carr, et al., 2011b; Linderman & Gosselink, 1994; Matson & Tran, 1993). The time-course of acid-base change after specific investigation protocols has also been investigated previously (Carr, et al., 2011b; Price, et al., 2003; Renfree, 2007), with the majority of research suggesting that both HCO3\(^{-}\) and pH levels in the blood peak between 60-150 min post-ingestion of ~ 0.3 g kg\(^{-1}\) BM doses. Figure 5 displays the blood-gas data obtained by Carr et al. (2011b). The results show that, regardless of the loading protocol used, the peak alkalosis readings occurred ~90 min after NaHCO3 ingestion.

Table 4 highlights a number of studies that have reported changes in blood gas variables (pH and HCO3\(^{-}\)) following acute NaHCO3 loading. As reported in the table, when grouping studies by dose, the higher the dose, the greater the mean change in both blood-gas variables. However, the two studies that used a 0.4 g kg\(^{-1}\) BM dose reported blood-gas changes that were similar to those using a 0.3 g kg\(^{-1}\) BM dose. In the 24 studies that used doses of ≥ 0.3 g kg\(^{-1}\) BM there was an average shift of 0.07 units and 5.31 mmol/L for pH and HCO3\(^{-}\), respectively.
Figure 5 - The absolute scores for bicarbonate concentration (HCO₃⁻) (a) and pH (b) after nine different supplement loading protocols. The time zero is the time that ingestion of the bicarbonate or placebo commenced. All doses of NaHCO₃ were at a dose of 0.3 g·kg⁻¹·BM. The legend indicates the nine different protocols, as described in Carr et al. (2011b).
Table 4 – The post-ingestion changes in blood pH and HCO$_3^-$ following acute NaHCO$_3$ loading. The table is divided into three sections, with studies that have used doses of $\leq 0.2$, 0.2-0.3 and $\geq 0.3$ g kg$^{-1}$ BM doses of NaHCO$_3$, respectively.

<table>
<thead>
<tr>
<th>Dose (g kg$^{-1}$ BM)</th>
<th>Post ingestion change</th>
<th>pH</th>
<th>HCO$_3^-$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Doses $\leq 0.2$ g kg$^{-1}$ BM</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Horswill (1988)</td>
<td>0.1</td>
<td>0.02</td>
<td>1</td>
</tr>
<tr>
<td>Inbar (1983)</td>
<td>0.15</td>
<td>0.06</td>
<td></td>
</tr>
<tr>
<td>McKenzie (1986)</td>
<td>0.15</td>
<td>0.06</td>
<td>1.9</td>
</tr>
<tr>
<td><strong>Mean ± SD</strong></td>
<td>0.13 ± 0.03</td>
<td>0.05 ± 0.02</td>
<td>1.45 ± 0.64</td>
</tr>
<tr>
<td><strong>Doses 0.2-0.3 g kg$^{-1}$ BM</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Costill (1984)</td>
<td>0.2</td>
<td>0.05</td>
<td>4.1</td>
</tr>
<tr>
<td>George (1988)</td>
<td>0.2</td>
<td>0.1</td>
<td>5.9</td>
</tr>
<tr>
<td>Horswill (1988)</td>
<td>0.2</td>
<td>0.06</td>
<td>3.2</td>
</tr>
<tr>
<td>Iwaoka (1989)</td>
<td>0.2</td>
<td>0.07</td>
<td>3.5</td>
</tr>
<tr>
<td>Katz (1984)</td>
<td>0.2</td>
<td>0.04</td>
<td></td>
</tr>
<tr>
<td>Kindermann (1977)</td>
<td>0.2</td>
<td>0.06</td>
<td>5.5</td>
</tr>
<tr>
<td>Gao (1988)</td>
<td>0.25</td>
<td></td>
<td>4.5</td>
</tr>
<tr>
<td><strong>Mean ± SD</strong></td>
<td>0.21 ± 0.02</td>
<td>0.06 ± 0.02</td>
<td>4.45 ± 1.08</td>
</tr>
<tr>
<td><strong>Doses $\geq 0.3$ g kg$^{-1}$ BM</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aschenbach (2000)</td>
<td>0.3</td>
<td>0.06</td>
<td>4</td>
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<tr>
<td>Bird (1995)</td>
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<td>0.06</td>
<td></td>
</tr>
<tr>
<td>Bouissou (1988)</td>
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<td>0.08</td>
<td></td>
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<tr>
<td>Bouissou (1989)</td>
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<td>0.08</td>
<td>5.3</td>
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<td></td>
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<td>5</td>
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<td>0.06</td>
<td></td>
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<td>Pruscin (2008)</td>
<td>0.3</td>
<td>0.08</td>
<td>7.5</td>
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<tr>
<td>Robertson (1987)</td>
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<td>0.06</td>
<td>5.4</td>
</tr>
<tr>
<td>Robertson (1987)</td>
<td>0.3</td>
<td>0.08</td>
<td>6</td>
</tr>
<tr>
<td>Robertson (1987)</td>
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<td>0.09</td>
<td></td>
</tr>
<tr>
<td>Robinson (1987)</td>
<td>0.3</td>
<td>0.08</td>
<td></td>
</tr>
<tr>
<td>Rupp (1982)</td>
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<td>0.05</td>
<td></td>
</tr>
<tr>
<td>Stephens (2002)</td>
<td>0.3</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>Sutton (1981)</td>
<td>0.3</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>Wilkes (1983)</td>
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<td>0.09</td>
<td>7.3</td>
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<td>Wijnen (1984)</td>
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<td>0.15</td>
<td></td>
</tr>
<tr>
<td>Bishop (2005)</td>
<td>0.4</td>
<td>0.06</td>
<td>6.6</td>
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<tr>
<td>Goldfinch (1988)</td>
<td>0.4</td>
<td>0.08</td>
<td>3.5</td>
</tr>
<tr>
<td><strong>Mean ± SD</strong></td>
<td>0.31 ± 0.03</td>
<td>0.07 ± 0.02</td>
<td>5.31 ± 1.41</td>
</tr>
</tbody>
</table>
Gastro-intestinal side effects

A disadvantage of NaHCO₃ supplementation is the possibility of gastro-intestinal (GI) upset, resulting in symptoms such as nausea, stomach pain, diarrhoea and vomiting (Burke & Pyne, 2007). This is a serious practical consideration for athletes in a competition setting. The fear or personal experience of GI upset could contribute to the avoidance of sodium bicarbonate usage. While particular symptoms have been recorded and quantified in some studies (Stephens, et al., 2002; Van Montfoort, et al., 2004) a limitation of many NaHCO₃ supplementation trials is the lack of documentation of side effects with particular protocols of ingestion (Matson & Tran, 1993). Knowledge of protocols less likely to induce GI distress may increase athletes’ usage and benefit from NaHCO₃ supplementation.

The symptoms associated with NaHCO₃ loading may be due to an increase in gastric emptying following the ingestion of NaHCO₃ (Oster, Stemmer, Perez, & Vaamonde, 1988). When NaHCO₃ is consumed, there exists the possibility of an increased osmolality of the gastrointestinal tract and water may be shifted from the plasma to the intestine to counteract the hypertonicity (Heigenhauser & Jones, 1991; Linderman & Gosselink, 1994). Moreover, the increase of [HCO₃⁻] in the blood requires a large quantity of water in the intestine to maintain the isotonic solution (Requena, et al., 2005). Some studies have modified the ingestion protocols to help alleviate the problems associated with NaHCO₃ loading by using gelatine capsules, though these present the problem that a large number must be ingested (Burke & Pyne, 2007). Intravenous administration has been used, with the aim of minimizing these gastrointestinal problems (Mitchell, et al., 1990). However, this method of delivery is not always practical, nor is it legal according to the current sports anti-doping policies. As mentioned above, other ways to counteract the negative
side-effects associated with NaHCO₃ loading include split-dose methods such as those implemented in serial loading protocols.

Carr et al. (2011b) found that, among eight different supplement protocols, the lowest incidence of GI distress occurred after the ingestion of NaHCO₃ capsules co-ingested with 7 ml kg⁻¹ BM of fluid and a standardized meal (1.5 g carbohydrate per kg BM). Furthermore, the authors suggested that the co-ingestion of a meal is a more important consideration for NaHCO₃ supplementation than fluid volumes or the capsule or solution form of NaHCO₃ used. The highest incidence of GI side effects occurred 90 min after the commencement of NaHCO₃. The results from their study also showed that when the loading protocol was shortened (e.g. 30 min), subjects were more likely to suffer from more severe GI symptoms. Their results for different loading protocols and the subsequent effect they had on GI symptoms are summarised in Figure 6 (Carr, et al., 2011b).
Figure 6 - The absolute scores for gastro-intestinal symptoms following nine different supplement loading protocols. The time zero is the time that ingestion of the bicarbonate or placebo commenced. All doses of NaHCO₃ were at a dose of 0.3 g kg⁻¹ BM. The legend indicates the nine different protocols, as described in Carr et al. (2011b).
Summary

As highlighted in Part B of this literature review, sodium bicarbonate loading has been well researched over the past decade (McNaughton, Siegler, & Midgley, 2008). Performance enhancements in high-intensity exercise of ~1-7 min have commonly been demonstrated (Carr, et al., 2011a; Gao, et al., 1988; Goldfinch, et al., 1988; Matson & Tran, 1993; McNaughton & Cedaro, 1991), often associated with pre-exercise increases in blood [HCO$_3^-$] and pH (Matson & Tran, 1993).

Historically, an acute dose of NaHCO$_3$ of 0.3 g·kg$^{-1}$ BM taken 1-3 h prior to exercise (Katz, Costill, King, Hargreaves, & Fink, 1984; Tiryaki & Atterbom, 1995) has been the standard protocol (McNaughton & Cedaro, 1992), although gastro-intestinal upset can occur (Burke & Pyne, 2007; Carr, et al., 2011a). Serial doses of ~0.5·g·kg$^{-1}$ BM per day over several days (Douroudos, et al., 2006; McNaughton & Thompson, 2001) has also been tested as a strategy that potentially stimulates blood alkalosis with minimal side effects, with positive results. More recently, there has been some evidence to suggest that altering the pH concentration through NaHCO$_3$ supplementation before performing HIT in a chronic setting, may lead to long-term performance improvements (Edge, et al., 2006b). Despite the extensive research in the area of NaHCO$_3$ supplementation for sports performance, there still remain a few unanswered questions which will be explored further in this thesis.
**Literature Review: Conclusion**

The review of literature has outlined the strategies involved in improving muscle buffering capacity during exercise and the effects that these strategies have on both the physiology and performance in human subjects. More specifically, the review focussed on A) The effect of high-intensity interval training (HIT) on physiology and exercise performance, and; B) The effect of NaHCO$_3$ supplementation on physiology and exercise performance.

The primary purpose of HIT is to repeatedly stress the physiological systems involved during exercise at a level that would not be achievable through continuous training. This form of training is commonly implemented by using maximal or supra-maximal sport specific activity to induce a training response. The benefits associated with HIT can be attributed to both peripheral and central adaptations. As discussed, one of the major benefits associated with HIT is the adaptations associated with enhanced muscle buffering capabilities. The review has covered a range of studies that have investigated the use of HIT in both trained and untrained subjects and delved into the protocols required to improve muscle buffering and therefore, performance in athletes.

Sodium bicarbonate loading in sports performance has been investigated over the past century. When performance has been enhanced through NaHCO$_3$ supplementation, it is often during high-intensity exercise lasting ~1-7 min and is commonly associated with pre-exercise increases in blood bicarbonate concentration [HCO$_3^-$] and pH. As discussed in the literature review, there is a plethora of research describing different NaHCO$_3$ loading protocols/doses and their effect on both physiological markers and performance in athletes. Even though there have been hundreds of publications in the area of NaHCO$_3$ and performance, there still remains unanswered questions, especially in relation to determining the optimal loading protocols that can elicit improvements in performance.
while at the same time, minimizing the gastro-intestinal symptoms associated with its ingestion. Additionally, there remain questions related to the exact physiological mechanisms involved with the ergogenic potential of NaHCO₃ loading.

