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northumbria UNIVERSITY NEWCASTLE

Multiple System Modelling and Analysis of Physiological and Brain Activity and Performance at Rest and During Exercise

DINESHEN CHUCKRAVANEN

A thesis submitted in partial fulfilment of the requirement of the University of Northumbria at Newcastle for the degree of Doctor of Philosophy

Research undertaken in the School of Life Sciences and in collaboration with the School of Computing, Engineering, and Information Sciences

May 2012

Declaration

I declare that the work which is contained in this thesis has not been submitted for any other award, and that it is my own work. All ethical approval procedures were followed, and the research work was conducted in the School of Life Sciences and in collaboration with the School of Engineering, Northumbria University, Newcastle, United Kingdom.

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Abstract

One of the current interests of exercise physiologists is to understand the nature and control of fatigue related to physical activity to optimise athletic performance. Therefore, this research focuses on the mathematical modelling and analysis of the energy system pathways and the system control mechanisms to investigate the various human metabolic processes involved both at rest and during exercise. The first case study showed that the PCr utilisation was the highest energy contributor during sprint running, and the rate of ATP production for each anaerobic subsystem was similar for each athlete. The second study showed that the energy expenditure derived from the aerobic and anaerobic processes for different types of pacing were significantly different. The third study demonstrated the presence of the control mechanisms, and their characteristics as well as complexity differed significantly for any physiological organ system. The fourth study showed that the control mechanisms manifest themselves in specific ranges of frequency bands, and these influence athletic performance. The final study demonstrated a significant difference in both reaction time and accuracy of the responses to visual cues between the control and exercise-involved cognitive trials. Moreover, the difference in the EEG power ratio at specific regions of the brain; the difference in the ERP components' amplitudes and latencies; and the difference in entropy of the EEG signals represented the physiological factors in explaining the poor cognitive performance of the participants following an exhaustive exercise bout. Therefore, by using mathematical modelling and analysis of the energy system pathways and the system control mechanisms responsible for homeostasis, this research has expanded the knowledge how performance is regulated during physical activity and together with the support of

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Glossary

ADP:	Adenosine Diphosphate
ATP:	Adenosine Triphosphate
ATP-PCr:	Adenosine Triphosphate - Phosphocreatine
BMI:	Body Mass Index
BPM:	Beats per Minute
GME:	Gross Mechanical Efficiency
RER:	Respiratory Exchange Ratio
RPE:	Ratings of Perceived Exertion
$\dot{V}O_{2max}$:	Maximum oxygen uptake
FD:	Fractal Dimension
RQA:	Recurrence Quantification Analysis
DET:	Determinism
TT:	Trapping Time
RR:	Recurrence Rate
CWT:	Continuous Wavelet Transform
DWT:	Discrete Wavelet Transform
PCr:	Phosphocreatine
PSD:	Power Spectral Density
EEG:	Electroencephalogram
ECG:	Electrocardiogram
ERP:	Event Related Potential
MFI:	Multidimensional Fatigue Inventory
VAS:	Visual Analogue Scale
RVIP:	Rapid Visual Information Processing
LF:	Low Frequency
ULF:	Ultra Low Frequency
HF:	High Frequency

Notations

dt:	change in time
ϵ:	is an element of
F:	statistics F-ratio
Hz:	hertz (frequency)
J:	Joules (energy)
K:	kelvin (temperature)
km:	kilometre
$kg.m^{-2}$:	kilogram per metre square
$kg.m^{-3}$:	kilogram per metre cube
$Lmin^{-1}$:	litres per minute
ms^{-1}:	metre per second
m:	metre
MJ:	Megajoules
ms:	millisecond
^{o}C:	degrees celcius (temperature)
$\mathbb{R}$:	real number
RR:	beat-to-beat interval
p:	statistics probability
r:	correlation coefficient
s:	second
S.D:	standard deviation
μV^2:	microvolt square
V:	volts
W:	watts (power)
Wkg^{-1}:	Watts per kilogram
$\sum$:	summation
$\int$:	integral

List of abstracts accepted, presentations, preparation for publication

- D Chuckravanen, M. Angelova, A St Clair Gibson, Thomas K, Stone M, Ansley L, Thompson K.G (2009), 'Recurrence Quantification Analysis of the System Control Mechanisms', Recurrence plots at the crossroad between theory and application: A flexible approach for studying complex systems, 3rd International Symposium on Recurrence Plots, Montréal, Québec, Canada. (Oral presentation)

- D Chuckravanen et al. (2009) 'The effect and complexity of various pacing strategies on the energy expenditure during a 20-km cycling time trial', First International Sports Science and Sports Medicine Conference Newcastle Upon Tyne, Br. J. Sports Med. vol. 43 (e2). (Poster presentation)

- D Chuckravanen, S Rajbhandari, M Angelova, A St Clair Gibson, L Ansley, KG Thompson (2009), 'Continuous Wavelet Analysis of Physiological Data for Various Pacing of a 20-km Time Trial, North East Postgraduate Conference, Newcastle University. (Poster Presentation)

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- Paper and Preparation for Journal of Cognitive Neuroscience with title 'Effect of an exercise bout on mental performance' (Unpublished paper);

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CHAPTER ONE

Introduction

1.1 Definitions

Exercise physiology, one of the main disciplines of sport and exercise science, is developed from its “parent”, physiology, which is concerned with the study of the function and characteristics of the living systems (Garland, 1994). The studies can range from the basic unit of organisms, for example the cell, to the more complex organs and organ systems such as the brain and circulatory system respectively. As the focus of physiology is, by definition, at the level of organs, and systems within systems, exercise physiologists therefore should not only know how the different parts of an organism work together to achieve a particular function, but also they should have a thorough understanding of how the human body responds to exercise and training (Bangsbo, 1996; Elia, 1992). Then, these concepts that are developed from exercise physiology can be used to train the athlete, and improve the athlete’s sport performance (Wilmore and Costill, 2005). One of the main factors that affect sport performance and involves all body systems, is exercise-induced fatigue which results from excessive exertion and leads to a decrease in bodily and mental functions (Chen et al., 2004; Gao and Chen, 2003; Wu et al., 2003). Therefore, one of the goals of exercise physiologists is to investigate how to delay the onset of exercise fatigue, or how to use efficiently the available metabolic resources in the body to complete a physical activity (Lambert

et al., 2004; Munir Che Muhamed, 2008; Noakes, 2000; St Clair Gibson et al., 2005).

1.2 Background

Sports performance, in an athletic context, is the pursuit of excellence where an athlete measures his or her performance quantitatively or qualitatively to advance towards his or her desired goal. In those physical activities where the result is measurable and defined, such as a race (*e.g.* time), a jump (*e.g.* height) or the maximum distance covered by an object (*e.g.* javelin throw, projecting heavy weights), the end result is quantifiable which makes it easier to monitor the progress of the athlete. Several physical aspects can influence sport performance. One of these aspects is the neuromuscular factor (Tsiganos et al., 2008) that arises from the relationship between the nervous system, and the musculoskeletal system (*e.g.* endurance, flexibility, genetics, muscular strength, reaction time, and training). Moreover, in many sports (*e.g.* running and cycling), the establishment of an effective rhythm will keep an athlete organised, and physically efficient for an excellent performance (Plagenhoef, 1985). Subsequently, this rhythm will impose a cadence on musculoskeletal activity, mental control as well as psychological factors. These psychological factors, for instance, can be self-motivation, level of alertness and mental acuity that are the product of a number of integrated factors like physical fatigue or other unrelated sport stresses (*e.g.* personal circumstances, environmental conditions) that are not within the athlete's personal control. The athlete is required to have the ability to adapt in these unexpected environmental factors. Another aspect is coaching and external support for the athlete that are important in providing assistance and direction (*e.g.* in terms of nutrition, sport technique, tactics and training) to the aspiring competitor for

success to occur (Bangsbo, 1996; Plagenhoef, 1985; Tsiganos et al., 2008; Wilmore and Costill, 2005).

1.3 Current issues in Exercise Physiology

The ability to exercise depends on various physiological systems. When these systems are incapable of withstanding such requirements of physical activity, fatigue may occur. In exercise physiology, various theoretical control models of fatigue have been proposed (Edwards, 1983; Hill and Lupton, 1923; Munir Che Muhamed, 2008; St Clair Gibson et al., 2004; Ulmer, 1996; Weir et al., 2006) with the aim of providing a clearer picture of the underlying mechanisms of fatigue either from energy or biorhythms perspectives. For instance, it has been suggested that muscle fatigue creates a momentary decrease in the performance capacity of exercising muscles, owing to a failure in sustaining a certain amount of expected force or power (Hargreaves, 2008). This muscle fatigue is also described as the sensation of tiredness with associated decrease in muscular performance and function (Abbis and Laursen, 2005; Hargreaves, 2008). These descriptions take into account the elaboration of fatigue from various disciplines which are part of exercise science arenas such as physiology, biomechanics and psychology. Several review articles have been published, over the past few years, in an attempt to provide a better insight into the nature of the physiological adaptations to exercise, and how these physiological adaptations results in a delay on the development of fatigue (Abbis and Laursen, 2005; Hargreaves, 2008; Lambert et al., 2004; Noakes, 2000; St Clair Gibson et al., 2005). However, these physiological models cannot explain exactly the cause and effect of fatigue on sports performance. These theoretical models will be elaborated further in the literature review chapter. The next section will explain the importance and the role

of mathematical modelling and analysis of physiological signals in exercise physiology.

1.4 Importance of mathematical modeling and analysis

Mathematics, especially in biology and medicine, provides a wealth of opportunities for mathematical modellers and analysts to help clarify the underlying mechanisms that control the physiological systems as well as to investigate the energy production and depletion from the various energy pathways (Weswick and Kearney, 2003). This is because the modelling process enables the mathematical modeller to focus on separating the essential from the inessential. A mathematical model can be used to investigate theories that are not easily amenable to experiment, and that are time consuming as well as costly to conduct. Hence, mathematics is a valuable tool in these areas to test out ideas or hypotheses (Sloan et al., 1996). In addition, it is also a concise but yet a powerful and popular language that allows both the hidden similarities and distinct features among different physiological systems to be discovered (Sloan et al., 1996).

1.5 Aims & Objectives

This research focuses on the mathematical modelling and analysis of the energy system pathways, and the system control mechanisms (present in the physiological systems) which are responsible for the regulation of homeostasis (*i.e.* balance or harmony within the physical body system) required to enhance sports performance. Various mathematical methods, commonly utilised in the biological and medical field (Higuchi, 1988; Rioul and Vetterli, 1991; Shannon, 1948; Zbilut et al., 1995) are used for modelling and analysis to see how the physiological systems dynamically interact and function. These mathematical

models are based on observations and data collection from multiple biological and physiological systems under laboratory conditions to understand the integrative control systems that create human behaviour and control physical activity. These predictive complex system control models and analysis can be used to improve our understanding of sports performance in terms of quantifiable and reliable measures. The objectives are elaborated in terms of research studies following a thorough literature review of the existing physiological control models of exercise fatigue. These are addressed further in the following section.