Given the performance enhancements that can be attributed to improved buffering mechanisms with both HIT and NaHCO₃ loading, an important question was postulated by Edge et al. (2006b). He suggested that reducing H⁺ accumulation during training (via pre-training ingestion of NaHCO₃), may lead to further improvements in mitochondrial function and therefore, endurance exercise performance. Edge et al. (2006b) was the first, and to my knowledge, the only researcher that has looked at the combination of both of these strategies in an athletic setting. The study showed promising results for improved athletic performance when HIT and NaHCO₃ were combined in a chronic setting (8-weeks). More recent research has suggested that reducing H⁺ accumulation during training (via pre-training ingestion of NaHCO₃), may lead to further improvements in mitochondrial function (Bishop, et al., 2010). Therefore, this may partly explain the improvements found by Edge et al. (2006b) as the mitochondria are central to the conversion of energy and production of ATP during exercise. However, the study was performed on recreational subjects, so the application of these results to a highly-trained population remains unknown. As already noted, highly-trained athletes have well-developed buffering systems and mitochondrial function that are far superior to their less-trained counterparts and consequently, it is uncertain if the benefits of HIT in combination with NaHCO₃ loading would be the same in a highly trained population.

Following the research discussed in this review, it seems obvious that there are some questions that remain unanswered and protocols that need clarification. The studies in this
thesis aimed to address some of those questions. More specifically, the possibility of whether the results from the Edge et al. (2006b) study would apply to highly-trained athletes is one of the primary questions that this thesis intended to investigate. In order to answer this question, studies investigating optimal methods of both HIT and NaHCO$_3$ loading in trained subjects were performed. Therefore, the aim of this collection of studies was to add to the knowledge of both HIT and NaHCO$_3$ supplementation and give a better understanding of how these strategies may affect athletes in competitive levels of sporting competition.
Chapters 3, 4, 5 & 6

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Chapter Seven:

Thesis Summary
Thesis Summary

The main aim of this thesis was to determine whether the combination of both HIT and NaHCO$_3$ would contribute to any additional performance benefits in highly-trained athletes. To complete this research, initial investigation of some of the questions related to the optimal application of NaHCO$_3$ supplementation and HIT to highly-trained athletes in both a competition and training setting was undertaken. Studies two and three raised some interesting questions in relation to the potential benefits and mechanisms associated with NaHCO$_3$ loading, which were subsequently investigated in the final study of the thesis.

The first study of the thesis aimed to establish an appropriate protocol for HIT in well-trained rowers. The protocol implemented was taken from similar studies that showed benefits in other sports (cycling, swimming). To evaluate the effectiveness of the HIT protocol, it was compared to a more traditional approach to rowing training using intensities based on the power output at differing blood lactate concentrations (control). The major finding of study one (chapter three) was that HIT significantly improved rowing performance and physiology when compared to the more traditional approach to training. The study used seven HIT sessions to induce significant performance improvements of ~8 s in a 2000 m rowing time-trial, compared to ~2 s improvement in the control trial. It also extended the findings of previous investigations into HIT showing that $\dot{V}O_2$ peak and lactate threshold can be significantly improved using HIT in already well-trained athletes (7.0% and 4.3% respectively). The study was the first to demonstrate that acute (4-weeks) HIT significantly improved performance in already well-trained rowers. The HIT protocol used in study one with well-trained rowers was also used in study three with highly-trained
rowers. While there was no control group for comparison in study three (both groups performed HIT), there was a mean improvement of ~5.6 s in 2000 m time after the 4-week period of HIT. Given the ~8 s improvement in the lesser trained rowers following HIT, the improvement in the highly-trained group was still very impressive, suggesting the HIT protocol is also effective in bringing about improvements in national representative athletes. Furthermore, these results are even more impressive considering in study three, the athletes were in the peak-training phase of their season, leading up to a world championship regatta.

With an effective method of HIT established, the second study (chapter four) compared two different methods of NaHCO₃ loading (acute and serial loading) with the intention that the most effective method would be used in study three where HIT and NaHCO₃ were combined. One of the main purposes for examining the serial loading approach was due to the numerous reports in the literature regarding acute NaHCO₃ loading and the associated gastro-intestinal side-effects (Burke & Pyne, 2007; Carr, et al., 2011b; Matson & Tran, 1993). In order to minimise the side-effects involved in taking a large bolus of NaHCO₃ over a relatively short period of time, the serial loading approach seemed to have merit, coupled with some previous supporting research (McNaughton, et al., 1999a). However, to our knowledge, there were no studies to compare the performance effects of both serial and acute methods in trained athletes. The major finding of study two was that the relatively novel concept of serial NaHCO₃ loading (split doses over three days) was also an effective method for improving 4-min time-trial performance in cyclists, and provided similar benefits to the more traditional acute approach of NaHCO₃ loading. Our results were consistent with previous research that has shown, for short-term high-intensity
exercise, acute NaHCO₃ can improve performance by ~3%, perhaps by providing a greater buffering capacity for the increased H⁺ associated with this type of activity (Carr, et al., 2011a). In contrast, one of the interesting findings from this study was that the serial loading approach resulted in similar performance improvements, in the absence of any changes to the extracellular buffering markers (HCO₃⁻, pH). This result posed a question related to the actual mechanisms behind the benefits associated with NaHCO₃ loading, and more specifically, whether other mechanisms (such as the role of the Na⁺ content in NaHCO₃) may be involved, other than the suggested blood-buffering benefits. This question contributed to the design of study four.

As the first study to compare both acute and serial NaHCO₃ loading protocols on the performance of well-trained cyclists, study two substantially adds to the research literature on NaHCO₃ supplementation. The findings from this study would suggest that serial NaHCO₃ loading may be more beneficial than the acute method in certain circumstances. The main benefit of serial loading is that the supplementation protocol finishes the night before an event or competition. For sports or athletes that require maximal concentration with minimal distractions in the hours preceding their event, serial loading may be the preferred option. However, given there was a small trend towards acute loading producing better performance results when compared to serial loading (not statistically significant), and there were minimal side-effects associated with both protocols, it was decided that acute loading method would be more appropriate for study three.

The findings from studies one and two were used to design study three (chapter five). The study involved 4-weeks of HIT (same protocol as used in study one) in highly-trained
Australian representative rowers. Prior to each HIT session, athletes in the experimental group undertook an acute NaHCO₃ loading protocol (as used in study two). The findings showed that despite improvements in 2000 m rowing performance, 4-min power output and 4mmol L⁻¹ power output in highly-trained rowers during the training period, NaHCO₃ supplementation prior to interval training did not provide any additional benefits to these performance variables (not statistically significant). The findings were irrespective of clearly influenced blood variables before (pH and HCO₃⁻) and after (pH) training sessions in the NaHCO₃ supplemented group. The changes to the blood variables (pH and HCO₃⁻) pre- to post-loading were similar to those seen in study two following acute loading. It had been suggested that altering the pH level prior to HIT may provide beneficial adaptations to the mitochondria, which may, in turn, lead to improved exercise performance (Edge, et al., 2006b). The Edge et al. study was the only other study to our knowledge that had investigated this area of chronic NaHCO₃ loading in humans. While we were successful in altering the pH levels in the blood prior to each training session, we did not find an additional performance benefits at the end of the training period. It is a possibility that we did not find similar findings due to the level of athletes that were used. The athletes in our study were highly-trained compared to the recreationally trained subjects in the Edge et al. study. Thus it is possible that the highly-trained athletes already possessed enhanced mitochondrial function and buffering systems that could not be further improved through the combination of HIT and chronic NaHCO₃ supplementation. Additionally, our study was only 4-weeks in duration, compared to 8-weeks in the Edge et al. study, which may not have been long enough to realise any benefit. Another difference between our study and the Edge et al. study was that we matched the placebo substance for equimolar Na⁺ concentration (using NaCl), while they used a lower dose of NaCl, unmatched for Na⁺. Furthermore, the only other study we are aware of that has combined NaHCO₃ and HIT in
a chronic setting (using rats) also failed to match the Na\(^+\) concentration in the placebo group (using water) in their study (Bishop, et al., 2010). In contrast to our study, both of these studies found significantly improved performance in the NaHCO\(_3\) supplemented group when compared to the placebo group, which again raised questions about the role of Na\(^+\) in exercise performance, and consequently motivated the development of study four. As an entire group the athletes involved in study three reduced their 2000 m rowing time by an average of 5.6 s, suggesting that either the HIT protocol (as used in study one) may be effective for improving performance even in highly-trained athletes, or the possibility that both the NaHCO\(_3\) and the NaCl (placebo) supplementation in conjunction with the HIT provided an ergogenic effect enhancing performance to a similar degree in both groups.

The benefits of NaHCO\(_3\) loading are often attributed to the buffering effects of the HCO\(_3^-\) and not necessarily the role of Na\(^+\) in the ingestion of NaHCO\(_3\). Therefore, given the conflicting results concerning the effects of NaHCO\(_3\) induced alkalosis on exercise performance in both the literature and in studies two and three of this thesis, the aim of study four (chapter six) was to examine the effects of NaHCO\(_3\) (SB) and NaCl (SC) on haematocrit and performance in well-trained cyclists when compared to a placebo substance (D). The results from study two, which showed that serial NaHCO\(_3\) loading was effective in improving performance independent of changes to blood [HCO\(_3^-\)] and pH, raised questions about the mechanisms behind NaHCO\(_3\) loading and also, the validity of matching the Na\(^+\) content in a placebo substance. Study three further supported these questions where we did not find any performance benefit after chronic NaHCO\(_3\) supplementation in combination with HIT. The only two other studies, to our knowledge,
examining the combination of NaHCO₃ and HIT (Bishop, et al., 2010; Edge, et al., 2006b), found a benefit in the NaHCO₃ group; however, unlike the other two studies, we matched the Na⁺ content in our placebo substance. Kozak-Collins et al. (1994) stated that, if grouped according to placebo substance, the studies that used calcium carbonate or a small amount of sodium chloride (NaCl) found a benefit in performance with NaHCO₃ ingestion while studies using a placebo that contained higher Na⁺ content were inconclusive. Many previous studies have used NaCl as a placebo or control substance to match the Na⁺ content in NaHCO₃, although very few have considered the potential effects of Na⁺ by providing an alternate control. Ingestion of Na⁺ is thought to cause changes in intravascular volume which may subsequently, enhance exercise performance (Kozak-Collins, et al., 1994; Mitchell, et al., 1990; Saltin, 1964). However, despite this, the findings from study four suggest that SB loading significantly enhanced mean 2-min cycling performance when compared to both SC and D trials. There were also no significant differences between SC and D for mean 2-min cycling performance or any differences in haematocrit measures between groups at all time-points. Consequently, based on this study, using NaCl (matched for Na⁺) may still be a valid placebo substance; however, given the findings in study two and three, there may be roles other than extracellular buffering associated with NaHCO₃ which are not yet realised and warrant further investigation.

The studies included in this thesis make specific additions to NaHCO₃, HIT and exercise performance research. The HIT protocol used in studies one and three was successful in bringing about significant performance improvements in already well-trained and highly-trained rowers after just four weeks. Furthermore, in light of the findings regarding serial
NaHCO$_3$ loading trialled in study two, coaches and athletes may implement this alternative method of supplementation to avoid some of the complications associated with acute loading before an event. Therefore, this work not only contributes to the knowledge base surrounding NaHCO$_3$ loading and HIT, but it provides a useful contribution to future experimental designs on studies in this area, as well as giving successful practical applications for athletes and coaches to use in the high performance setting.
Limitations

The following limitations of this thesis are acknowledged.

- The thesis aimed to develop methods to improve athletic performance in highly-trained athletes. However, as there is a limited pool of highly-trained athletes to draw from, the sample size is often small in the studies included in this thesis. Adding in lower level athletes to improve sample size would have lowered the ability to apply findings to the target group.

- A limitation of working with highly-trained athletes (specifically in studies one and three), is that athletes involved in the studies were often brought together for short training periods in preparation for national/international competition, and the performance tests used had to comprise a regular part of the prescribed testing for their sport, which could not be altered. Therefore, as researchers, we often had to make accommodations to the constraints involved with these limitations.