An in-depth knowledge into the underlying mechanisms of exercise fatigue seems beneficial to optimise sports performance (Lambert et al., 2004; Noakes, 2000). Therefore, in order to assess the cause and effect of the exercise-induced fatigue, this research focuses, firstly, on the modelling and analysis of the biological energy system pathways to investigate the energy expenditure during high intensity exercise of short duration and an endurance physical activity; and secondly to investigate the system control mechanisms that regulate the internal milieu of the physiological systems in an attempt to observe how the physiological systems dynamically interact and function.

The following objectives were devised to investigate these research goals using mathematical modelling and analysis of the physiological systems data in an attempt:

- To elucidate how the human organism regulates the amount and the rate of adenosine triphosphate (ATP) to better understand the cause and effect of exercise-induced fatigue on athletic performance during maximal exercise of short duration;

- To find out how pacing, for an endurance time-trial exercise, affects the energy production from the aerobic and anaerobic systems and the homeostatic disturbance (if ever) that pacing may cause to the human organism;
- To find out the nature and characteristics of the system control mechanisms that regulate homeostasis in the internal milieu of the human organism subjected to different pacing of a prolonged exercise for sustaining performance;
- To be able to understand how a potential central regulator paces the human organism during a physical activity, and how the physiological systems dynamically interact or function;
- To investigate whether there are finite metabolic resources in the brain by assessing cognitive performance, in terms of reaction time and accuracy of responses, while performing both cognitive and physical tasks to exhaustion.

1.6 Structure of thesis

The thesis is organised into nine chapters. Chapter One introduces the theme of this research, Chapter Two provides a thorough literature review of the existing exercise-induced physiological control models, the importance of energy system pathways and homeostatic control mechanisms followed by a description of mathematical techniques used in this research in analysing physiological signals together with their current uses. Chapters Three, Four, Five, Six and Seven consist of the 5 experimental case studies, based on mathematical modelling and analysis, to critically analyse the challenging physiological control models. Chapter

Eight provides a general discussion of the findings through a critical evaluation of the existing physiological control theories of exercise-induced presents in the light of the mathematical findings from the experimental case studies. The thesis concludes with Chapter Nine which emphasizes the significance and importance of this research with respect to previous findings and established mathematical and theoretical contributions to the current physiology and sports science research about the nature and control of exercise-induced fatigue. Before embarking on the literature review, the experimental case studies are briefly described in the following paragraphs. The first case study was based on the mathematical modelling and analysis of elite athletes' sprint data (from a 100-m race) which was obtained from the International Association of Athletics Federations (IAAF) to indirectly study the regulation and the rate of adenosine triphosphate (ATP) utilization during maximal exercise of short duration. A type of Gamma distribution (Hogg and Craig, 1978) was used to model the rate of production and decay of each anaerobic energy subsystem as it is a flexible model to represent exponential distribution, and a good fit for the sum of independent exponential random variables (Schmidt, 1985; Wlodarczyk and Kierdaszuk, 2006).

For the second case study, the energy expenditure of ten well-trained and healthy male cyclists was mathematically modelled and analysed for both aerobic and anaerobic energy system pathways during various pacing trials that were self pace, even pace and variable pace. These different types of pacing were used by the cyclists to complete 20-km cycling time trials, and all these trials were conducted in the physiology laboratory of the School of Life Sciences at Northumbria University. For studies 3 and 4, the same time trial data together with other biological variables (such as heart rate, volume of oxygen consumption and

blood lactate concentration) were analysed to investigate the control mechanisms underlying various physiological systems (*i.e.* the power outputs representing the integrative behaviour of the whole human body system, the cardiovascular system and the respiratory system).

The third experimental study was based on the mathematical analysis of the nature and characteristics of the system control mechanisms underlying the physiological data during the 20-km cycling time trials associated with pacing. In order to analyse these physiological control mechanisms, mathematical techniques, including fractal analysis and recurrence analysis were used to find any similarities, distinct features, or characteristics of the biological mechanisms that modulate human behaviour and physical activity. These specific mathematical techniques were employed since fractal analysis is a common technique used in physiology and medicine to find self-similarity of biological signals (Glenny, 1991; Tapanainen, 2002); and recurrence analysis is widely used as a graphical and quantification tool to detect shifts in physiological states and nonstationarities (Trulla et al., 1996; Zbilut et al., 1995). Furthermore, from the recurrence plot, several recurrence quantitative analysis measures were computed to find whether the physiological system activities being assessed were predictable, stable and resilient during physical activity. For the fourth experimental case study, a different mathematical method was utilized to investigate the system control mechanism to see how a central regulator theoretically paces the physical body during a 20-km cycling time trial exercise for various pacing trials. According to certain theoretical control models of fatigue, physical exercise is modulated by a central regulator within the brain. Therefore, this fourth study was conducted to find out how a central regulator paces the physical body during exercise. This was accomplished

by analysing the physiological data including power outputs, volume of oxygen consumption and heart rate using Continuous Wavelet Transform (CWT). CWT was used to produce a two-dimensional view of the physiological signal in order to observe and analyse the local and general characteristics as well as behaviour of the physiological signal (Mallat, 1989). Moreover, CWT has been described as an indirect way of assessing the functions of the central system (*i.e.* the brain and the brain stem) by observing any changes at different frequency bands of the biological signals (David et al., 2007).

According to certain theoretical models (Lambert et al., 2004; St Clair Gibson et al., 2004), fatigue is not always peripheral but rather is a result of brain function. Therefore, the fifth experimental case research study was designed and conducted to investigate whether there is a finite level of metabolic resources in the brain. This was achieved by performing both cognitive tasks (*i.e.* Rapid Visual Information Processing and Modified Stroop tests) and physical tasks to exhaustion with subsequent assessment of the effect of these exhaustive tasks on cognitive performance (this was evaluated in terms of reaction time and accuracy of responses to visual cues).

1.7 Contribution to knowledge

This research was important since through the mathematical modelling and analysis of the collected biological data, the various energy systems and the interactions between the physiological systems (peripheral and central systems) were mathematically assessed in an attempt to unlock the principle control of activity in these different physiological systems as well as their “hidden” properties

and characteristics that regulate physiological behaviour. Moreover, this research work endeavoured to unearth the nature and control of the exercise-induced fatigue in exercise physiology so as to improve the sport performance of athletes using a set of mathematical theories together with the existing biological control model theories.

1.8 Principal related works in this area

While a number of theoretical models have been produced in the field of exercise physiology, the following articles were specifically concerned in this research followed by articles describing currently and commonly used mathematical techniques in biology and medicine.

(i) Theory (Physiological control models)

- Hill and Lupton (1923)
- St Clair Gibson et al. (2004)
- Lambert et al. (2004)
- Weir et al. (2006)

(ii) Mathematics in biology and medicine

- Recurrence analysis works by (Webber et al.,1990) and (Zbilut et al., 1995);
- Wavelet transform (Rioul and Vetterli., 1991);
- Fractal analysis (Higuchi, 1988);
- Entropy (Shannon, 1948; Rosso et al., 2002).

CHAPTER TWO

Literature Review

2.1 Introduction

The study of complex regulatory systems controlling biorhythms and activity arising from physiological systems, and the availability of metabolic resources in a human body system is important for optimising and understanding athletic performance (Lambert et al., 2004; Manfredini et al., 1998; Murphy, 1996; St Clair Gibson et al., 2005). These physiological and metabolic signals constitute an enriched source of biological information where the application of linear statistical analysis may find correlations but may fail to explain the nature and characteristics of the control mechanisms responsible for these correlations (Ottesen et al., 2004).

When statistical analysis is merged with mathematical modelling and analysis of these dynamics, however, new insight into the nature of the physiological responses and control mechanisms may be revealed. Furthermore, in the long term mathematical models may help generate new mathematical and physiological theories of the control of physical activity and human behaviour (McSharry et al., 2005; Ottesen, 1997; Ottesen et al., 2004). Moreover, physiological and brain activities change irregularly in time (Bassingthwaighte, 1994; Glass, 1988; Keener, 1998; Winfree, 2001), mathematical methods, including nonlinear techniques, are required to model and analyse the system control mechanisms that regulate the

physiological homeostasis (*i.e.* there is balance in the biological activities within a particular physiological system), and control the energy contribution from the energy pathways required to sustain athletic performance (Bangsbo, 1996; Lambert et al., 2004; St Clair Gibson et al., 2005). The system control mechanisms are biological mechanisms or processes responsible for the regulation of any particular physiological system (Cannon, 1926).

In the next section, various theoretical models of fatigue, in an exercise physiology context, are described followed by a description of possible factors that influence sport performance, and how these can be optimised to improve sports performance. Subsequently, the mathematical techniques used in this thesis are elaborated as well as their importance to model and analyse biological systems is provided.

2.2 Evolution of theoretical exercise-induced physiological models

Various physiological models have been devised in the aim to understand the development of exercise-induced fatigue. Currently, the physiological theories to explain the cause and the effect of fatigue on exercise performance include the: (i) cardiovascular/anaerobic/catastrophic model, (ii) energy supply/energy depletion model, (iii) biomechanical model (iv) thermoregulatory model, (v) teleoanticipation model, (vi) integrative central regulator model, (vii) neuromuscular fatigue model, (viii) task dependency model and (ix) psychological/motivational model (Abbis et al., 2005; Hargreaves, 2008; Lambert et al., 2004; Noakes, 2000; St Clair Gibson and Noakes, 2004; Ulmer, 1996; Weir et al., 2006). Therefore, these various

theoretical models and theories that endeavoured to explain the development of fatigue during exercise are described in the following subsections.

2.2.1 Cardiovascular/Anaerobic/Catastrophic model

The cause of the exercise-induced fatigue which is developed during high intensity exercise of short duration is still unknown. From studies of Fletcher and Hopkins (1907) and that of Sir Archibald Vivian Hill, and colleagues (Hill et al., 1923; Hill, 1924), it was suggested that this form of exercise is limited by a peripherally-based metabolite that induced failure of the skeletal muscle contractile function independent of the reduction in skeletal muscle activation which is caused by the central nervous system (CNS) (Noakes, 2000).

The classical theory, also defined as the Cardiovascular /Anaerobic /Catastrophic (CAC) model of exercise physiology (Hill, 1923; Noakes, 2000), states that the fatigue associated with high-intensity exercise results from a skeletal muscle "anaerobiosis". This "anaerobiosis" is developed when the increasing oxygen demand of the exercising muscles cannot be supplied by the heart. Therefore, this inadequate oxygen supply to the exercising muscles prevented the neutralisation of the progressive accumulation of lactic acid that Hill (1923) believed would prevent skeletal muscle relaxation leading to skeletal muscle rigor (Hill, 1923; 1924; 1927; Noakes, 2000).

However, it is known that it is the depletion of adenosine triphosphate (ATP) within the exercising skeletal muscle that causes rigor (Fitts, 1994). This peripheral model also led to the "catastrophe theory" of Edwards (1983) which states that physical activity stops when the biochemical and physiological limits of the body

are surpassed causing a catastrophic failure of the mechanism responsible for intracellular homeostasis.

According to the cardiovascular/anaerobic/catastrophic model, during maximal exercise, fatigue occurs when the cardiovascular system is unable to provide the required amount of oxygenated blood to the active muscle and the waste products accumulate in the skeletal muscle. Therefore, endurance athletes, such as marathoners have superior aerobic capacity. This means that these endurance athletes can run at speeds that require them to utilise about 75% of their maximal oxygen uptake ($\dot{V}O_{2max}$) during a race at a high level of oxygen uptake (Wilmore and Costill, 2005). This feat is ascribed to the ability of the heart to pump large amount of blood as well as the ability of extracting oxygen at the exercising muscles (Noakes, 2000). Subsequently, if there is an insufficient amount of oxygenated blood at the working muscles, then this endurance athlete would rely primarily on anaerobic metabolism which would result in a higher blood lactate accumulation during physical activity. This happens because the rate of removal of blood lactate is relatively less than its rate of production (Brooks et al., 1996).

In addition, Brooks and his colleagues found that there was a good correlation between power output reductions in endurance sports exercise and increases in blood lactate concentrations. Findings from Lucia et al. (2002) showed that elite cyclists were not capable of maintaining high level of workload for prolonged duration when the blood lactate concentration in the active muscles was beyond the lactate threshold. Unfortunately, the accumulation of blood lactate caused a decrease in the intramuscular pH which prevented the phosphofructokinase (PFK) activity, which in turn reduced the release of calcium ions that consequently decreased force production within a skeletal muscle (Brooks et al., 1996).

2.2.2 Energy Supply/Energy Depletion model

The cardiovascular/anaerobic model is further extended into another model called the energy supply/energy depletion model that associates fatigue during exercise to the failure of the energy metabolic pathways to produce sufficient amount of energy (ATP) to the active muscles, or to the depletion of endogenous substrates such as carbohydrates (Gollnick et al., 1973; Shulman and Rothman, 2001). This energy depletion model is the direct result of the depletion of the fuel substrates that are muscle and liver glycogen when the physical exercise exceeds more than 2 hours (Noakes, 2000). This model highlights the importance of conserving energy production through energy pathways (*i.e.* the anaerobic system and the aerobic system) and incorporating a strategy to utilise efficiently the available metabolic energy resources during the exercise. Shulman and Rothman (2001) highlighted that athlete who had greater amount of glycogen in muscles at the start of the physical activity had an extended duration of exercise to fatigue by being more resistant to this exercise-induced fatigue.

2.2.3 Biomechanical model

Another model involving the biomechanics of the human body states that the role of muscles is regarded as elastic energy systems which work in the same principle as springs and torque producers during physical activity (Pennisi, 1997; Roberts et al.,1997). This model predicts that the more elastic is the muscle, the less torque this particular muscle is required to produce which in turn increases the efficiency of the muscle system. This will eventually enhance sport performance, especially in weight-bearing activities such as running, by decreasing the rate of substrates

accumulation that might induce exercise fatigue; and by decreasing the rate of rise of body temperature so as the physical body is delayed in reaching critical core temperature, which is responsible in preventing exercise activity (Noakes, 2000).

2.2.4 Thermoregulatory model

Environmental conditions greatly affect exercise ability (Cheuvront and Haymes, 2001; Nybo and Nielsen, 2001). The thermoregulatory fatigue model proposes that when the human body core temperature increases to a critical level of about 40^{o}C, fatigue occurs (Gonzalez-Alanso et al., 1999). This is because the central nervous system (CNS) has a reduced ability to maintain a constant neural drive (Nybo and Nielsen, 2001) at or above this critical temperature of 40^{o}C. During prolonged exercise, the beginning of hyperthermia (*i.e.* body temperature much above normal) is related to a reduction in cerebral circulation that results in a decreased supply of substrates or metabolic resources to and from the brain (Nybo and Nielsen, 2001).

When the body temperature increases, the skin blood flow increases together with an increase in sweating rate that creates a stressful environment for the circulatory system. When the skin temperature is increased, there is a decrease in mean arterial pressure, stroke volume, and total peripheral resistance together with an increase in heart rate (Rowell, 1986). The decrease in stroke volume as the skin temperature increases lead finally to a reduced cardiac output that eventually impedes the oxygenated blood supply to the active muscles. Moreover, Parkin and colleagues (1999) found there was a greater glycogen utilisation rate in heat stress (such as 40^{o}C) as compared to a colder environment (3^{o}C).

2.2.5 Teleoanticipation model

In contrast to the previous physiological models that focus on the efficiency of expenditure of the energy systems, Ulmer (1996) proposed a totally different hypothesis of the physical fatigue during exercise. He proposed a hypothetical model of a control system which optimised performance especially during heavy physical activity. This model was founded on a standard feedback control loop where efferent signals from the central nervous system (CNS) contain information to determine muscle metabolic rate and exercise intensity. Then, afferent signals from the muscles or peripheral organs, feedback information to change movement and power output in order to optimise sport performance accordingly. Couple with that, it was also proposed (Ulmer, 1996) that the model was more complex than this simple feedback control loop when taking into consideration the additional presence of endogenous reference signals, and factors such as training, muscle reserve, muscle metabolic rate and past experiences. This subsequently gave rise to the 'teleoanticipation model' which included both feed forward planning and feedback control from afferent changes, which are principally regulated by the known endpoint of an event and the distance still to be covered to complete that event.

2.2.6 Integrative Central Regulator model

Working from the teleoanticipation model, another model of integrative central neural regulation of effort and fatigue was proposed (Lambert et al., 2004; St Clair Gibson and Noakes, 2004). In this physiological control model, it was suggested that physical activity is regulated by a central controller in the brain, and that the human body functions as a complex system during exercise. In this model, a central regulator paces the body during exercise to make sure that the physical

activity is completed without homeostatic failure by sending and receiving information to and from central and peripheral sensors and body systems in a deterministic manner (Lambert et al., 2004).

2.2.7 Neuromuscular Fatigue model

The neuromuscular Fatigue model or the central fatigue model (Davis and Bailey, 1997) states that it is not the rate of supply of substrate (*i.e.* oxygen/fuel) to muscle that limits the performance during physical activity but rather the processes involved in the skeletal muscle recruitment. The nervous system is important in coordinating physiological responses in the human body and the neuromuscular fatigue model also suggests that there is a reduction in force or power output despite the fact that perception of effort increases. This reduction in force or power is suggested to be associated with central activation failure (*i.e.* a reduction of muscle activation by the CNS) or the neuromuscular propagation failure (i.e. the decreasing response of the muscle to an electrical stimulus). The decreasing ability of the muscle to respond to an electrical stimulus is due to the reduction in the muscle action potential called the M-Wave, or a decrease in the speed of conduction of action potentials to the working muscle. The reduction in M-Wave occurs when there is a decrease in ionic transmembrane gradient such as sodium and potassium ions (Fowles et al., 2002; Nielsen and Clausen, 2000). The studies that supported the idea that fatigue is linked to the CNS have also examined and found changes in the CNS neurotransmitter (serotonin and dopamine) concentrations in the brain during prolonged exercise that in turn was suggested to diminish the level of arousal (excitement) and skeletal muscle recruitment (Davis et al., 2000) which are altogether related to an increase in the perception of effort that eventually affects exercise performance. This neuromuscular fatigue model

also incorporates the peripheral fatigue theory which describes fatigue can occur at the muscle site and include failure of the excitation-contraction coupling mechanism (Behm and St-Pierre, 1997) which is the physiological process of converting an electrical stimulus to a mechanical response (Sandow, 1952). Therefore, this evidence demonstrates that the central nervous system fatigue model contributes to fatigue during prolonged exercise lasting tens of minutes to hours (Baker et al., 1993).

2.2.8 Task Dependency model

Following the neuromuscular model and the integrative central controller model, it was argued that various studies (Calbet et al., 2003; Edwards et al., 1995) indicated that decreases in performance, owing to exercise-induced fatigue, cannot be explained fully by any one of these models (Weir et al., 2006). Instead the concept of task dependency model was recommended as a suitable model of fatigue in which mechanisms of fatigue vary depending on the specific exercise and this model includes characteristics of the central and peripheral contributions to fatigue, and their relative importance depends on the type of exercise (Weir et al., 2006).

2.2.9 Psychological/Motivational model

This model postulates that the capability to maintain exercise performance comes from a conscious effort, and is frequently included as an additional component of the central fatigue model hypothesis (Davis and Bailey, 1997). However, this model does not support one principle of the muscle recruitment model which holds that performance during exercise is controlled at a subconscious level. The psychological/motivational model does not agree that the conscious brain can

override fundamental physiological functions and cause irreversible damage to the human body.

2.2.10 Summary of the physiological control models

Following the hypothesis of these physiological control models, it is clear that there is an uncertainty of these biological theories in successfully explaining the real cause of fatigue during physical activity. Therefore, this research focused on the mathematical analysis and modelling of the various physiological systems in the hope to observe the nature and characteristics of the system control mechanisms that are responsible for human behaviour and control physical activity during exercise. These mathematical modelling and analysis were performed on physiological data collected, under laboratory conditions, from athletes or club-level healthy participants at rest and during exercise. These mathematical modelling and analysis attempted to find the cause of fatigue during exercise as well as the nature and characteristics of the system control mechanisms responsible for homeostatic regulation of the physiological systems. In so doing, mathematical theories as well as physiological theories can be developed or extrapolated from the existing biological control theories to understand better the control of fatigue during exercise so as to boost athletic performance.

By taking into account the different factors that influence sports performance from the theories of these exercise physiological models, the next sections describe (i) the energy system pathways that are aerobic and anaerobic systems; (ii) the importance of pacing in competition; (iii) the relationship between pacing and ratings of perceived exertion (RPE); (iv) the importance and effect of biorhythms on sports performance; (v) the importance of homeostasis in physiological systems; (vi) the arousal state for optimum performance (cognitive load and

information processing), and (vii) finally the relevance and importance of mathematical methods, commonly used in biology and medicine, in the modelling and analysis of biological data in exercise physiology.