- The intervention studies in this thesis assessed training methods over a 4-week training block; whereas the length of an in-season is much greater. If time permitted, we would have preferred to extend the intervention period for our training studies to ~8-weeks.

- The mode of exercise test consisted of both rowing and cycling (two studies for each). Ideally, all studies would have been performed on the same group of athletes from the same sport, using the same exercise tests. However, access to the same group of athletes was limited so in order to gain an understanding of a similar athletic population (in terms of physical demands and characteristics) both cycling and rowing were used. Given the physiological demands of both track-cycling and
rowing are very similar, the thesis alternated between cyclists and rowers with the intention that the results achieved for both modes could be applied to the other population.

- At times the collection of more mechanistic variables in order to gain a greater physiological understanding of the performance results following the training intervention studies would have been preferred. However, because of the calibre of athletes recruited and the many caveats often imposed with investigating such a population it was not possible to employ some more invasive or time consuming methods to obtain this data (e.g muscle biopsies).
Practical Applications

Study 1 - The effects of high-intensity interval training in well-trained rowers

In study one, HIT involved 8 x 2.5 min intervals at 90% of the velocity achieved at \( \dot{VO}_2 \) peak. Recovery intensity between intervals was set at 40% of the velocity achieved at \( \dot{VO}_2 \) peak until a heart rate of \( \leq 70\% \) of maximum was reached. Athletes performed 2 HIT sessions per week for 4 weeks. The practical applications of this training protocol are as follows:

- There was an improvement of ~8 s in 2000 m time-trial performance following 4-weeks of HIT. This performance improvement equates to approximately a 4.5 boat-length improvement in a 2000 m single sculling race compared to a 1 boat length improvement following traditional training.

- HIT was a successful method in improving \( \dot{VO}_2 \) peak when compared to the more traditional training.

Study 2 - The effects of serial and acute NaHCO\(_3\) loading in well-trained cyclists

In study two, the acute loading protocol involved the ingestion of a 0.3 g kg\(^{-1}\) BM dose of NaHCO\(_3\) (capsule form), commencing 90 min prior to their test time (taken in five equal doses spread over a 60 min period). Serial loading involved the ingestion of a 0.4 g kg\(^{-1}\) BM dose of NaHCO\(_3\), to be taken in three equal amounts throughout the day, for a period of three days (completing their last dose at 7pm the night prior to testing). The practical applications of these methods of supplementation are as follows:
- Both acute and serial NaHCO₃ loading improve 4-min maximal cycling performance in well-trained cyclists.
- The performance improvement associated with both acute and serial NaHCO₃ loading when compared to a placebo trial represented a 2.3-3.3% higher average power output.
- The practical application of either NaHCO₃ loading protocol in an event such as a 4000 m individual pursuit in track cycling may provide a significant advantage over opponents.
- Adopting a serial loading approach may allow athletes to concentrate on their event without the associated distractions of acute NaHCO₃ loading on the day of competition.
- Both acute and serial NaHCO₃ loading protocols used in this study produced minimal gastro-intestinal distress/side effects sometimes experienced with NaHCO₃ supplementation.

**Study 3 - The effects of chronic sodium bicarbonate loading and interval training in highly-trained rowers**

The HIT protocol used in study three was the same as the protocol used in study one. The NaHCO₃ supplementation protocol was the same as the acute loading protocol used in study two. Athletes supplemented with NaHCO₃ before each HIT session (2 x a week for 4 weeks). The practical applications for this combined HIT/NaHCO₃ method are as follows:
- There were no additional performance benefits in combining NaHCO₃ supplementation and HIT in highly-trained rowers when compared to a placebo group.

- Independent of which group subjects were in, the current study observed a mean improvement of 5.6 s in 2000 m rowing performance. Even though there was no control group for comparison, this improvement in performance supports the findings from study one which found significant improvements following HIT in well-trained rowers. This would suggest that the HIT protocol used in both studies may be effective in both well-trained and highly-trained populations.

- The improvements are impressive given the highly-trained nature of the athletes in the current study and the peak phase of their training when the study was conducted.

**Study 4 - The effects of NaHCO₃ and NaCl loading on haematocrit and high-intensity cycling performance**

The NaHCO₃ supplementation protocol was the same as the acute loading protocol used in study two - 0.3 g·kg⁻¹ BM dose of NaHCO₃ (capsule form), commencing 90 min prior to their test time (taken in five equal doses spread over a 60 min period). The practical applications from this study are as follows:

- There was a 3.2% improvement in mean 2-min cycling power output following NaHCO₃ loading when compared to a placebo trial in well-trained cyclists.

- Acute NaHCO₃ loading also contributed to a 9.6% improvement in peak power when compared to the placebo.
- The practical application of these results could transfer to any sports where a similar time-frame and intensity is required (e.g. various track cycling, swimming and running events).

- There were no associated benefits to cycling performance following NaCl loading.

**Overall key practical applications**

- Implementing the following HIT protocol over 4-weeks (twice a week) may result in rowing performance improvements in already well-trained athletes:
  
  - 8 x 2.5 min intervals at 90% of $\dot{VO}_2$ peak.
  
  - Recovery intensity at 40% of $\dot{VO}_2$ peak until a heart rate of $\leq70\%$ of maximum or 3 min is reached.

- An acute loading protocol involving the ingestion of a 0.3 g kg$^{-1}$ BM of body mass dose of NaHCO$_3$ (capsule form), commencing 90 min prior to exercise (taken in five equal doses spread over a 60 min period), may result in a performance improvement of $\sim3\%$ in events lasting $\sim4$ min.

- A serial loading protocol involving the ingestion of a 0.4 g kg$^{-1}$ BM body mass dose of NaHCO$_3$, (taken in three equal amounts throughout the day), for a period of three days (completing the last dose at 7pm the night prior to exercise) may result in a performance improvement of $\sim2.5\%$ in events lasting $\sim4$ min.

- The serial NaHCO$_3$ loading approach may be a more appropriate protocol to use in circumstances where athletes want to minimise the distractions involved in the hours leading up to a competition or event.
Future Directions

- Considering study one was the first study (to our knowledge) to show improvements after 4-weeks of HIT in well-trained rowers, additional investigations are necessary to determine the optimal volume and periodisation of HIT into a rowing training program.

- While HIT has been used successfully in many different sports, the protocols implemented often differ slightly with the intensities/durations/repetitions used. It would be advantageous to investigate if other sports (using well-trained athletes) implementing the exact same HIT protocol used in this thesis would benefit from similar performance improvements.

- The exact mechanisms behind performance changes associated with HIT in well-trained athletes are still relatively unknown. A study examining mechanistic changes (e.g muscle buffer capacity) following similar HIT protocols in well-trained rowers is warranted.

- The potential mechanisms contributing to performance enhancement in the absence of substantial blood pH changes following serial NaHCO₃ loading (0.4 g kg⁻¹ BM dose of NaHCO₃, taken in three equal amounts throughout the day, for a period of three days) requires further examination.

- Future research should employ longer periods of chronic NaHCO₃ supplementation (e.g > 8 weeks) in order to determine any effectiveness in highly-trained athletes. Additionally, it would be advantageous to examine whether the use of different performance tests in the same study (e.g time to fatigue test, short duration time-
trial, long duration time trial) exhibit different results and decipher which type of tests are the most sensitive to NaHCO₃ supplementation.

- To our knowledge, there have been no published studies examining whether chronic NaHCO₃ loading during training might enable athletes to train harder and therefore achieve greater levels of training adaptation. A potential benefit from NaHCO₃ loading may only be realised if the training load is not rigorously controlled (as in this thesis), enabling athletes to possibly train harder in the supplemented state. Consequently a study should be performed to ascertain whether chronic loading permits greater training loads and, subsequently, greater performance improvements.

- Further investigation regarding the exact mechanisms behind both Na⁺ and HCO₃⁻ loading for exercise should be conducted.


APPENDICIES

APPENDIX I:

Circulating adiponectin concentration is altered in response to high-intensity interval training

Presented here in a similar manuscript format as under review in:

Journal of Strength and Conditioning Research
Abstract

Adiponectin influences metabolic adaptations that would prove beneficial to endurance athletes and yet to date there is little known about the response of adiponectin concentrations to exercise, and in particular, the response of this hormone to training in an athlete population. This study aimed to determine the response of plasma adiponectin to acute exercise following two different training programs. Seven state level representative rowers [age: 19 ± 1.2 years (mean ± SD), height: 1.77 ± 0.10 m, body mass: 74.0 ± 10.7 kg, \( \dot{V}O_2 \text{peak} 62.1 ± 7.0 \text{mL-kg}^{-1} \cdot \text{min}^{-1} \)] participated in the double-blind, randomized, cross-over investigation. Rowers performed an incremental graded exercise test before and after completing four weeks of high-intensity interval ergometer training and four weeks of traditional ergometer rowing training. Rowers’ body composition was assessed at baseline and following each training program. Significant increases in plasma adiponectin occurred in response to maximal exercise after completion of the high-intensity interval training \((p = 0.016)\) but not following traditional ergometer rowing training \((p = 0.69)\). The high-intensity interval training also resulted in significant increases in mean four min power output \((p = 0.002)\) and \( \dot{V}O_2 \text{peak} \ (p = 0.05) \), as well as a decrease in body fat percentage \((p = 0.022)\). Mean four min power output, \( \dot{V}O_2 \text{peak} \) and body fat percentage were not significantly different following four weeks of traditional ergometer rowing training \((p > 0.05)\). Four weeks of high-intensity interval training is associated with an increase in adiponectin concentration in response to maximal exercise. The potential for changes in adiponectin concentration to reflect positive training adaptations and athlete performance level should be further explored.
Introduction

Human adipose tissue is recognised as having an endocrine function in combination with its role in energy metabolism and storage (5). Adiponectin is one protein secreted by adipose tissue and its targets include the liver and skeletal muscle where, among other functions, it regulates energy metabolism and insulin sensitivity (20, 26). Adiponectin has been shown to activate adenosine monophosphate-activated protein kinase (AMPK) in skeletal muscle (7) and evidence suggests that enhanced activity of AMPK can stimulate increased number and oxidative capacity of mitochondria in the muscle (24). Upregulation of AMPK also promotes fatty acid oxidation through improved oxidative phosphorylation (3, 5, 20), which has the potential to benefit endurance exercise performance by sparing muscle glycogen and blood glucose.

Adiponectin influences metabolic adaptations that would prove beneficial to endurance athletes and yet to date there is little known about the response of adiponectin concentrations to exercise, and in particular, the response of this hormone to training in an athlete population. Immediately post-exercise, adiponectin levels of trained rowers have been reported to increase or decrease depending on performance level of the athlete (17). Rowers selected for national team representation at the Olympic games experienced an increase in adiponectin following a maximal 2000 m row, whereas non-selected rowers, who did not perform as fast, showed a reduction in circulating adiponectin (17). In healthy and active males, cycling at 50% and 60% $\dot{V}O_2_{\text{max}}$ for durations of 60-120 min did not influence post-exercise concentrations of adiponectin (8, 19, 25), while running at ~ 80% $\dot{V}O_2_{\text{max}}$ for 30 min was associated with an increase (19). Exercise intensity and training status may influence plasma adiponectin in response to exercise. In an athlete population
with already well developed mitochondrial biogenesis it remains unknown if acute exercise intensity is an important influencer of adiponectin concentration, or if a period of high intensity training influences the response to an acute bout of exercise.

In combination with the effects on substrate metabolism, adiponectin concentration has been inversely related to body mass (8, 9, 15). Resting levels of adiponectin have been shown to increase in healthy females following 10 weeks of exercise training at 70% \( \dot{V}O_2 \text{max} \) (21) and in obese populations exercise has been shown to increase adiponectin mRNA expression in adipose tissue, while a combination of diet and exercise increased circulating levels of adiponectin (2). Jürimäe and colleagues (17) reported that adiponectin concentration at rest does not change over six months of high volume, low intensity rowing training, although these athletes did not experience a significant change in fat mass across the training period either. The relationship between changes in adiponectin concentration and changes in body fat in athletes requires further investigation.

Given that adiponectin influences metabolic adaptations that would prove beneficial to endurance athletes, we investigated the response of adiponectin to four weeks of aerobic training and four weeks of interval training in trained rowers. Training that induces higher levels of adiponectin may be associated with adaptations also associated with successful endurance performance.
Methods

Subjects

Participants were junior state and national level rowers from the Tasmanian Rowing Team (male n=5, female n=2) [age: 19.0 ± 1.2 years (mean ± SD), height: 1.77 ± 0.10 m, body mass: 74.0 ± 10.7 kg, \(\dot{VO}_2\) \text{peak} 62.1 ± 7.0 mL·kg\(^{-1}\)·min\(^{-1}\), body fat: 17.1 ± 6.3%]. On entry to the study each rower completed a physical activity readiness questionnaire, a coronary artery disease risk factors questionnaire, and provided their informed consent. The study was approved by the Institutional Human Research Ethics Committee.

Experimental overview

The study was a randomised, cross-over design. Rowers completed a baseline maximal graded exercise test and were then randomly allocated into one of two groups to complete two ergometer training sessions per week over a four-week period before re-testing. After re-testing the cross-over occurred and each rower completed the alternative ergometer training program. Each rower completed four-weeks of high-intensity interval training and four-weeks of traditional endurance training. The investigators were blinded to the training program (either high-intensity interval or traditional rowing ergometer training program) each rower was completing. The rowers were informed that there was no reported advantage of one training program over the other and that the study was investigating two types of ergometer training protocols. At week 0, week 5 and week 10 rowers completed the graded exercise test. Venous blood was collected pre- and post-graded exercise test and body composition was assessed pre- and post- training intervention. Rowers used the same ergometer during each test session and testing was conducted at the same time of day.
**Graded exercise test**

The maximal exercise test was conducted to determine each rower’s peak power, lactate threshold and relative peak oxygen consumption (\(\dot{\text{VO}_2\text{peak}}\)) according to the methods of Gore (11). Rowers arrived at the exercise lab for the test having fasted for two hours. Each rower was also instructed not to exercise in the 12 hours leading up to their test. All rowers were familiar with the equipment (Concept IIc rowing ergometer, Concept 2 Inc., Vermont, US) and protocols that were used. The protocol involved completing six four-min workloads relative to their most recent 2 km ergometer trial result before an all-out effort for the final four min. Each stage was separated by a one min recovery period, during which blood lactate was measured using a LactatePro (Arkray, Kyoto, Japan), and rating of perceived exertion was recorded using the 6-20 Borg Scale (1). Heart rate was measured during each stage of the test (Polar Electro Oy, Kempele, Finland). Expired gases (O2 and CO2) were collected for analysis during all stages of the exercise test to enable determination of each rower’s \(\dot{\text{VO}_2\text{peak}}\). Peak power was used to determine the intensity at which each participant would row during the interval training sessions, and blood lactate was used to determine the traditional rowing training intensity.

**Training protocols**

Following baseline testing rowers were randomly assigned to one of two groups, each of which completed either high-intensity ergometer interval training or traditional ergometer training for four-weeks before re-testing and a cross-over of training intervention occurred. The experimental period involved the incorporation of two ergometer sessions per week. All training sessions supplementary to the ergometer sessions were similar between rowers as they were all part of the same squad preparing for the same race schedule. The only major difference in training between the groups was the ergometer protocol they
completed twice a week. The interval training sessions consisted of eight 2.5 min intervals at 90% of the power that corresponded to the \( \dot{V}O_2 \text{peak} \) achieved during the incremental exercise test. Recovery between each interval was at an intensity of 40% of \( \dot{V}O_2 \text{peak} \) power and the recovery duration was until heart rate returned to 70% of maximum heart rate, up to a maximum of five min. The traditional training program involved rowers completing two ergometer sessions per week; one with a duration of 35 min and the other 40 min. The intensity of each session was set to power outputs that corresponded to blood lactate measurements of 2 and 3 mmol-L\(^{-1}\) determined from the incremental exercise test, as previously described (6). The traditional endurance training program was designed to match the interval training program for energy expenditure. The training program was completed for four weeks at which time re-testing occurred. A four week period was chosen as this has been shown to result in physiological adaptations that benefit performance (4).

**Body composition**

Body composition was assessed prior to each incremental exercise test via dual-energy x-ray absorptiometry (Lunar DPX-L, Lunar Radiation Corporation, WI, USA) to determine body fat percentage, lean mass and fat mass.

**Activity and diet monitoring**

During each four week training period rowers kept a log book in which they recorded their mean training wattages, heart rate and recovery times. Training diaries were completed to enable the determination of training volume for each training program. Each rower completed a log book of training in which mean watts for each ergometer training session
were recorded along with team rowing training. Rowers also kept a diary of dietary intake in the 24 hours preceding the incremental exercise test. On the following testing occasions this dietary intake was replicated.

**Blood collection and adiponectin determination**

A five mL blood sample was obtained from the forearm antecubital space of each rower pre- and immediately post-exercise on the day of the incremental exercise test. Haematocrit was determined via capillary tube centrifugation in duplicate and hemoglobin was determined with a HemoCue (HemoCue AB, Ängelholm, Sweden). Lithium Heparin collection tubes were centrifuged at 4°C for 15 min at 2500 rpm. Plasma was then transferred to new tubes for storage at -80°C until analysis. Plasma was analysed for adiponectin concentration using a sandwich enzyme-linked immunosorbent assay (DRP300, R&D Systems Inc, MN, USA) according to the manufacturer’s instructions. Absorbance was measured at 450 nm using a microplate reader (Tecan Trading AG, Männedorf Switzerland). Post-exercise values were adjusted for changes in plasma volume.

**Statistical analyses**

All statistical analyses were performed using SPSS version 14.0 for Windows (SPSS, Chicago, IL, USA). An ANOVA was used to determine changes across maximal exercise testing trials. When a significant main effect was found, post-hoc t-tests were used to determine where the differences existed. Cohen’s d was calculated to determine effect size (ES) and interpreted as 0.2, 0.6, 1.2, 2.0 and 4.0 for small, moderate, large, very large and extremely large effects (14). Training data were analysed using a t-test. Statistical significance was set at \( p < 0.05 \). Data are presented as mean ± standard deviation (SD).
Results

There was no significant order effect of training ($p > 0.05$), therefore all data collected were analysed as one group. Analysis of total work completed during the high-intensity interval ergometer training program was not significantly different from that of the traditional ergometer training program ($p = 0.25$).

Adiponectin concentration was significantly different across maximal exercise trials ($p = 0.02$). Completion of the high-intensity interval ergometer training program resulted in a significant increase in adiponectin concentration from pre-exercise to post-exercise ($p = 0.016$, ES= 0.47) (Figure 1). There was a moderate to large increase in resting adiponectin concentration after high-intensity interval ergometer training although this was not significant ($24 \pm 29\%$, $p = 0.09$, ES = 0.91). After traditional rowing ergometer training there was no significant change in the response of adiponectin concentration to maximal exercise ($p = 0.69$, ES 0.14), and there was no significant change in resting adiponectin concentration across the four week period ($0.3 \pm 10\%$, $p =0.91$, ES = 0.05).
Figure 1. Adiponectin concentration pre- (pre) and post (post)-graded exercise test at baseline (week 0) and following four weeks (week 4) of a. traditional ergometer training or b. high-intensity interval ergometer training. * = significantly different ($p = 0.016$).
Relative $\dot{V}O_2_{\text{peak}}$ was significantly different across maximal exercise trials ($p = 0.031$). Improvements in relative $\dot{V}O_2_{\text{peak}}$ were evident following completion of high-intensity interval training ($7.3 \pm 7.1\%$, $p = 0.05$, ES = 0.64) but not after traditional rowing training ($0.7 \pm 5.2\%$, $p = 0.7$, ES = 0.22).

Mean power on the final stage of the graded exercise test was significantly different across the testing sessions ($p = 0.044$). Four min power was significantly increased following completion of high-intensity interval training ($6.1 \pm 3.9 \%$, $p = 0.002$, ES = 0.23), but not after traditional rowing training ($4.5 \pm 4.9 \%$, $p = 0.09$, ES = 0.18).

Fat percentage was significantly different across testing times ($p = 0.032$). Fat percentage significantly decreased following high-intensity interval training ($p = 0.022$, ES = 0.17); however, there was no significant change across four weeks of traditional rowing training ($p = 0.64$, ES = 0.03) (Figure 2). Lean mass was not significantly different following high-intensity interval training ($p = 0.12$, ES = 0.01) or traditional training ($p = 0.68$, ES = 0.01). Fat mass was significantly reduced following high-intensity interval training ($p = 0.03$, ES = 0.13) but not traditional training ($p = 0.62$, ES = 0.03).
Figure 2. Body fat % change from baseline to week 4 of a. traditional ergometer training or b. high-intensity interval ergometer training. * = significantly different from baseline ($p = 0.022$).
Discussion

To our knowledge this is the first investigation to determine adiponectin response to a maximal exercise test following a period of high-intensity ergometer interval training and a period of traditional ergometer training in trained rowers. The major novel finding of this study is that while resting adiponectin concentration did not significantly change in response to either training period, adiponectin levels were significantly increased post-exercise following four weeks of high-intensity interval training. Interval training was also associated with improvements in four min mean power output, $\dot{V}O_2$ peak and a decrease in body fat percentage. An increase in post-exercise adiponectin concentration may reflect positive training adaptations associated with improvements in physiological indicators of rowing performance.

Adiponectin concentrations were unchanged across the training period, however, there was an increase in levels following a maximal incremental exercise test after a period of high-intensity interval training. Traditional ergometer training was not associated with significant changes suggesting that the increase in post-exercise adiponectin may reflect differences in adaptation to a particular training program ie. interval training was associated with greater improvements in variables associated with successful rowing performance. Jürimäe et al (17) has shown that post-exercise adiponectin concentrations following a 2000 m rowing time trial were significantly increased in rowers of a higher performance level, those rowers which were selected for a national team for Olympic representation, when compared to non-selected rowers. In combination with the findings of the current study this suggests that training status and exercise intensity may influence the acute exercise response of adiponectin. The intensity of the high-intensity interval
program in the current study was higher than that which was completed in the traditional ergometer rowing training program, and this may have mediated the changes in adiponectin concentration post-exercise.

A number of studies in untrained and obese individuals have shown an increase in resting adiponectin concentration following a period of low to moderate intensity aerobic exercise (18, 21, 22). In contrast, a majority of investigations have reported no change in resting concentration, particularly in healthy trained populations (13, 17). In the present study resting adiponectin levels following a four week period of high-intensity intervals were increased. While this was not statistically significant, the effect size was large (ES = 0.9) suggesting a trend towards increased resting adiponectin concentration following a short term period of interval training. Recently Moghadasi (22) has shown an increase in adiponectin gene expression in adipose tissue and plasma adiponectin concentrations following a 12 week period of high-intensity endurance training. However, these subjects were untrained and had lower levels of circulating adiponectin than the athletes in the present study both before and after training. Consequently, it may be that trained subjects have less capacity to elevate resting adiponectin concentrations due to prior adaptations.

The increases in plasma adiponectin concentration may be associated with improved performance and enhanced recovery of an athlete (17) due to its influence on skeletal muscle bio-energetics. Adiponectin has been shown to upregulate AMP-activated protein kinase (AMPK) activity, increasing PGC-1α signalling and in turn, mitochondrial biogenesis (3, 5, 10, 12). In the present study there was an improvement measured in four min mean power output and peak in response to the high-intensity interval training, but
not the traditional ergometer rowing training. It is possible that the improvement in the peak four min power output and peak evident after the high-intensity training, but not after traditional ergometer training, may have been partly mediated by adiponectin, although this remains speculative.