2.3 Energy system pathways and physical activity

To perform a marathon run or sprint, skeletal muscle is fuelled by one compound, (Gajewski et al., 1986) the adenosine triphosphate (ATP). ATP is an organic compound (with chemical formula $C_{10}H_{16}N_5O_{13}P_3$) which consists of high-energy bonds, and is utilized to transport energy to cells for biochemical activities such as muscle contraction through its hydrolysis (*i.e.* reaction with water) to ADP (adenosine diphosphate) (Atul et al., 2010). The human body stores a small amount of this energy currency which is sufficient to meet the energy demands of an all-out explosive exercise for a few seconds (Baechle and Earle, 2000; Wilmore and Costill, 2005). Therefore, the human organism is required to resynthesize ATP on a continual basis to meet the energy demands and there are several metabolic pathways that the body utilises for the replacement of ATP. Which particular pathway will normally predominate depends on the physical activity being performed. Firstly, the ATP-PCr (Adenosine Triphosphate-Phosphocreatine) system will produce about 5 to 8 seconds worth of energy in an all-out activity such as sprinting after the initial storage of ATP in the muscles is used up (Baechle and Earle, 2000; McArdle et al., 2000, Wilmore and Costill, 2005).

Then, for longer duration or activity, the lactic system predominates whereby carbohydrate is broken down and used to produce ATP in a metabolic process called the anaerobic glycolysis. The chemical reactions, during anaerobic glycolysis, take place without the presence of oxygen, and the by-product of anaerobic exercise is lactic acid (Brooks et al., 1996; Mole, 1983). This lactic acid

system can be the source of fuel to an athlete for about 45 seconds, and for exercise duration greater than this, the aerobic system will begin to predominate (Baechle and Earle, 2000; McArdle et al., 2000). For exercise duration extending more than 2 minutes, the body shifts slowly towards the aerobic metabolic energy pathway to replace the initial ATP stores by breaking down carbohydrate or fat (depending on various factors such as exercise intensity, diet and training) to produce ATP within the aerobic system. During the aerobic exercise, the human body has enough time to use oxygen in the biochemical reactions (Brooks et al., 1996).

2.3.1 The importance of pacing in sporting activities

The changes in the pattern of speed or velocity during a time-trial race (*e.g.* running), has drawn the attention of sport scientists and exercise physiologists to examine the concept of pacing (Abbis and Laursen, 2008; Foster et al., 1993; Tucker, 2009). The three well-known types of pacing are negative, positive and even pacing that depend greatly on the event duration and the consequences of slowing down because of power output reduction (Foster et al., 1993; Hettinga et al., 2006 and Tucker et al., 2006). It was posited that pacing during physical activity is developed as a preventive measure to optimize sports performance according to the limit of the individual physical or physiological abilities, and hence preventing their physiological systems from large fluctuations in homeostatic disturbances during an exercise bout (Lambert et al., 2004; St Clair Gibson et al., 2006; Tucker et al., 2006). It was suggested that changes in the concentration of intramuscular metabolites (Foster et al., 1993), body core or brain temperature and other physiological factors determine the power output during shorter competitions of 1 to 30 minutes duration. Then, subsequently the conscious brain

integrates all these factors together creating a conscious fatigue which can be measured using the ratings of perceived exertion scale (RPE) (Borg, 1998; Lambert et al., 2004; Ulmer, 1996), a psychological instrument, to measure sensations of fatigue.

2.3.2 Relationship between pacing and rating of perceived exertion (RPE)

In a perception-based model for exercise performance by (Tucker, 2009), it was suggested that changes in the homeostatic status, as represented by momentary rating of perceived exertion (RPE), enables changes in pacing strategy or power output in a responsive or anticipatory manner that were founded from pre-exercise expectations and feedback from different peripheral physiological systems.

It was shown that RPE increases linearly with the percentage of completed event (Faulkner et al., 2008; Joseph et al., 2008; Swart et al., 2009), and has also proportional relationship when plotted against percentage of exercise task completed (whether it is duration or distance). Altogether, these observations show that an athlete is always comparing how they feel at any instant during a competition with how they expected to feel at that particular instant. If RPE is more than expected at any moment during a physical activity, then the power output of the athlete will reduce to a point of giving up a competition. Otherwise, if RPE is less than expected, then their power output will increase accordingly and hence this process of regulating muscular power output via RPE appears to occur continuously throughout a time-trial exercise or an exercise bout. Thus, the regulation of muscular power output takes into consideration the proportion of the distance that remains to be completed and the momentary value of RPE (St Clair Gibson et al., 2006; Tucker, 2009). The next important factor which is described in

the next section is the influence of the biological rhythms that govern the human behaviour and physical activity.

2.4 The importance and effect of biorhythms on sports Performance

The examination of biorhythms or circadian rhythms is described as chronobiology which is defined as “the study of rhythm patterns in biological phenomena” (Manfredini et al., 1998). In chronobiology, the nature of these biorhythms patterns is identified within the German model, namely “zeitgebers” meaning time-givers, and the biorhythms are considered as external environmental cues. These external cues influence the ability of an athlete to adapt to daylight, seasons and time zone. The psychomotor, physiological, cognitive and psycho-emotional processes are all influenced by these biorhythms (Manfredini et al., 1998; Murphy and Cambell, 1996; O’Conner and Bensky, 1995; Seligman, 1990). Within the sports performance context, by understanding the patterns of the biorhythms and the energy flow within the human body, the factors that may deter an athlete’s optimal performance can be identified.

In the scientific community, biorhythms are considered as geophysical phenomena related with the rhythmic rotation of the earth on its axis as well as the shift from dawn to dusk (Murphy, 1996). It is known (Meyer-Bernstein and Morin, 1996) that the human biological clock is located within the hypothalamus, particularly in the suprachiasmatic nucleus (SCN). This theory suggests that light stimuli trigger the SCN through a process called phototransduction (that is, the light from photo receptors are transformed into an electrical potential. Then, the brain coordinates information among myriads of nerve fiber pathways. The influence upon athletic performance varies once the electrical impulse is transferred from the suprachiasmatic nucleus to these nerve fibers. This includes sleep, physical

activity, rest, adjusting the core body temperature, secretion of hormone; melatonin levels (light impedes melatonin secretion and hence causes drowsiness), biological drive, and psychological/emotional behaviours (Meyer-Bernstein, 1996; O'Conner, 1995).

Now, some common biological rhythms associated with human performance are explained. Firstly, the psychomotor rhythms may affect sport performance through the synchronization of neurotransmitters and motor neuron synthesis for both coordination and reaction time (Meyer-Bernstein and Morin, 1996; Murphy and Cambell, 1996). Secondly, the physiological rhythms may also influence performance owing to the synchronization process of neurotransmitters, the ability to generate ATP, the ability to develop lactic acid tolerance for speed as well as strength and power (Nielsen et al., 2001) and the elasticity of muscle fibers for flexibility (Pennisi, 1997; Roberts et al., 1997). Moreover, the physiological rhythms stress the heart to pump as efficiently as possible during endurance exercise (Manfredini et al., 1998, Wimmer, 2003). Thirdly, there are also cognitive rhythms which affect performance through memory and attention followed by psycho-emotional rhythms which affect sports performance such as pressures in a competition (Manfredini et al., 1998; Meyer-Bernstein and Morin, 1996; Murphy and Cambell, 1996).

There are other individual variables that may affect biorhythms including chronological age, eating habits, genetic predisposition, lifestyle, and overtraining (Roth et al., 1994; Seligman, 1990). These variables account for the individual peak performance of an athlete which has to be at an exquisite level for elite athletic performance. Most athletes who get exhausted during training sessions are experiencing the effect of the body and mind wanting to go back to rest (Roth

et al., 1994; Seligman, 1990). This affects ultimately their quality of psychomotor, physiological and cognitive rhythms which influences the athlete's potential for optimal performance. Thus, practice and training need to be held at multiple different times throughout the day for elite athletes to gain maximal recovery and high level of training efficiency on a continual basis (Roth et al., 1994; Seligman, 1990; Wimmer, 2003).

2.5 Importance of homeostasis within the human body system

An undesirable change in the internal conditions of the human organism could result in disease or death owing to the failure of homeostasis of the physiological systems. Homeostasis refers to the living system ability to maintain a stable set of internal conditions subjected to changes in the external or internal environment (Cannon, 1926). Examples of internal conditions are body temperature, blood pressure, and the composition of body fluids which must remain relatively stable for the correct functioning of the human body system. Therefore, in order to maintain homeostasis, an organism must react to its external environment by making internal adjustments which are activated by homeostatic reflexes (Sawka et al., 1988; Schmidt and Simon, 1982; Simon et al., 1986).

For instance, a simple example of homeostatic reflexes occurs when we stay outside on a hot day. If our body does not adjust to the heat, body temperature will rise to such a level that brain cells, may die at a high rate. However, homeostatic reflexes help sustain a constant internal body temperature. Therefore, when the solar heat strikes the skin, nerve endings acting as receptors sense this heat, and send a message to the brain (control centre) which then sends nerve impulses (efferent signals) that cause the blood vessels (effector), in the skin, to expand. The resulting increase in blood flow to the skin produces greater heat loss from the

skin surface. The brain also instructs the sweat glands (effectors) to increase production, because evaporation of sweat cools the skin (King, 2004). All these processes can be summarized in a simple feedback and feed-forward loop as shown in Figure 2.1 (King, 2004; Sawka et al., 1988; Schmidt and Simon, 1982; Simon et al., 1986).

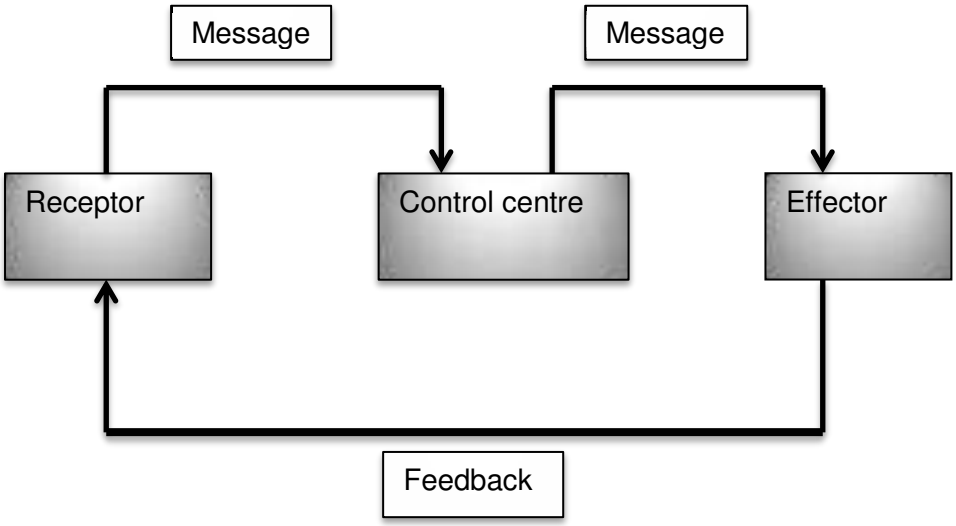

Figure 2.1: An illustration of the basic processes that happen during homeostasis (King, 2004; Sawka et al., 1988; Schmidt and Simon, 1982; Simon et al., 1986).