Improvements in rowing performance have been shown in previous research to be correlated with increased body weight and lean body mass, and decreases in fat mass. The traditional ergometer rowing training did not produce any significant changes in body composition over the four weeks of training. However, the high-intensity interval training was associated with a significant decrease in body fat percentage over the four weeks. Previous research involving type two diabetics and obese individuals have reported a link between body composition and adiponectin concentration, with reductions in body weight and fat mass mediating increases in adiponectin concentration (8, 9, 15, 23, 27). In the present study, body fat percentage of rowers was decreased following four weeks of high-intensity interval training, and there was a moderate to large effect of the training on resting adiponectin concentration. Past research by Jürimäe and colleagues (17) found no significant correlation between changes in body composition and adiponectin concentration, despite a significant decrease in body fat percentage over a six month training period in trained rowers. Body fat percentage was reduced in these athletes, however, fat mass remained unchanged (17). Interestingly, when the groups of rowers were separated into those selected for Olympic representation and those not selected, the selected rowers experienced a significant decrease in fat mass and a trend for an increase in resting adiponectin concentration over the training period (17), supporting the present findings. The relationship between body composition and plasma adiponectin
concentration appears to be influenced by fat mass, and possibly related to training adaptations. While an individuals’ body composition prior to the initiation of the training may play a role in the change in adiponectin levels (16, 17, 27) a decrease in fat mass and increase in resting adiponectin concentration has been shown in the current study, and this has been associated with performance improvements.

In conclusion, four weeks of high-intensity interval training is associated with an increase in adiponectin concentration in response to maximal exercise. High-intensity interval training also resulted in improvements in peak and four min power, and a reduction in body fat. The potential for adiponectin to mediate these beneficial training adaptations requires further investigation.

**Practical Applications**

Four weeks of high-intensity interval training in competitive rowers results in improvements in $\dot{V}O_2\text{peak}$, average peak power and reductions in body fat. In the present study these changes were associated with a moderate to large increase in resting adiponectin concentration and a significant increase post-exercise. Previous research has found that rowers selected for national team representation at the Olympic Games (17) showed an increase in post-exercise adiponectin concentration when compared to non-selected rowers that did not perform as well. In combination with the current study, these findings suggest that an increase in post-exercise adiponectin concentration may reflect positive training adaptations and athlete performance level. Changes in resting adiponectin concentration over training periods and in response to maximal exercise need to be
explored further to determine if monitoring concentrations may prove a useful tool to monitor training load and adaptation, thereby reducing the potential for non-functional overreaching.
References


APPENDIX II:

Participant Information Sheets and Informed Consent

Participant Information Sheet – Study One
Informed Consent Form – Study One
Participant Information Sheet – Study Two
Informed Consent Form – Study Two
Participant Information Sheet – Study Three
Informed Consent Form – Study Three
Participant Information Sheet – Study Four
Informed Consent Form – Study Four
PROJECT TITLE: The effects of high-intensity interval training in highly-trained rowers

INVESTIGATORS: Matt Driller
James Fell
John Gregory
Andrew Williams
Cecilia Shing

Purpose of Study:

You are invited to be a part of a study investigating different types of ergometer training on performance improvements in talent identified athletes.

The main requirement of this study is that you are willing to participate in two individually designed ergometer training sessions per week over an 8-week period. Before and after each phase of training you will perform tests of your physical capacity (maximal exercise test, time-trial tests) to determine the effects of the ergometer training.

There are a number of procedures to be undertaken by participants involved in this study. Testing procedures will be conducted at the laboratory of The University of Tasmania (in Launceston) or in the field at your dedicated training facility. You will/have been thoroughly screened to assess your suitability for exercise and to reduce the risk of any untoward episode as a result of training and testing procedures. Procedures you may have to complete include:
1. Completion of a Physical Activity Readiness Questionnaire (PARQ), Medical History Questionnaire and a Pre-Test Questionnaire.
2. An ergometer test of maximal aerobic function (VO$_2$ max) designed to measure your aerobic fitness and peak power output. Breath samples will be collected during this test as well as blood samples.
3. A 2-kilometre time trial on the rowing ergometer
4. Individually structured ergometer training twice per week during the training phase under the direction of a coach/sports scientist.

1. We will ask you to complete the Medical History Questionnaire and PARQ prior to you beginning the exercise testing. You should not participate in this study if you have ever been diagnosed as suffering from stroke or neurological disease or if you suffer from symptomatic cardiovascular disease, hypertension or diabetes. You should not participate in this study if there is a chance that you may be pregnant or you plan to become pregnant during the study. If your answers to these questionnaires indicate that you meet any of the exclusion criteria you will be excluded from any further involvement in the study.
2. We will assess your aerobic fitness using an ergometer test of maximal aerobic function (VO$_2$\textsubscript{max} test). This will require you to exercise on an ergometer at progressively harder workloads until you reach exhaustion. This “ramp” in exercise intensity will commence at an easy workload and will gradually get harder until you get too tired to continue. The entire test should take about 30 min. During exercise you will breathe room air through a mouthpiece which will allow us to calculate your ability to consume oxygen. This test is routinely performed as part of normal athlete servicing. An investigator with First Aid qualifications will be present during all tests. During the tests fingertip samples of blood and exhaled samples of air will be taken for measurement of blood lactate concentration and oxygen consumption. Aseptic techniques will be used to ensure minimal risk of infection to you and the investigators. Blood samples will also be taken from a forearm vein on the day of testing. A total of no more than 50 mL of blood will be drawn on any single day. This compares to the 500 mL that is taken by the Red Cross when you donate blood. Blood samples collected will be only be used for research directly connected with this experiment. This test will take place in the exercise physiology lab at the University of Tasmania, Launceston Campus.

3. On a separate day you will be required to perform a 2-km time-trial test on the Concept 2 rowing ergometer. This is an ‘all-out’ test that you would be familiar with, intended to measure your ability at your sport. Positive training adaptations should lead to improved time-trial performance. This test will take place at a dedicated training facility that is easily accessible to you.

4. Two ergometer training sessions will be included in the training program that has been specifically designed for you and the demands of your sport.

5. The measurement of bone density will be performed using a Dual Energy X-Ray (DEXA) machine. This machine releases a small amount of radiation as it scans your body. The radiation dose per scan (~0.02mrem) equates to approximately an hours dose of background radiation and compares with a typical radiation dose from a chest x-ray of 30 mrem.

6. The duration of the test sessions, including setting up, exercise and recovery, will be about 1-2 hours.

Involvement in this study will provide the researchers with valuable information about the best type of ergometer training for improving performance in already well-trained athletes. This may help direct and structure future training programs.

All of your test results will remain confidential and will be coded with a number which will be kept separate from your personal details. Papers will be kept in a locked cabinet file at either the University of Tasmania or Tasmanian Institute of Sport. All electronic data will be stored in password protected files. A master code will be held at the University of Tasmania to allow for your data to be matched with data collected at a later stage. Only the researchers involved in this study will have access to data identifying you by name. Five years after the completion of this study, all raw data will be destroyed by shredding. You will be provided with a summary of your results upon completion of the study and in the event of significant findings over the course of the study which might affect you personally you will be informed immediately.

Upon your entry into the study, you will receive copies of the signed information sheet and consent form to keep.

Your participation in this study is at all times voluntary, which means that you may withdraw at anytime before, during or after your participation, without any prejudice; employment, personal or otherwise. If you do want to withdraw you may also request that any responses or data collected from
you be withdrawn also in which case this data will not be included in any publications that may arise from this study.

This study has obtained approval from the University of Tasmania Health and Medical Human Research Ethics Committee. Should you have any questions regarding this study please contact the investigators, James Fell on 6324 5485, or Matt Driller on 0410380199.

If you have any concerns of an ethical nature or complaints about the manner in which the project is conducted, you may contact the Executive Officer of the Health and Medical Human Research Ethics Committee (Tasmania) Network. The Executive Officer can direct participants to the relevant Chair that reviewed the research. You can contact the Executive officer via telephone 6226 7479, or by e-mail human.ethics@utas.edu.au.
PROJECT TITLE: The effects of high-intensity interval training in highly-trained rowers

INVESTIGATORS: Matt Driller (UTAS/Tasmanian Institute of Sport - PhD candidate) James Fell (School of Human Life Sciences, UTAS) John Gregory (Tasmanian Institute of Sport) Andrew Williams (School of Human Life Sciences, UTAS) Cecilia Shing (School of Human Life Sciences, UTAS)

1. I have read and understood the ‘Information Sheet’ for this study.
2. The nature and possible effects of the study have been explained to me.
3. I understand that laboratory testing will be conducted at the Sports Performance Laboratory at The University of Tasmania in Launceston while field testing and training will be conducted at your dedicated training facility.
4. I understand that I will be required to complete a PARQ questionnaire and Medical History Questionnaire at the beginning of the study.
5. I understand that the study involves preliminary assessment including a pre-test questionnaire, body composition measures, a VO2max test and a 2000 m ergometer test.
6. I understand that I will be required to participate in two dedicated ergometer training sessions per week for an eight week period.
7. I understand that the study involves the collection of small volumes of blood throughout the study, and that appropriately qualified personnel will perform blood sampling and physiological measurements.
8. I understand that I will be asked to undergo a bone density scan at the beginning and end of the eight week period
9. I understand that research students are participating in the measures made in the study and in monitoring exercise sessions.

10. I understand that all research data will be treated confidentially.

11. Any questions that I have asked have been answered to my satisfaction.

12. I agree that research data collected for this study may be published provided I cannot be identified as a participant.

13. I understand that research data will be securely stored on the University of Tasmania premises and/or the Tasmanian Institute of Sport premises for a period of at least 5 years. The data will be destroyed 5 years after the completion of this project.

14. I agree to participate in this study and understand that I may withdraw at any time without prejudice or penalty. I understand that my participation in this study is entirely voluntary and without any remuneration, financial or otherwise.

Name of Participant ..........................................................

Signature of Participant ...................................................... Date ....................

If participant is less than 18 years of age.

Name of Legal Guardian.....................................................

Signature of Legal Guardian ........................................... Date ....................

I have explained this project and the implications of participation to the volunteer and I believe that the consent is informed and that he/she understands the implications of participation.

Name of Investigator ..........................................................

Signature of Investigator ..................................................... Date .....................
PROJECT TITLE: The effects of serial and acute NaHCO₃ loading in cyclists

INVESTIGATORS: Mr Matt Driller
Dr James Fell
Dr Andrew Williams
Mr John Gregory

Purpose of Study:

You are invited to be a part of a study investigating the effects of altering blood pH during exercise on cycling performance.

The main requirement of this study is that you are willing to ingest 0.4 g per kg of your body mass of sodium bicarbonate commonly known as baking soda, or a placebo substance (cellulose) each day for 4 days, on 3 separate occasions. Furthermore, after ingestion of the supplement, you will be required to perform a 4-minute cycling ergometer performance trial in the laboratory. The 4-minute test will need to be performed on 3 separate occasions over a 2-week period.

There are a number of procedures to be undertaken by participants involved in this study. Testing procedures will be conducted at the Tasmanian Institute of Sport exercise physiology laboratory. You will/have been thoroughly screened to assess your suitability for exercise and to reduce the risk of any untoward episode as a result of training and testing procedures. Procedures you may have to complete include;
1. Completion of a Physical Activity Readiness Questionnaire (PARQ) and a Medical History Questionnaire. You should not participate in this study if you have ever been diagnosed as suffering from stroke or neurological disease or if you suffer from symptomatic cardiovascular disease, hypertension or diabetes. You should not participate in this study if there is a chance that you may be pregnant or you plan to become pregnant during the study. If your answers to these questionnaires indicate that you meet any of the exclusion criteria you will be excluded from any further involvement in the study.
2. A 4-minute all-out test on a cycle ergometer. During the test you will breathe room air through a mouthpiece which will allow us to calculate your ability to consume oxygen. This test is routinely performed as part of normal athlete servicing. An investigator with First Aid qualifications will be present during all tests. This test will take place on three separate occasions in the exercise physiology lab at the Tasmanian Institute of Sport.
3. Sodium bicarbonate supplementation or a placebo supplement in the 4 days prior to each testing session. You will be required to ingest 0.4 g per kg of your body mass of sodium bicarbonate (capsule form with ~2L of water) each day or a placebo (cellulose capsules). Sodium bicarbonate increases the pH of your blood and is not harmful in low doses. Furthermore, this ingestion protocol has been used by a number of research studies to induce a mild alkalosis within the body. In some
people there is a small chance of bloating and/or mild gastro-intestinal discomfort following the sodium bicarbonate ingestion. Every effort will be made to minimise this risk by providing you with instructions to reduce this (i.e. drinking plenty of water, supplementing the dose with food and using an appropriate does-timing method).