2.6 Arousal State and Sports performance

From certain physiological control models of exercise-induced fatigue and physiological studies, it was found that changes in the neurotransmitter concentration affect the level of arousal which subsequently affects the skeletal muscle recruitment during physical activity (Baker et al., 1993; Davis and Bailey, 1997; Fowles et al., 2002; Newham et al., 1991; Nielsen and Clausen, 2000).

The studies (Davis et al., 2000) that supported the idea that fatigue is linked to the central nervous system found in fact an increase in the dopamine and serotonin concentration in the brain during prolonged exercise. This, in turn, diminishes the

level of arousal and skeletal muscle recruitment (Davis et al., 2000; Polich et al., 1995) which are altogether related to a decrease in the perception of effort that eventually impoverishes exercise performance.

Furthermore, Yerkes and Dodson (1908) observed that as the arousal level increases, the physical and mental performance of an individual also increases accordingly but only up to a critical point called the optimal level (Figure 2.2), where performance is highest where ideally, an elite athlete is expected to reach. However, too much arousal will cause mental and physical performance to decrease like in cases of a stressful environment. Therefore, in order to optimise sports performance, research shall focus on investigating the energy system pathways of the human organism, the effect of pacing on these energy systems, the effect of the biological patterns on sport performance and lastly but not least the effect of the level of arousal on physical and cognitive performance.

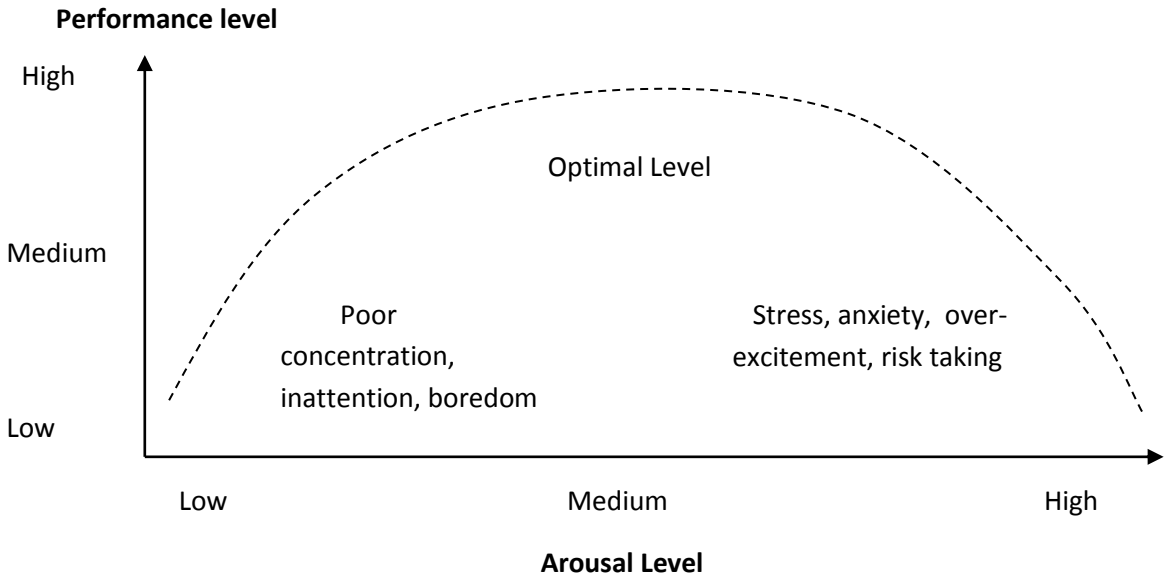

Figure 2.2: Level of performance as influenced by level of arousal (Yerkes and Dodson, 1908).

2.7 Importance of mathematics in biological systems

The importance of mathematics in biology or physiology is that it aims at the mathematical representation and modelling of biological processes. Furthermore, by describing the biological systems in a quantitative way, their biological behaviour can be simulated and their properties can be predicted which may not be an easy task to the biologist or physiologist (Baianu, 1987; Barnes and Chu, 2010; Goldbeter, 1996). Also recent development of mathematical tools can help understand the complex, nonlinear mechanisms in living systems as well as an increase in computing power can help perform lengthy or difficult calculations and simulations quicker (Eckmann, 1987; Marwan et al., 2007; Marwan, 2008). Therefore, the following sections will describe the importance and the workings of the mathematical tools that are currently used in medicine and biology and applied in this research to probe into the functioning of the various physiological systems in order to investigate the cause and control of fatigue during physical exercise.

2.7.1 Deterministic and stochastic signals

In order to understand the regulation of physiological systems, it is necessary to differentiate between deterministic and stochastic signal as the common characteristics of biological activities (Kac and Logan, 1976; Nelson, 1985; Priplata et al., 2006) A signal is said to be deterministic if its future values can be produced according to a set of known parameters and rules (Najim et al., 2004). For example, a deterministic cosine signal $y_d(t) = \cos(2\pi ft)$ can be predicted accurately based on condition that its frequency f is known (subscript d stands for deterministic and t is time). In order to distinguish between a deterministic outcome and a stochastic outcome, two cases are considered to elaborate this.

For example, if an output signal $y_r(k)$ is generated by repeatedly tossing an unbiased coin, there is no way to predict the k^{th} outcome of the output accurately, even if all the output values (head or tail) are known (subscript r in stochastic outcome $y_r(k)$ stands for random). These represent two distinct cases: $y_d(t)$ is purely deterministic while $y_r(k)$ is random or stochastic (Najim et al., 2004). The following sections describe the mathematical techniques used in this thesis, and their current applications in biology and medicine.

2.7.2 Fractals in biological systems

Fractal dimension (FD) is a means to measure the complexity of a physiological signal whereby the more complex a biological signal is, the higher the FD value. For instance, the fractal dimension of a single point is 0, a simple curve (line) is 1 and a plane is 2. In order to investigate the complexity of biological signals, fractal analysis using Higuchi's theorem (Higuchi, 1988) can be used to determine the fractal dimension. The Higuchi's fractal dimension, D_f, can be calculated directly from the real-time physiological signals and for instance, a curve such as the Koch Snowflake (Figure 2.3) has a fractal dimension 1.26. The figure 2.3 (Addison, 1997; Edgar, 1990) shows how the Snowflake is constructed. Level 0 starts with a straight line and then in level 1, the line is subdivided into 4 parts and in level 2, each part is again subdivided into another 4 parts and this procedure is repeated to create a complex fractal signal. The following paragraphs describe how the fractal dimension using the Higuchi's theorem works.

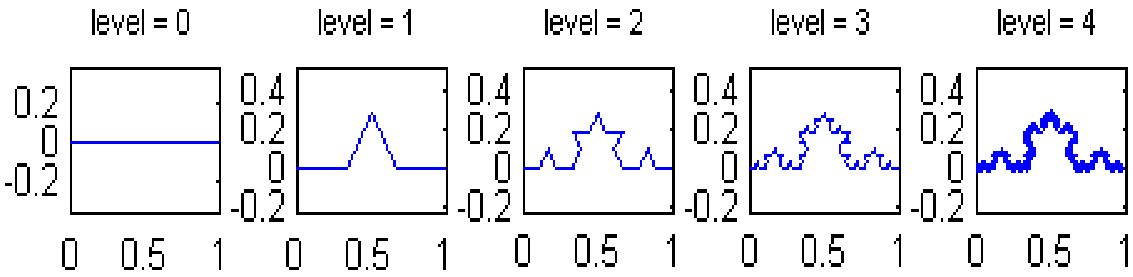

Fig. 2.3: The simulated Koch Snowflake is an example of fractal curve and has fractal dimension 1.26 (Addison, 1997; Edgar, 1990) starting with level 0 (extreme left) and increasing to level 4 (extreme right).

From a given time series X(1), X(2)... X(N), the algorithm constructs k new self-similar (fractal) time series X(k, m) as: X(k, m) = { X(m), X(m + k), X(m + 2k), ..., X(m + int [(N-m)/k] ·k) } for m = 1, 2, ... k where int [.] is an integer function. The length L_m (k) is computed (Equ. 2.1) for each of the k time series or curves X (k, m), and then averaged for all m forming the mean value of the curve length L (k), for each k (Higuchi, 1988). Then the fractal dimension (FD) is determined as the slope of least squares linear best fit from the plot of log (L(k)) versus log (1/k). This is summarized in Figure 2.4.

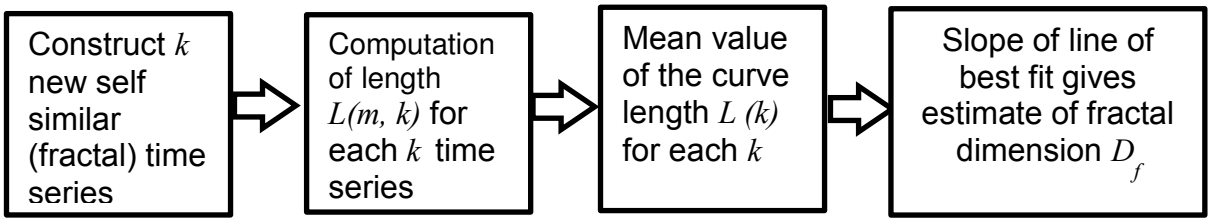

Fig. 2.4: Flowchart depicting the sequential steps involved in Higuchi's algorithm for the computation of fractal dimension (from left to right).

$$L_m(k) = \frac{1}{k}\left[\sum_{i=1,int\,(\frac{N-m}{k})} | X(m+ik) - X(m+(i-1)k| \left(\frac{N-1}{int\,(\frac{N-m}{k})}\right)\right] \ldots \text{ Equ. 2.1}$$

In equation 2.1, the length, $L_m(k)$, of each curve X^k_m is calculated and m represents the initial time with $m = 1, 2 \ldots k$ and, *k* is time interval; $int(r)$ is the integer part of a real number r and N represents the total number of samples. The length of the curve (Equ. 2.2) for a given time interval k, $L(k)$, is calculated as the mean of the k values $L_m(k)$ for $m = 1, 2 \ldots k$.

$$L(k) = \frac{\sum_{m=1}^{k} L_{m\,(k)}}{k} \quad \ldots \text{ Equ. 2.2}$$

Apart from determining the complexity of the signals in the hope to find any properties of the physiological activities that may be associated with the cause or control of exercise-induced fatigue, the wavelet analysis mathematical method is used to probe into the time and frequency analysis of the biological signals.

2.7.3 Wavelet Analysis

The time-based and frequency-based analysis of dynamic biological data are not suitable (Mallat, 1989) to analyse the nonstationary and irregular patterns of the complex physiological signals, Continuous Wavelet Transform (CWT) operates at every scale (ranging from 1 to 256) and time-position (Rioul and Vetterli, 1991) to observe changes (if any) at different frequency bands of the biological signals.

The ability of continuous wavelet transform to perform a time-scale analysis of dynamic data, and detect singularities (single points or lines) is an important tool in the analysis of non-stationary and fractal signals (Daubechies, 1992; Rioul and

Vetterli, 1991). A wavelet is a waveform of short period, and it has a mean amplitude value of zero over that duration, and therefore CWT splits up a signal into scaled and shifted versions of the original or *mother* wavelet, unlike the Fourier transform that breaks up a signal into a series of sine waves.

For instance, the Morlet wavelet, as shown in Figure 2.5, corresponds to higher frequency, and it can be observed more than the other wavelet families as well as it is commonly used in analysing biological signals (David et al., 2007). Wavelet analysis generates a time-scale view of a signal whereby scaling a wavelet signifies to compress or to stretch it, and it is usually denoted by scale factor a (Mallat, 1989). A low scale value a, representing a compressed wavelet, detects high frequency details of a signal while a high scale a detects low frequency details of the signal which is depicted in Figure 2.6. The relationship between the pseudofrequency (Hz) (this is a general theory connecting time-frequency representations) and the scale factor a is shown (See Appendix A, Figure A.1). The Continuous Wavelet Transform of a real time signal x(t) at a scale $a \geq 1$ and translational value b ϵ $\mathbb{R}$ (where $\mathbb{R}$ *represents real numbers*) is denoted as $X_w(a, b)$ and is shown below (Equ. 2.3) where $\Psi^*(\ )$ is the wavelet function and t is time where the integral to infinity means over the whole range of the real-time signal x(t).

$$X_w(a, b) = \frac{1}{\sqrt{a}} \int_{-\infty}^{\infty} x(t) \Psi^*\left(\frac{t-b}{a}\right) dt \qquad \text{… Equ. 2.3.}$$

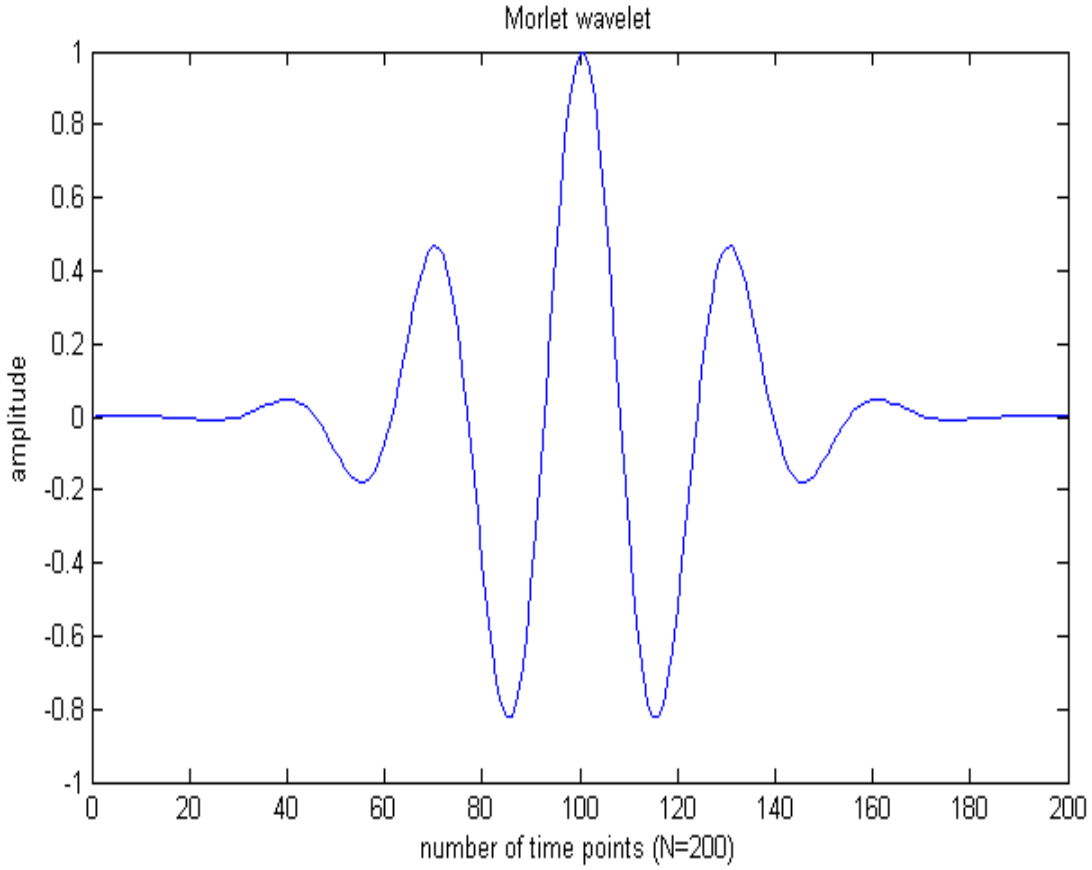

Figure 2.5: Morlet wavelet shape used for the CWT

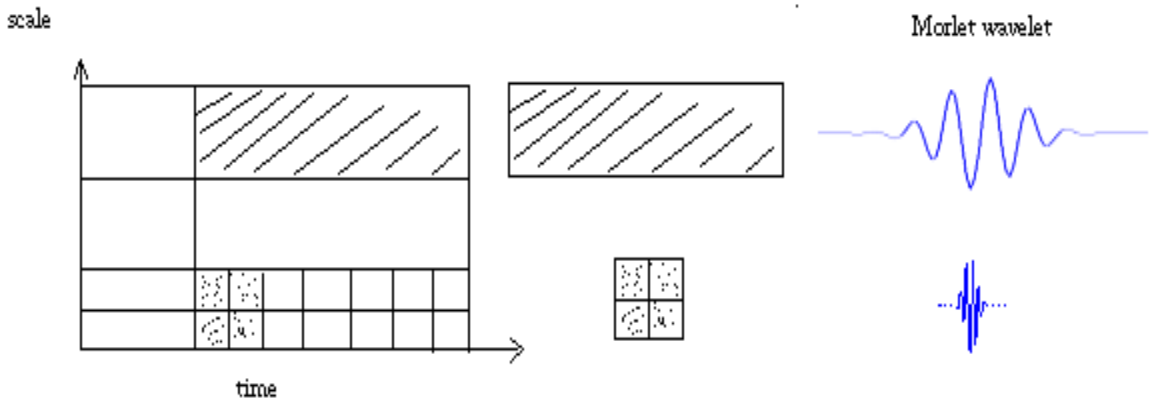

Figure 2.6: Illustration to show how the wavelet transform performs local analysis.

As shown in Figure 2.6, at high scale, the Morlet wavelet is dilated in the x-axis (representing time) to detect changes in low frequencies of a particular signal (the

top curve) and at low scale, the Morlet wavelet is compressed in the x-axis direction to detect changes in high frequencies of that signal and y-axis represents the scale. The continuous wavelet transform of a real time physiological signal enables changes in time, frequency and amplitude to be easily observed so that the frequency and intensity of happening of physiological events together with their respective times can be monitored to see how these changes are related to sport performance. The next subsection describes recurrence analysis a different mathematical method which was implemented in this research and it was used to investigate the behaviour of the physiological signals so as to understand better the characteristics of the system control mechanisms that regulate physical activity.

2.7.4 Recurrence Analysis to show patterns of complex system

In chaos theory, a recurrence plot (RP) is a graphical plot that shows for a particular moment in time, the times that a phase space trajectory travels about the same area in that phase space (Findlay 1911). This means it is a graph of:

$$\vec{z}\ (i) \approx \vec{z}\ (j) \quad \text{... Equ. 2.4}$$

In equation 2.4, i represents the horizontal axis and j represents the vertical axis, and $\vec{z}$ is a phase space trajectory.

For instance, biochemical processes may have a distinct recurrent behaviour that is periodicity (Eckmann et al., 1987). In addition, the recurrence of states (states are again close after some divergence time, is an important property of deterministic as well as dynamical systems and is typical for biological systems.

The recurrence of states in nature existed for a long time (Poincaré, 1900) and Eckmann et al., (1987) showed that from recurrence plots, one can observe the periodicity of states in a phase space (Figure 2.7). The phase space allows the representation of the behaviour of the physiological signal in geometric form and how the dynamics of this physiological signal evolves with time.

However, a phase space does not have a low dimension such as two or three to be easily visualized and higher dimensional phase spaces can only be observed by transforming them into a lower dimension such as 2 or 3 (Webber Jr. and Zbilut, 1994; Zbilut and Webber Jr., 1992). Therefore, recurrence plots allow the observer to analyse m-dimensional (multi-dimensional) phase space trajectory via a 2-dimensional representation of its recurrences. A recurrence of a state at time i and a different time j is viewed as a two-dimensional matrix with white and black dots respectively (time j is not a recurrent state of time i). This representation of states is named a recurrence plot (Zbilut and Webber Jr., 1992). The first order recurrence plot (RP) of a raw cosine function is shown in Figure 2.7 together with the corresponding phase-space plot and the effect of increasing the embedding dimension, m, of the recurrence plot is simulated in (See Appendix A, Figure A.2). By moving to a higher dimension, one may misinterpret the behaviour of a system. Several important quantification measures that were used in this research study were determined from the properties of the first-order recurrence plots. These measures are recurrence rate, determinism and trapping time. The equations of these parameters were developed (Webber Jr. and Zbilut, 1994; Zbilut and Webber Jr., 1992). The recurrence rate (RR) represents the percentage of recurrence points (black dots) in an RP or the probability that a specific state will recur.