4. Additional finger-tip blood samples before ingestion of sodium bicarbonate or cellulose in order to monitor changes in blood pH, NaHCO$_3$ and blood lactate concentrations. Aseptic techniques will be used to ensure minimal risk of infection to you and the investigators.

Involvement in this study will provide the researchers with valuable information concerning the effectiveness of sodium bicarbonate as a performance supplement in well-trained cyclists.

All of your test results will remain confidential and will be coded with a number which will be kept separate from your personal details. Papers will be kept in a locked cabinet file at either the University of Tasmania or Tasmanian Institute of Sport. All electronic data will be stored in password protected files. A master code will be held at the University of Tasmania to allow for your data to be matched with data collected at a later stage. Only the researchers involved in this study will have access to data identifying you by name. Five years after the completion of this study, all raw data will be destroyed by shredding. You will be provided with a summary of your results upon completion of the study and in the event of significant findings over the course of the study which might affect you personally you will be informed immediately.

Upon your entry into the study, you will receive copies of the signed information sheet and consent form to keep.

Your participation in this study is at all times voluntary, which means that you may withdraw at anytime before, during or after your participation, without any prejudice; employment, personal or otherwise. If you do want to withdraw you may also request that any responses or data collected from you be withdrawn also in which case this data will not be included in any publications that may arise from this study.

This study has obtained approval from the University of Tasmania Health and Medical Human Research Ethics Committee. Should you have any questions regarding this study please contact the investigators, James Fell on 6324 5485, or Matt Driller on 0410380199.

If you have any concerns of an ethical nature or complaints about the manner in which the project is conducted, you may contact the Executive Officer of the Health and Medical Human Research Ethics Committee (Tasmania) Network. The Executive Officer can direct participants to the relevant Chair that reviewed the research. You can contact the Executive officer via telephone 6226 7479, or by e-mail human.ethics@utas.edu.au.

For more information about the study please contact:
Matt Driller on 03 6324 5497 or 0410380199
John Gregory on 03 6336 2256
or James Fell on 03 6324 5485
Statement of Informed Consent
(Study 2)

PROJECT TITLE: The effects of serial and acute NaHCO₃ loading in cyclists

INVESTIGATORS: Matt Driller (UTAS/Tasmanian Institute of Sport - PhD candidate)
James Fell (School of Human Life Sciences, UTAS)
Andrew Williams (School of Human Life Sciences, UTAS)
John Gregory (Tasmanian Institute of Sport)

1. I have read and understood the ‘Information Sheet’ for this study.

2. The nature and possible effects of the study have been explained to me.

3. I understand that laboratory testing will be conducted at the Sports Performance Laboratory at the Tasmanian Institute of Sport in Launceston.

4. I understand that I will be required to complete a PARQ questionnaire and Medical History Questionnaire at the beginning of the study.

5. I understand that the study involves exercise assessment including a 4-minute cycle ergometer test with gas analysis.

6. I understand that the study involves the collection of small volumes of blood throughout the study, and that appropriately qualified personnel will perform blood sampling and physiological measurements.

7. I understand that I will be required to ingest 0.4 g of NaHCO₃ per kg of my body mass or a placebo substance (cellulose) each day before the testing session.

8. I understand the possible side effects or complications associated with NaHCO₃ ingestion.

9. I understand that all research data will be treated confidentially.

10. Any questions that I have asked have been answered to my satisfaction.

11. I agree that research data collected for this study may be published provided I cannot be identified as a participant.
12. I understand that research data will be securely stored on the University of Tasmania premises and/or the Tasmanian Institute of Sport premises for a period of at least 5 years. The data will be destroyed 5 years after the completion of this project.

13. I agree to participate in this study and understand that I may withdraw at any time without prejudice or penalty. I understand that my participation in this study is entirely voluntary and without any remuneration, financial or otherwise.

Name of Participant ……………………………………………

Signature of Participant ………………………………………….. Date ………………..

**If participant is less than 18 years of age.**

Name of Legal Guardian…………………………………………

Signature of Legal Guardian ……………………………………. Date ………………..

I have explained this project and the implications of participation to the volunteer and I believe that the consent is informed and that he/she understands the implications of participation.

Name of Investigator ……………………………………………

Signature of Investigator …………………………………….. Date ………………..
PROJECT TITLE: The effects of chronic sodium bicarbonate ingestion and interval training in highly-trained rowers

INVESTIGATORS: Mr Matt Driller  
Dr James Fell  
Dr Andrew Williams  
Mr John Gregory

Purpose of Study:

You are invited to be a part of a study investigating the effects of altering blood pH during high-intensity training on rowing performance.

The main requirement of this study is that you are willing to participate in two individually designed ergometer training sessions per week over a 4-week period. Furthermore, before each training session, you will be required to ingest 0.3 g per kg of your body mass of sodium bicarbonate (NaHCO₃) commonly known as baking soda, or 0.2 g per kg of your body mass of sodium chloride (control). Before and after the 4-weeks of training, you will perform tests of your physical capacity (submaximal/maximal exercise tests, time-trial test) to determine the effects of the ergometer training with sodium bicarbonate supplementation.

There are a number of procedures to be undertaken by participants involved in this study. Testing procedures will be conducted at the Tasmanian Institute of Sport exercise physiology laboratory. You will/have been thoroughly screened to assess your suitability for exercise and to reduce the risk of any untoward episode as a result of training and testing procedures. Procedures you may have to complete include;

1. Completion of a Physical Activity Readiness Questionnaire (PARQ), Medical History Questionnaire and a Pre-Test Questionnaire.
2. An ergometer test of maximal aerobic function (VO₂ max) designed to measure your aerobic fitness and peak power output. Breath samples will be collected during this test as well as finger-tip blood samples.
5. Individually structured ergometer training twice per week during the training phase under the direction of a coach/sports scientist.
6. Sodium bicarbonate supplementation or a control supplement prior to each training session.
7. Additional finger-tip blood samples during training sessions on two separate occasions in order to monitor blood pH, NaHCO₃ and blood lactate concentrations.

1. We will ask you to complete the Medical History Questionnaire and PARQ prior to you beginning the exercise testing. You should not participate in this study if you have ever been diagnosed as suffering from stroke or neurological disease or if you suffer from symptomatic cardiovascular disease, hypertension or diabetes. You should not participate in this study if there
is a chance that you may be pregnant or you plan to become pregnant during the study. If your answers to these questionnaires indicate that you meet any of the exclusion criteria you will be excluded from any further involvement in the study.

2. We will assess your aerobic fitness using an ergometer test of maximal aerobic function (VO$_2$ \textit{max} test). This will require you to exercise on an ergometer at progressively harder workloads until you reach exhaustion. This “ramp” in exercise intensity will commence at an easy workload and will gradually get harder until you get too tired to continue. The entire test should take about 30 min. During exercise you will breathe room air through a mouthpiece which will allow us to calculate your ability to consume oxygen. This test is routinely performed as part of normal athlete servicing. An investigator with First Aid qualifications will be present during all tests. During the tests fingertip samples of blood and exhaled samples of air will be taken for measurement of blood lactate concentration and oxygen consumption. Aseptic techniques will be used to ensure minimal risk of infection to you and the investigators. This test will take place on one occasion at the start of the study, in the exercise physiology lab at the Tasmanian Institute of Sport.

3. You will be required to perform a 2000 m time-trial test on the Concept 2 rowing ergometer. This is an ‘all-out’ test that you would be familiar with, intended to measure your ability in your sport. Positive training adaptations should lead to improved time-trial performance. This test will take place at a dedicated training facility that is easily accessible to you.

4. Two ergometer training sessions will be included in your training program each week that have been specifically designed for you and the demands of your sport. Each session will take between 45 min – 1 hour. The session will involve high-intensity intervals with periods of recovery between each bout of exercise. Exercise intensity will be prescribed from your maximal exercise test. These training sessions will be performed at your dedicated training facility.

5. Prior to each training session, you will be required to ingest 0.3 g per kg of your body mass of sodium bicarbonate (capsule form with ~1L of water) or 0.2 g per kg of your body mass of sodium chloride (control). Sodium bicarbonate increases the pH of your blood and is not harmful in low doses. Furthermore, this ingestion protocol has been used by a number of research studies to induce a mild alkalosis within the body. In some people there is a small chance of bloating and/or mild gastro-intestinal discomfort following the sodium bicarbonate ingestion. Every effort will be made to minimise this risk by providing you with instructions to reduce this (i.e. drinking plenty of water, supplementing the dose with food and using an appropriate does-timing method). The control (sodium chloride: table salt) has been associated with increased blood pressure. However, this association is only due to long term consumption of high salt food. The few doses required in this study present no risk of increased blood pressure. Currently there is no information regarding the safety to long-term consumption of supplements used in this project.

6. On two occasions throughout the study, you will be required to give finger-tip blood samples before ingesting the sodium bicarbonate (or control), after ingestion and after the training session. This is in order to analyse the blood pH, NaHCO$_3$ and blood lactate response to the sodium bicarbonate and the training.

Involvement in this study will provide the researchers with valuable information concerning the effectiveness of sodium bicarbonate as a training supplement in well-trained rowers. This may help to improve performance and direct future training programs.
All of your test results will remain confidential and will be coded with a number which will be kept separate from your personal details. Papers will be kept in a locked cabinet file at either the University of Tasmania or Tasmanian Institute of Sport. All electronic data will be stored in password protected files. A master code will be held at the University of Tasmania to allow for your data to be matched with data collected at a later stage. Only the researchers involved in this study will have access to data identifying you by name. Five years after the completion of this study, all raw data will be destroyed by shredding. You will be provided with a summary of your results upon completion of the study and in the event of significant findings over the course of the study which might affect you personally you will be informed immediately.

Upon your entry into the study, you will receive copies of the signed information sheet and consent form to keep.

Your participation in this study is at all times voluntary, which means that you may withdraw at anytime before, during or after your participation, without any prejudice; employment, personal or otherwise. If you do want to withdraw you may also request that any responses or data collected from you be withdrawn also in which case this data will not be included in any publications that may arise from this study.

This study has obtained approval from the University of Tasmania Health and Medical Human Research Ethics Committee. Should you have any questions regarding this study please contact the investigators, Matt Driller on 03 6324 5497 or James Fell on 03 6324 5485.

If you have any concerns of an ethical nature or complaints about the manner in which the project is conducted, you may contact the Executive Officer of the Health and Medical Human Research Ethics Committee (Tasmania) Network. The Executive Officer can direct participants to the relevant Chair that reviewed the research. You can contact the Executive officer via telephone 6226 7479, or by e-mail human.ethics@utas.edu.au.
Statement of Informed Consent

(Study 3)

PROJECT TITLE: The effects of chronic sodium bicarbonate ingestion and interval training in highly-trained rowers

INVESTIGATORS: Matt Driller (UTAS/Tasmanian Institute of Sport - PhD candidate)  
James Fell (School of Human Life Sciences, UTAS)  
John Gregory (Tasmanian Institute of Sport)  
Andrew Williams (School of Human Life Sciences, UTAS)

1. I have read and understood the ‘Information Sheet’ for this study.

2. The nature and possible effects of the study have been explained to me.

3. I understand that laboratory testing will be conducted at the Sports Performance Laboratory at the Tasmanian Institute of Sport in Launceston while field testing and training will be conducted at your dedicated training facility.

4. I understand that I will be required to complete a PARQ questionnaire and Medical History Questionnaire at the beginning of the study.

5. I understand that the study involves preliminary assessment including a pre-test questionnaire, body composition measures, a VO₂max test and a 2000 m ergometer test.

6. I understand that I will be required to participate in two dedicated ergometer training sessions per week for a four-week period.

7. I understand that the study involves the collection of small volumes of blood throughout the study, and that appropriately qualified personnel will perform blood sampling and physiological measurements.

8. I understand that I will be required to ingest 0.3g of NaHCO₃ or 0.2g of NaCl per kg of my body mass prior to each training session.

9. I understand the possible side effects or complications associated with NaHCO₃ ingestion.
10. I understand that research students are participating in the measures made in the study and in monitoring exercise sessions.

11. I understand that all research data will be treated confidentially.

12. Any questions that I have asked have been answered to my satisfaction.

13. I agree that research data collected for this study may be published provided I cannot be identified as a participant.

14. I understand that research data will be securely stored on the University of Tasmania premises and/or the Tasmanian Institute of Sport premises for a period of at least 5 years. The data will be destroyed 5 years after the completion of this project.

15. I agree to participate in this study and understand that I may withdraw at any time without prejudice or penalty. I understand that my participation in this study is entirely voluntary and without any remuneration, financial or otherwise.

Name of Participant ………………………………………………

Signature of Participant ………………………………………….. Date ………………...

If participant is less than 18 years of age.

Name of Legal Guardian…………………………………………

Signature of Legal Guardian ……………………………………. Date ………………..

________________________________________

I have explained this project and the implications of participation to the volunteer and I believe that the consent is informed and that he/she understands the implications of participation.

Name of Investigator ……………………………………………

Signature of Investigator ………………………………………... Date ………………..
PROJECT TITLE: The effects of NaHCO₃ and NaCl loading on haematocrit and high-intensity cycling performance

INVESTIGATORS: Mr Matt Driller
Dr James Fell
Mr Phil Bellinger
Mr Samuel Howe

Purpose of Study:

You are invited to be a part of a study investigating the effects of altering blood pH during exercise on cycling performance.

The main requirement of this study is that you are willing to ingest 0.3 g per kg of your body mass of sodium bicarbonate commonly known as baking soda, 0.2 g per kg of sodium chloride (salt), or a placebo substance (dextrose) on 3 separate occasions. Furthermore, after ingestion of the supplement, you will be required to perform a 2-minute cycling ergometer performance trial in the laboratory. The 2-minute test will need to be performed on 3 separate occasions over a 1-week period.

There are a number of procedures to be undertaken by participants involved in this study. Testing procedures will be conducted at the University of Tasmania exercise physiology laboratory. You will/have been thoroughly screened to assess your suitability for exercise and to reduce the risk of any untoward episode as a result of training and testing procedures. Procedures you may have to complete include:

1. Completion of a Physical Activity Readiness Questionnaire (PARQ) and a Medical History Questionnaire. You should not participate in this study if you have ever been diagnosed as suffering from stroke or neurological disease or if you suffer from symptomatic cardiovascular disease, hypertension or diabetes. You should not participate in this study if there is a chance that you may be pregnant or you plan to become pregnant during the study. If your answers to these questionnaires indicate that you meet any of the exclusion criteria you will be excluded from any further involvement in the study.

2. A 2-minute all-out test on a cycle ergometer. During the test you will breathe room air through a mouthpiece which will allow us to calculate your ability to consume oxygen. This test is routinely performed as part of normal athlete servicing. An investigator with First Aid qualifications will be present during all tests. This test will take place on three separate occasions in the exercise physiology lab at the University of Tasmania.

3. Ingestion of sodium bicarbonate supplementation, sodium chloride or a placebo supplement will occur 2 hours before each of the 3 testing sessions. You will be required to ingest 0.3 g per kg of
your body mass of sodium bicarbonate, 0.2g per kg of sodium chloride or a placebo (dextrose) (capsule form with ~1.5L of water) each day before the testing. Sodium bicarbonate increases the pH of your blood and is not harmful in low doses. Furthermore, this ingestion protocol has been used by a number of research studies to induce a mild alkalosis within the body. In some people there is a small chance of bloating and/or mild gastro-intestinal discomfort following the sodium bicarbonate and/or salt ingestion. Every effort will be made to minimise this risk by providing you with instructions to reduce this (i.e. drinking plenty of water, supplementing the dose with food and using an appropriate dose-timing method).

4. Participants will be required to keep a diet diary, recording consumption of foods the day prior to the initial test. This diet is to be replicated on the days preceding the next 2 tests.

5. Additional finger-tip blood samples before ingestion of sodium bicarbonate, sodium chloride or dextrose will be taken in order to monitor changes in haematocrit (blood concentration) readings. Aseptic techniques will be used to ensure minimal risk of infection to you and the investigators. The finger-tip blood samples will be taken 2 hours before the test, 1 hour before the test, immediately before the test and immediately after the test.

Involvement in this study will provide the researchers with valuable information concerning the effectiveness of sodium bicarbonate and salt as a performance supplement in healthy athletes.

All of your test results will remain confidential and will be coded with a number which will be kept separate from your personal details. Papers will be kept in a locked cabinet file at either the University of Tasmania or Tasmanian Institute of Sport. All electronic data will be stored in password protected files. A master code will be held at the University of Tasmania to allow for your data to be matched with data collected at a later stage. Only the researchers involved in this study will have access to data identifying you by name. Five years after the completion of this study, all raw data will be destroyed by shredding. You will be provided with a summary of your results upon completion of the study and in the event of significant findings over the course of the study which might affect you personally you will be informed immediately.

Upon your entry into the study, you will receive copies of the signed information sheet and consent form to keep.

Your participation in this study is at all times voluntary, which means that you may withdraw at anytime before, during or after your participation, without any prejudice; employment, personal or otherwise. If you do want to withdraw you may also request that any responses or data collected from you be withdrawn also in which case this data will not be included in any publications that may arise from this study.

This study has obtained approval from the University of Tasmania Health and Medical Human Research Ethics Committee. Should you have any questions regarding this study please contact the investigators, James Fell on 6324 5485, or Matt Driller on 0410380199.

If you have any concerns of an ethical nature or complaints about the manner in which the project is conducted, you may contact the Executive Officer of the Health and Medical Human Research Ethics Committee (Tasmania) Network. The Executive Officer can direct participants to the relevant Chair that reviewed the research. You can contact the Executive officer via telephone 6226 7479, or by e-mail human.ethics@utas.edu.au.

For more information about the study please contact:
Matt Driller on 03 6324 5497 or 0410380199
PROJECT TITLE: The effects of NaHCO\textsubscript{3} and NaCl loading on haematocrit and high-intensity cycling performance

INVESTIGATORS: Mr Matt Driller (UTAS/Tasmanian Institute of Sport - PhD candidate) Dr James Fell (School of Human Life Sciences, UTAS) Mr Phil Bellinger (School of Human Life Sciences, UTAS) Mr Samuel Howe (School of Human Life Sciences, UTAS)

1. I have read and understood the ‘Information Sheet’ for this study.

2. The nature and possible effects of the study have been explained to me.

3. I understand that laboratory testing will be conducted at the Exercise Laboratory at the University of Tasmania in Launceston.

4. I understand that I will be required to complete a PARQ questionnaire and Medical History Questionnaire at the beginning of the study.

5. I understand that the study involves exercise assessment including a 2-minute cycle ergometer test with gas analysis.

6. I understand that the study involves the collection of small volumes of blood throughout the study, and that appropriately qualified personnel will perform blood sampling and physiological measurements.

7. I understand that I will be required to ingest 0.3 g of NaHCO\textsubscript{3} per kg of my body mass, 0.2g of sodium chloride or a placebo substance (dextrose) on the day of each the testing session.

8. I understand the possible side effects or complications associated with NaHCO\textsubscript{3} and/or salt ingestion.

9. I understand that all research data will be treated confidentially.

10. Any questions that I have asked have been answered to my satisfaction.
11. I agree that research data collected for this study may be published provided I cannot be identified as a participant.

12. I understand that research data will be securely stored on the University of Tasmania premises and/or the Tasmanian Institute of Sport premises for a period of at least 5 years. The data will be destroyed 5 years after the completion of this project.

13. I agree to participate in this study and understand that I may withdraw at any time without prejudice or penalty. I understand that my participation in this study is entirely voluntary and without any remuneration, financial or otherwise.

Name of Participant ………………………………………………

Signature of Participant …………………………………………..   Date ………………...

**If participant is less than 18 years of age.**

Name of Legal Guardian……………………………………………

Signature of Legal Guardian …………………………………….   Date ………………..

I have explained this project and the implications of participation to the volunteer and I believe that the consent is informed and that he/she understands the implications of participation.

Name of Investigator …………………………………………………

Signature of Investigator ……………………………………………   Date ………………..
APPENDIX III:

Questionnaires

Health screening questionnaire
PAR-Q
Gastro-intestinal symptoms questionnaire
RPE
# Health Screening Questionnaire

**Exercise Screening Questionnaire**

<table>
<thead>
<tr>
<th>Name</th>
<th>Occupation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Address</td>
<td>Day Phone</td>
</tr>
<tr>
<td></td>
<td>Evening Phone</td>
</tr>
<tr>
<td>Contact Person (1)</td>
<td>Contact Person (2)</td>
</tr>
<tr>
<td>Phone</td>
<td>Phone</td>
</tr>
<tr>
<td>Today’s Date</td>
<td>Doctor</td>
</tr>
<tr>
<td>Date of Birth</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>Age</td>
</tr>
</tbody>
</table>

Please answer the following questions by placing a tick in the appropriate box.

<table>
<thead>
<tr>
<th>Health Status</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Have you ever had a stroke or heart condition?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Have you ever had high blood pressure?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Have any family members had heart problems before age 60?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 Have you experienced chest pain when engaged in physical activity?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 Have you experienced chest pain when not engaged in physical activity?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 Have you ever had, or do you currently have, high blood cholesterol?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 Have you ever suffered from asthma or breathing difficulties?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 Have you ever smoked – cigarettes, pipes or cigars?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 Are you pregnant or have you been pregnant within the last three months?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 Have you been hospitalised within the last six months?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11 Are you currently taking any medication(s)?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12 Have you ever had, or do you currently have, diabetes, epilepsy, hernia, dizziness or loss of consciousness?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13 Have you ever had any disease or injury of the back, joints, bones or muscles that may be aggravated by exercise?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14 Are you aware of any other health-related issues that may affect your participation in physical exercise?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Please provide details of "Yes" answers in the space provided below.
Details of "Yes" answers, medications, possible contraindications to exercise, etc.


Please answer the following questions by placing a tick in the appropriate box.

<table>
<thead>
<tr>
<th>Exercise Participation</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Have you been participating in regular physical activity? If yes, what type and how often?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<p>| 2 How would you describe your current physical condition? (Tick one or more boxes).     |</p>
<table>
<thead>
<tr>
<th>unwell</th>
<th>overweight</th>
<th>unfit</th>
<th>healthy</th>
<th>fit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- I have understood all the questions and have answered them to the best of my knowledge.
- I certify that I have disclosed fully any conditions that may affect my participation in physical exercise.

Date

Staff Name

Client Signature

Staff Signature
Physical Activity Readiness Questionnaire (PARQ)

For most people, physical activity should not present any problem or hazard. The PARQ has been designed to identify the small number of adults for whom physical activity might be inappropriate and those who should have medical advice concerning the type of activity most suitable. If you are over 69 years of age, and you are not used to being very active, check with your doctor before beginning to exercise.