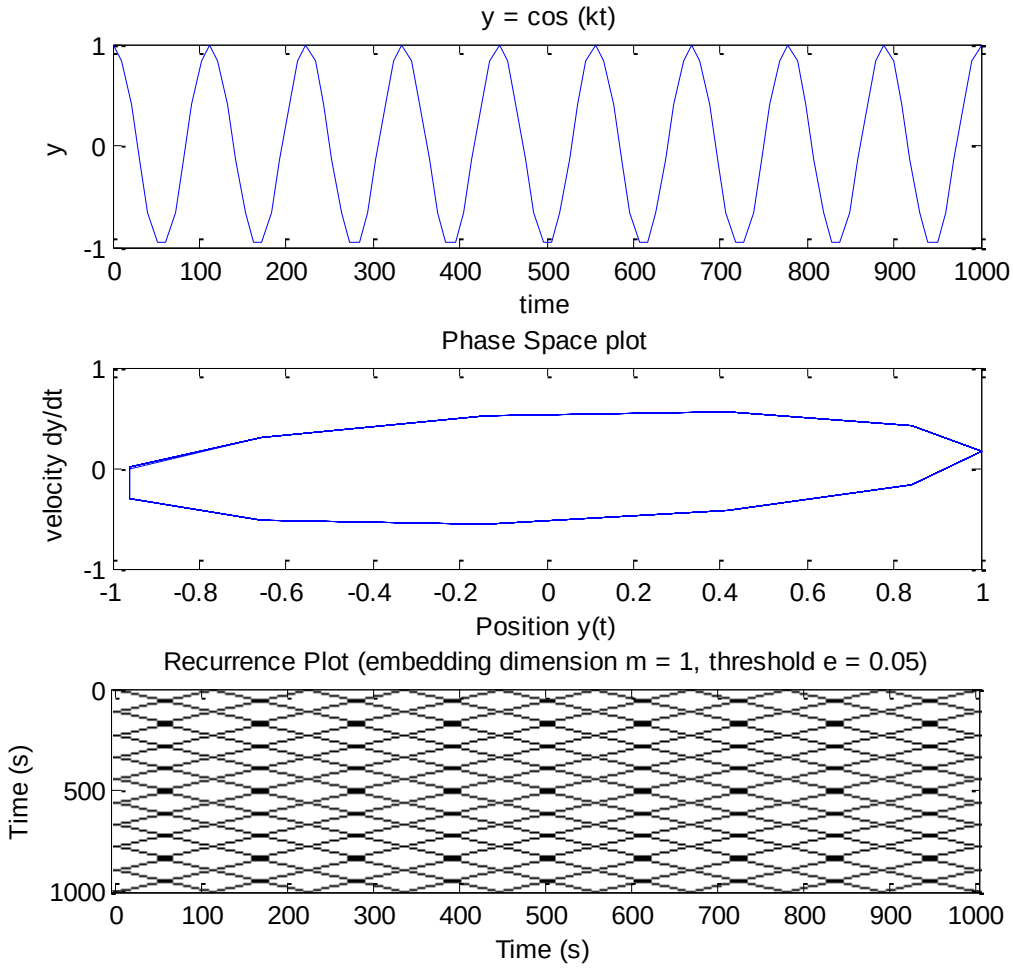

Figure 2.7: This figure shows a simulated raw cosine signal (top), its phase plot (middle) and its corresponding recurrence plot 1st order and threshold e being 0.05 (bottom).

In equation 2.5, R (i, j) represents all the recurrence points on the recurrence plot with notation i representing states in the x-axis and notation j representing states in the y-axis, and the variable N represents the total number of recurred points that appear in the recurrence plot.

$$RR = \frac{1}{N^2} \sum_{i,j=1}^{N} R\ (i,j) \qquad \text{...Equ. 2.5}$$

The next measure is the percentage of points that form diagonal lines in the

recurrence plot of minimal length l_{min}. $P(l)$ represents the frequency distribution of the diagonal lines of lengths l, and this measure is known as determinism which is related to the predictability of the dynamical system. For example, white noise has a recurrence plot with practically only dots and very few diagonal lines (Zbilut and Webber Jr., 1992; Webber Jr. and Zbilut, 1994), and the deterministic process has a recurrence plot with some single dots but many lengthy diagonal lines. Determinism is represented by DET as shown in the equation below (Equ. 2.6):

$$DET = \frac{\sum_{l=l_{min}}^{N} \boldsymbol{l \cdot P}(l)}{\sum_{i,j=1}^{N} R(i,j)} \quad \text{…Equ. 2.6}$$

Furthermore, the trapping time (TT) of a dynamical system (Equ. 2.7) is a measure of how long this system remains in a specific state and it is a measure of the average length of the vertical lines of the dynamical system. $P(v)$ is the average length of the vertical lines, v_{min} is the minimum length of the vertical lines and v is the length of the vertical lines.

$$TT = \frac{\sum_{v=v_{min}}^{N} v \cdot P\,(v)}{\sum_{v=v_{min}}^{N} P(v)} \quad \text{…Equ. 2.7}$$

The mathematical measures, described before, are commonly used in assessing the complexity of the biological signals (Webber and Zbilut, 1996), the occurrence of physiological events both in time and frequency domain as well as the predictability and stability of the physiological systems. Furthermore, there are specific mathematical measures or methods that are currently used to assess brain signals and these are described in the following sections.

2.7.5 Electroencephalogram (EEG) power at characteristic frequency ranges

Electroencephalography (EEG) measures the voltage fluctuations within the neurons of the brain, and it is normally recorded for a short period of time

(between 20 to 40 minutes) from various electrodes that are attached to the scalp (Niedermeyer and da Silva, 2004; Nunez and Srinivasan, 1981). EEG is a valuable tool for research and diagnosis (Abou-Khakil and Musilus, 2006) with the advantage of millisecond-range temporal resolution which is not possible with X-ray computed tomography (CT) or magnetic resonance imaging (MRI) (Niedermeyer and da Silva, 2004). Another important feature with EEG activity is that it oscillates at characteristic frequency ranges which are associated to different states of the brain function (*e.g.* waking and sleeping). For instance, attention related studies within the sporting context by Baumeister et al. (2008) revealed that expert golfers had higher fronto-midline theta band power (power of the EEG frequencies ranging from 4 to 8 Hz), and higher parietal alpha power (power of the EEG frequencies ranging from 8 to 12 Hz) as compared to novices (Aurlien et al., 2004; Tatum et al., 2008). This was associated to focused attention and an economy in the parietal sensory information processing that gave rise to more successful putting performance of expert golfers. Several researchers associated frontal theta power values with attention and the parietal alpha band power to the somatosensory information processing (Gevins et al., 1997, Slobounov et al., 2000; Baumeister et al., 2008). Moreover, Smith et al. (1999) stated that increased frontal theta power was associated with task complexity and focused attention while decreased parietal alpha power was related to increased information processing in a cognitive and visuomotor task owing to a higher activity of neural populations in the somatosensory cortex. Therefore, in this research, the frontal theta power and the parietal alpha power were investigated as well as the ratio of power of these two EEG frequency ranges characteristics was determined and named the 'cognitive ratio'.

2.7.6 Event Related Potentials (ERPs)

The event-related potentials represent the averaged EEG or brain responses that are time-locked to the presentation of a stimulus (*e.g.* visual or auditory). This technique is employed typically in cognitive science and psychophysiological research (Chapman and Bragdon, 1964). In this research, the ERP components N100, P200 and P300 were assessed as they are related to the cognitive functions. In neuroscience, the N100 is a large negative-going evoked potential and occurs around 80 to 120 ms after the triggering of a stimulus over the fronto-central region of the scalp. The N100 ERP component is linked to a person's arousal (Nash and Williams, 1982) and selective attention (Hillyard et al., 1973). Moreover, the P200 ERP component (a positive going electrical potential varying between 150 to 275 ms) represents higher-order perceptual processing which is regulated by attention and it is elicited as a normal response to visual stimuli (Luck and Hillyard, 1994). The P200 is widely studied in relation to visual search and attention as well as cognitive matching system by comparing sensory inputs with stored memory (Freunberger et al., 2007; Furutsuka, 1989). Finally, the P300 component, a positive-going brain potential occurring at around 300 ms, is normally linked to a person's reaction to a stimulus, engagement of attention, and processes in evaluating or categorizing a stimulus (Polich, 2007). Moreover, the P300 ERP component has maximum amplitude over the frontal, central and parietal brain areas (Polich, 2003; 2007). Therefore, the presence, magnitude and latency of these ERP components were used as metrics in this research for cognitive functions. The next section describes a common mathematical method currently adapted to clinical research in assessing the level of consciousness and information flow based on the EEG oscillations.

2.7.7 Shannon Entropy and Beyond

Certain physiological control of exercise fatigue (Davis and Bailey, 1997; Noakes, 2000) states that there is a conscious neural effort that maintains physical performance. Therefore, to investigate this hypothesis, the information entropy or simply entropy was used in this research. The entropy of a signal is becoming an emerging and promising mathematical method in analysing the loss of consciousness and quantifying the amount of information flow especially in the electroencephalogram (EEG) signals. The current clinical uses of the entropy principles involve the monitoring of anaesthetic depth by assessing the loss of consciousness using both the degree of spatial and temporal integration of neuronal activity in the brain (Bruhn et al., 2003). The following paragraph describes the mathematical principles of entropy.

Actually, information entropy was initially developed by Shannon (1948) to measure and evaluate the information content of a transmitted communication signal. Therefore, the entropy (H) is defined as the average amount of information per source output and is expressed by following equation:

$$H = - \sum p_i \log_2 p_i \text{ (bits/source output)} \qquad \text{…Equ. 2.8}$$

Where p_i represents the probability of occurrence of the i^{th} output, $\log_2$ is the logarithm function to base 2, and the summation $(\sum)$ of all the probabilities is equal to 1. From the entropy measures together with the EEG and ERP metrics, it was hoped to be able to observe any changes or associations in the brain activities with cognitive performance.

2.8 Summary

The fatigue which is developed during exercise seems to be a 'blackbox' phenomenon which exercise physiologists are trying to solve by creating physiological control models based on various physical and physiological observations or factors. Some of these models have some common principles while other models totally conflict with the principles of the cause and control of fatigue during physical activity. This thesis, therefore, attempted to unlock the nature and cause of fatigue by investigating the energy systems and the control mechanisms that regulate and sustain homeostasis in the physiological systems using mathematical methods that are currently used in medicine and biology. The following chapters describe the five experimental case studies, based on different mathematical techniques, which were used to model and analyse different physiological systems in the hope to answer the objectives and aims of this thesis in understanding better the principles or mechanisms responsible for the exercise-induced fatigue.

CHAPTER THREE

Study One

Mathematical modelling and analysis of elite athletes' sprint data to study the rate and regulation of ATP during a maximal exercise of short duration.

According to the review of literature of the existing biological control models of exercise-induced fatigue that the cardiovascular/anaerobic/catastrophic model (Edwards, 1983; Hill et al., 1923; Hill, 1924) and the energy supply/energy depletion model (Shulman and Rothman, 2001), it is not clear how the depletion of substrates (adenosine triphosphate) affects sprint performance. Therefore, this experimental case study was conducted to find out how the human organism regulates the amount and the rate of adenosine triphosphate so as to observe how these factors affect performance specifically during a maximal exercise of short duration.

3.1 Introduction

The energy which is produced during anaerobic metabolism has been of growing interest to exercise physiologists in order to understand how this specific energy pathway affects sprint performance or high-intensity exercise of short duration (de Koning JJ et al., 2011; Hill et al.,, 1923; Lambert et al., 2004). Despite there has been quite a bit of physiological laboratory work during short duration maximal exercise (Bogdanis, 1996; Gaitanos et al. 1993), most proposed theoretical models (Edwards, 1983; Hill et al., 1923; Hill, 1927; Noakes, 2000; Ulmer, 1996), however, cannot explain how the human body controls the rate of adenosine triphosphate (ATP) production to prevent a severe fall in ATP concentration in the active muscles.