Date: ………………………

Client :………………………………………………

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Has a doctor ever said that you have a heart condition and that you should only do physical exercise recommended by a doctor?</td>
<td>Yes/No</td>
</tr>
<tr>
<td>2. Do you feel pain in your chest when you do physical activity?</td>
<td>Yes/No</td>
</tr>
<tr>
<td>3. In the past month, have you had chest pain when you were not doing physical activity?</td>
<td>Yes/No</td>
</tr>
<tr>
<td>4. Do you lose your balance because of dizziness or do you ever lose consciousness?</td>
<td>Yes/No</td>
</tr>
<tr>
<td>5. Do you have a bone or joint problem that could be made worse by a change in your physical activity?</td>
<td>Yes/No</td>
</tr>
<tr>
<td>6. Is your doctor currently prescribing drugs (eg. water pills) for your blood pressure or heart condition?</td>
<td>Yes/No</td>
</tr>
<tr>
<td>7. Do you know of any other reason you should not do physical activity?</td>
<td>Yes/No</td>
</tr>
<tr>
<td>8. Are you over a male over 45 or a female over 55?</td>
<td>Yes/No</td>
</tr>
</tbody>
</table>

If a person answers “YES” to any of these questions, vigorous exercise or exercise testing should be postponed until medical clearance has been obtained (and Coronary Artery Disease Risk Factor Checklist has been successfully completed).
Coronary Artery Disease Risk Factor Thresholds

<table>
<thead>
<tr>
<th>Question</th>
<th>Yes/No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Has a member of your immediate family (father/brother &lt; 55 yrs; mother/sister &lt; 65 yrs) been diagnosed with heart disease?</td>
<td></td>
</tr>
<tr>
<td>2. Are you a current cigarette smoker or have quit within the last 6 months?</td>
<td></td>
</tr>
<tr>
<td>3. Have you been told that you have high total blood cholesterol, high very low density lipoprotein (VLDL) cholesterol or that your high density lipoprotein (HDL) cholesterol is low?</td>
<td></td>
</tr>
<tr>
<td>4. Have you been told that you have impaired fasting glucose (plasma glucose &gt; 6.1 mmol/L noted on 2 separate occasions) or type 2 diabetes?</td>
<td></td>
</tr>
<tr>
<td>5. Do you have a body mass index (BMI) greater than or equal to 30 kg/m2 or a waist circumference greater than 100 cm?</td>
<td></td>
</tr>
<tr>
<td>6. Do you currently undertake exercise on less than 4 days each week (at least 30 minutes in duration for each bout)?</td>
<td></td>
</tr>
</tbody>
</table>

If a person answers “YES” to 2 or more of these questions, vigorous exercise should be postponed until medical clearance has been obtained.

Resting Blood Pressure …/…… mmHg (seated)

Clinician’s Name: ………………………

Clinician’s Signature: ………………………
Gastro-Intestinal Symptoms Questionnaire

For the following symptoms use a vertical line to mark your rating on the lines below...

**Stomach problems**

None

**Nausea**

None

**Dizziness**

None

**Headache**

None

**Flatulence**

None

**Urge to urinate**

None

**Urge to defecate**

None

**Bloating**

None

**Stomach cramps**

None
Urge to vomit

None

Unbearable

Vomiting

None

Unbearable

Diarrhoea

None

Unbearable

Muscle cramps

None

Unbearable
Borgs RPE Scale

6  No exertion at all
7  Extremely light
8
9  Very light
10
11 Light
12
13 Somewhat hard
14
15 Hard (heavy)
16
17 Very hard
18
19 Extremely hard
20 Maximal exertion
APPENDIX IV:

Diaries

Training Diary

Diet Diary
Athlete Training Diary

Name: __________________________
Personal Details
(To be completed by the athlete)

Full name (Surname/first name):
___________________________________________

Date of birth (dd/mm/yy):______________________

Age in years today: __________

Home address: _____________________________________

Post Code: __________

Gender (male/female):____________________________________________________

(To be completed by the TIS/UTAS)

ID Number: ___________________________

Training Group: _________________

If you have any questions regarding the training or this training diary, please don’t hesitate to contact:

Matt Driller
0410380199
We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the last 7 days. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the vigorous activities that you did in the last 7 days. Vigorous physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

1. During the last 7 days, on how many days did you do vigorous physical activities like heavy lifting, digging, aerobics, or fast bicycling?
   
   _____ days per week
   
   □ No vigorous physical activities → Skip to question 3

2. How much time did you usually spend doing vigorous physical activities on one of those days?

   _____ hours per day
   _____ minutes per day

   □ Don’t know/Not sure

Think about all the moderate activities that you did in the last 7 days. Moderate activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

3. During the last 7 days, on how many days did you do moderate physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

   _____ days per week
   
   □ No moderate physical activities → Skip to question 5

4. How much time did you usually spend doing moderate physical activities on one of those days?

   _____ hours per day
Think about the time you spent walking in the last 7 days. This includes at work and at home, walking to travel from place to place, and any other walking that you might do solely for recreation, sport, exercise, or leisure.

5. During the last 7 days, on how many days did you walk for at least 10 minutes at a time?

   _____ days per week

   [ ] No walking  → Skip to question 7

6. How much time did you usually spend walking on one of those days?

   _____ hours per day
   _____ minutes per day

   [ ] Don’t know/Not sure

The last question is about the time you spent sitting on weekdays during the last 7 days. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

7. During the last 7 days, how much time did you spend sitting on a week day?

   _____ hours per day
   _____ minutes per day

   [ ] Don’t know/Not sure

This is the end of the questionnaire, thank you for participating.
Training Diary Instructions

1. Athletes are required to complete the personal details section and training record sections.

2. The training record requires a number of entries as explained below. Keep a record of your overall weekly training activities on the page provided.

   - **Date**: record the day and date of the month for the training session e.g. Monday 16th Feb
   - **Time of day**: record for each of the training sessions e.g. 3:30pm
   - **Type of training**: type of training (E.g. wind-trainer, road, MTB, weights, other)
   - **Distance Covered and/or Training duration**: record how many kilometres you cycled, and if you know - how long you were training for in minutes.
   - **Average watts**: record at the end of each session (if you have a power meter).
   - **Intensity level**: Record on average how hard you felt the session was. On a continuum of 1 to 10, 1 being very, very easy and 10 being maximal (see below)

<table>
<thead>
<tr>
<th>Rating</th>
<th>Descriptor</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Rest</td>
</tr>
<tr>
<td>1</td>
<td>Very, Very Easy</td>
</tr>
<tr>
<td>2</td>
<td>Easy</td>
</tr>
<tr>
<td>3</td>
<td>Moderate</td>
</tr>
<tr>
<td>4</td>
<td>Somewhat Hard</td>
</tr>
<tr>
<td>5</td>
<td>Hard</td>
</tr>
<tr>
<td>6</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Very Hard</td>
</tr>
<tr>
<td>8</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Maximal</td>
</tr>
</tbody>
</table>

**Heart rate**: if you are wearing a heart rate monitor record the average heart rate from the session.
## Training Record – Weekly training

<table>
<thead>
<tr>
<th>Day</th>
<th>Session</th>
<th>Date</th>
<th>Time</th>
<th>Type of Training</th>
<th>Distance Covered</th>
<th>Total Time Training</th>
<th>Intensity Rating (1-10)</th>
<th>Heart Rate Average (Bpm)</th>
<th>Average Watts</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>02/07/09</td>
<td>11:30am</td>
<td>Road cycling</td>
<td>~80 km</td>
<td>140 minutes</td>
<td>8</td>
<td>155 bpm</td>
<td>N/A</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Example

**Note** – No training is to be performed the day of the testing.

**Comments:**
Name: ______________________

Instructions:

Please read these important instructions carefully

• Record ALL the food and drinks consumed.
• Record the food at the time of eating and NOT from the memory at the end of the day.
• You should include all the meals & snacks, plus sweets, drinks (including water etc.)
• Remember to include any additions to foods already recorded such as sauces, dressings or extras e.g. gravy, salad dressings, stuffing, sugar, honey, syrups etc., butter or margarine (e.g. added to bread, crackers, vegetables).

Describing food and drink – guidelines

• Give as many details as possible about the type of food that you eat - brand name of food where applicable (e.g. Miracle margarine)
  - Breakfast cereal e.g. Weetbix
  - Fruit e.g. fresh, dried, stewed
    - fat/ sugar content of food
  - Milk e.g. whole or skim or low fat
  - Soft drink e.g. regular or diet/ low joule
  - Name and type of cheese e.g. cheddar full fat or cheddar reduced fat
**Meat, fish or poultry** (please specify - full fat or trimmed or if the meat was cooked with fat on, but removed before eating) e.g.
- Beef - silverside full fat/trimmed fat
- Fish - cod fillet
- Chicken fillet with skin/without skin

**Type of cooking oil** e.g. olive oil or vegetable oil
- Give the details of the method of cooking for all foods
  e.g. Fried, boiled, roasted, steamed, poached etc

**Recording the amounts of foods you eat**
It is very important that you record the quantity of each food and drink you consume.
Here are a few suggestions:

**For cooked foods**
- **Weigh** cooked food and not raw food.
- **Weigh** the food after it is served.
- If you do not eat all the food you first served out, measure and write how much is left.
- For a mixed dish like casserole, record the total amount eaten. It will help us if you name the ingredients e.g. chicken, carrots, potatoes
- Please specify if the food was home cooked or take away.

**For foods such as sandwich and salad**
- If prepared at home, weigh the bread slice and all the individual fillings e.g. 2 slices of ‘Helgas’ wholemeal bread – slice weight on packet 45g or two slices of mixed grain homemade bread – weight 100g total. 10g olive oil margarine, 50g iceberg lettuce.
- If the sandwich was bought, specify the type of bread and the type of fillings.

**For fruits**
- Please weigh if at home otherwise specify small, medium or large as serve.

**Spreads and sugar**
- Butter, margarine, jam, mayonnaise and sugar can be measured in teaspoons (tsp.) or tablespoons (tbsp.) e.g. 1
level tsp. of butter, 2 rounded tsp. of brown sugar, 1 heaped tbsp. of reduced fat cream etc.

For canned and pre-packed foods
- Specify the brand and amounts e.g. Weight can be quoted as half of 425g can of Heinz baked beans.

While eating out
- Use the photos (attached at the end of the diet diary) as a guide e.g.
  - 1 portion of rice of size A
  - 1 portion of steak size B
  - 2 portions of cake size A
  - 1 portion of peas size A
- Use comparisons for describing portion sizes where easy to do so e.g. Potato – size of hen’s egg

It is important that you do not adjust what you eat and drink because you are keeping a record. Remember we are interested in your eating habits, not the perfect diet.
Example sheet

Day: 1    Date: 23/09/2006    Day of the week: Saturday

Please record:
- Everything you eat and drink today, including sweets, snacks, nibbles, sauces and dressings
- Method of cooking (e.g. boiled pasta)
- Type of food (e.g. boiled wholegrain pasta)
- Quantity of food (e.g. 100g boiled wholegrain pasta)

<table>
<thead>
<tr>
<th>Meal/Snack (Time of day)</th>
<th>Details of food and drink (Including cooking method)</th>
<th>Amount served</th>
<th>Amount left (If any)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early morning</td>
<td>Plain water</td>
<td>1 glass</td>
<td>Nil</td>
</tr>
<tr>
<td>Breakfast</td>
<td>Weetbix Pura light milk Black coffee</td>
<td>45g (1½ biscuits) 100 gm 1 cup</td>
<td></td>
</tr>
<tr>
<td>Mid-morning</td>
<td>Banana</td>
<td>150g</td>
<td>Skin (~ 20g)</td>
</tr>
<tr>
<td>Lunch</td>
<td>Bread Helga’s wholemeal Olive grove margarine Ice berg lettuce Tomato Ham Cheese – Table cape vintage</td>
<td>60 gm (2 slice) 5 gm 30 gm 30 g 20g 30g</td>
<td></td>
</tr>
<tr>
<td>Afternoon</td>
<td>Cadbury’s dairy milk chocolate</td>
<td>1 x 40g</td>
<td></td>
</tr>
<tr>
<td>Evening meal</td>
<td>Stir fry chicken and vegetable - Contained carrots, beans, capsicum- green and red, mushroom, chicken breast (trimmed), olive oil, soy sauce and spices White rice Full strength Boags draught beer</td>
<td>300g 100g 1 x 375mL</td>
<td></td>
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<tr>
<td>Supper</td>
<td></td>
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</tr>
</tbody>
</table>
Please record:
- Everything you eat and drink today, including sweets, snacks, nibbles, sauces and dressings
- Method of cooking (e.g. boiled pasta)
- Type of food (e.g. boiled wholegrain pasta)
- Quantity of food (e.g. 100g boiled wholegrain pasta)

<table>
<thead>
<tr>
<th>Meal/Snack (Time of day)</th>
<th>Details of food and drink (Including cooking method)</th>
<th>Amount served</th>
<th>Amount left (If any)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early morning</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breakfast</td>
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<td></td>
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<tr>
<td>Mid-morning</td>
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<td></td>
</tr>
<tr>
<td>Lunch</td>
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<tr>
<td>Afternoon</td>
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<tr>
<td>Supper</td>
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</tr>
</tbody>
</table>

Have I written everything that I ate today??
Did I remember to include the oils and fats I cooked with??
What about the ‘drink’ I had before dinner……, with dinner…. after dinner….. and what about the one still in my hand….??