Therefore, in this study mathematical modelling and analysis was used in an attempt to validate certain physiological theories of control fatigue. In fact, a large fall in ATP levels occurs during times of maximal exercise of short duration such as sprinting (Fox et al., 1993; Matthews et al., 1971; Mackenzie, 1998). To date, various research studies have incorporated single equation models to analyse the anaerobic metabolism (Di Prampero et al., 1993; Laurent and Locatelli, 2002; Lloyd, 1967; Peronnet and Thibault, 1989; Ward-Smith, 1985; Ward-Smith and Mobey, 1995). However, one study (Ward-Smith and Radford, 2000), has tentatively developed a mathematical model to represent the biochemical processes during the anaerobic metabolism based on several assumptions. They considered the total finishing times or duration of the sprints using a fourth-order Runge-Kutta mathematical method whereby temporal information of what is happening at discrete time intervals was lost and, by taking the height of the centre of mass of all sprinters who participated in the sprinting event to be equal, which was not the case according to their different weights and heights (Ferro et al., 2001; IAAF, 2008). Based on these assumptions, they found that the overall maximum anaerobic power of the sprinters for the 100m event at the 1987 World Championships was 51.6 Wkg^{-1}. The oxygen independent glycolysis, being the highest contributor of energy, was 11.7% greater than the energy derived from phosphocreatine utilisation anaerobic energy subsystem.

In order to extend the previous models, the aims of this experimental study were, therefore, firstly to develop mathematical models to determine indirectly the rate of ATP production and utilisation through the anaerobic subsystems that are endogenous ATP (*i.e.* ATP initially stored in the exercising muscles), Phosphocreatine (PCr) utilization and oxygen-independent glycolysis. Secondly,

this research aimed to assess how the anaerobic subsystems can be exploited further to improve high intensity short duration sporting activities such as sprint performance.

3.2 Method

3.2.1 Data collection and preliminary calculations

In this experimental case study, the International Association of Athletics Federations (IAAF) 10-m split times, for the Men's 100-m Final at the 1999 world championships, in Sevilla Spain, were used to model mathematically high-intensity exercise of short duration (Table 3.1) to investigate the elite athletes' sprint performance. In addition, the professional level of these sprinters would represent a good baseline for comparison purposes of the anaerobic subsystems and aerobic system at a track and field event. This mathematical model was then used to investigate the availability of metabolic resources, as well as the rate of energy production among the elite athletes.

The mean (± standard deviation) height, mass and body mass index (BMI) of the athletes were 1.78 (±0.03) m, 75.8 (±6.6) kg and 23.8 (±1.5) $kg \cdot m^{-2}$ respectively (IAAF, 2008). Each elite sprinter's height, weight and reaction times was used to mathematically model the energy systems. The wind speed was $+0.2 ms^{-1}$, the air temperature was $27^{o}C$ (300.15 K), air density was 1.179 kgm^{-3} and the mean reaction time of the sprinters was 0.141 (±0.01) s.

Sprinter ranking number	Distance covered/m									
	10	20	30	40	50	60	70	80	90	100
	Time elapsed for each 10m interval/ s									
1	1.86	2.89	3.81	4.69	5.55	6.39	7.24	8.09	8.94	9.80
2	1.88	2.88	3.79	4.68	5.53	6.38	7.24	8.10	8.96	9.84
3	1.87	2.89	3.81	4.71	5.57	6.41	7.29	8.18	9.07	9.97
4	1.91	2.93	3.85	4.76	5.63	6.50	7.36	8.24	9.12	10.00
5	1.87	2.89	3.81	4.71	5.60	6.37	7.33	8.22	9.11	10.02
6	1.91	2.95	3.88	4.77	5.65	6.52	7.39	8.28	9.16	10.04
7	1.91	2.93	3.85	4.74	5.62	6.51	7.40	8.28	9.17	10.07
8	1.97	2.99	3.93	4.83	5.72	6.61	7.50	8.38	9.31	10.24

Table 3.1: 10-m split data intervals for the 100m sprint race in Sevilla Spain 1999. (Courtesy Ferro, A.; Rivera, A.; Pagola, I.; Ferreruela, M.; Martín, A.; Rocandio, V. "Biomechanical Analysis of the World Championships in Athletics Sevilla'99: 100, 200, 400m sprint events". New Studies in Athletics, 16 1\2 (2001)).

3.2.2 Data analysis

All computations were performed using Matlab software R2008a as the programming platform as well as optimization toolbox together with Microsoft Excel 2007 for data handling purposes.

3.2.2.1 First Law of Thermodynamics

The mathematical equations used in this study were based on the First Law of Thermodynamics (Lehninger, 1971); At the start of a sprint, the rate of the chemical energy production is converted to heat energy (H) and external work energy (W). The rate of change of energy is expressed per unit body mass (Wkg^{-1})

and it is written in differential form (Equ. 3.1) where the left term represents the rate of chemical energy conversion (C), and the first and second terms on the right hand side are the rate of heat energy (H) and external mechanical work (W) respectively. The rate of heat energy is proportional to the instantaneous velocity $v(t)$ (Ward-Smith and Radford, 2000).

$$\frac{dC}{dt} = \frac{dH}{dt} + \frac{dW}{dt} \quad \text{… Equ. 3.1}$$

Furthermore, the rate of external mechanical work is expressed as the sum of the rate of change of kinetic energy of the sprinter to move forward; the potential energy of the sprinter relative to his crouching state at the beginning of the race; and the work done to overcome aerodynamic drag. The parameters for each of the energy components for the external mechanical work can be determined using already developed equations (Laurent and Locatelli, 2002; Ward-Smith and Radford, 2000) (See also Appendix B, section B.1).

3.2.2.2 Rate of change of potential energy relative to crouching state

For a typical athlete (Baumann, 1976), the centre of mass is raised from its initial position (h_o) of 0.65m in the blocks to about 1.0 m which was assumed to be the centre-of-mass height (h_{cm}) of a standing athlete, and was used same for all athletes for analysis (Laurent and Locatelli., 2002; Ward-Smith and Radford, 2000). Therefore, the change in height (Δh) of the centre-of-mass of the sprinter (Baumann, 1976) above the horizontal running surface relative to his crouching state position is given by equation 3.2.

$$\Delta h = (h_{cm} - h_0)\sin\theta \quad (where\ \theta \neq 0) \quad \text{… Equ. 3.2}$$

In equation 3.2, the angle θ, measured in radians, can be expressed further (Mitra, 2006) as $2\pi ft$, where f, in this case, is the stride frequency which is equal to the number of stride cycles per second, and variable t is the time measured in seconds. It was also shown that the stride frequency is well estimated by taking the inverse of the stride period (frequency is inversely proportional to time) (Stokes, 1998), and hence, the stride velocity is the product of stride length and stride frequency (Kamen, 2002). The centre-of-mass height (h_{cm}) for each sprinter is $0.57h_s$ for healthy men (Grimshaw et al., 2004; McGinnis, 2005) and the stride length is given by $1.35h_s$, where h_s is the standing height of the athlete (Hoffman, 1971; Rompottie, 1972).

3.2.2.3 Rate of change of anaerobic energy

Moreover, the rate of chemical energy conversion can also be expressed as the sum of the rate of energy produced from the aerobic and anaerobic metabolic pathways (Ward-Smith, 2000). By combining this sum with equation 3.1, therefore, the following formula as shown in equation 3.3 can be derived:

$$\frac{dCan}{dt} = \left(\frac{dH}{dt} + \frac{dW}{dt}\right) - \frac{dCae}{dt} \quad \text{… Equ. 3.3}$$

The rate of aerobic energy is subtracted from the sum of the rate of heat energy and mechanical work to determine the rate for the anaerobic energy. The component on the left hand side (Equ. 3.3) is the rate of change of anaerobic energy (C_{an}) and the components on the right hand side are the rate of change of heat energy (H), mechanical work (W) and aerobic energy (C_{ae}) respectively. The associated rate of change of aerobic energy is determined in accordance with theoretical equations (See Appendix B, section B.2) previously developed by Van Ingen Schenau (1991).

3.2.2.4 Modelling the rate of energy production for each anaerobic subsystem

The mathematical model that was used to represent the rate of production and decay of each anaerobic energy subsystem, was based on a type of Gamma distribution model since it is a flexible distribution to model biochemical processes that are hypothetically to be exponentially distributed, and a good fit for the sum of independent random variables (Hogg and Craig, 1978; Wlodarczyka and Kierdassuk, 2006). The Gamma mathematical model is expressed and characterised with respect to different parameters in terms of a shape (α) parameter, and a scale (β) parameter which is also known as the rate parameter (Equ. 3.4). For this model, the shape α was taken as 2 in accordance with previous works of Hogg and Craig (1978) so that a first-order in time t (Equ. 3.5) was obtained to represent the characteristics of the three anaerobic subsystem power distribution curves and hence, this makes computations faster (Gu et al., 1996).

The gamma distribution *G* (Equ. 3.4) comprises of the gamma function which is denoted by $\Gamma(\alpha)$ and this mathematical notation is the factorial of $(\alpha - 1)$, and α is an integer number greater or equal to 1. The variables β, α, and t represent the scale, shape and time respectively, and the variable e represents the exponential value.

$$G(t;\alpha,\beta) = \frac{\beta^{\alpha}}{\Gamma(\alpha)}\ t^{\alpha-1}e^{-\beta t} \quad \text{… Equ. 3.4}$$

$$G(t;\beta) = \beta^2 \cdot t \cdot e^{-\beta t}\ , \quad \text{… Equ. 3.5}$$

The initial estimates for the scale parameters β_1, β_2 and β_3 for ATP endogenous, Phosphocreatine (PCr) utilization and oxygen-independent glycolysis were determined by finding the time constants corresponding to the respective maximum of the mean anaerobic power distribution curve for all the sprinters. These rate parameters served as initial estimates or inputs to run the computational program (See Appendix B, sections B.3 and B.4).

By using equation 3.5, the rate of change of the anaerobic metabolism was expressed as the sum of multiple gamma distributions to represent the three anaerobic subsystem powers, and this was mathematically represented in equations 3.6, 3.7 and 3.8. The symbol P_n represents the instantaneous powers for each anaerobic subsystem measured in *watts per kilogram*. The nonlinear parameters (β_n) are the rate parameters of the respective anaerobic subsystem, and they are initially determined by taking the inverse of the time constant (τ_n) for each anaerobic subsystem. The subscript n is an integer number ranging from 1 to 3 and it represents the three anaerobic subsystems. In equation 3.7, the variable P_1 denotes the rate of energy released from endogenous ATP, the variable P_2 is the rate of energy released from Phosphocreatine (PCr) utilisation, and the variable P_3 represents the rate of energy released from the oxygen-independent glycolysis anaerobic subsystem (See Appendix B, sections B.3 and B.4).

$$\frac{dCan}{dt} = {\beta_1}^2 \cdot t \cdot e^{-\beta_1 t} + {\beta_2}^2 \cdot t \cdot e^{-\beta_2 t} + {\beta_3}^2 \cdot t \cdot e^{-\beta_3 t} \quad \text{… Equ. 3.6}$$

$$\frac{dCan}{dt} = \mathrm{P}_1(t) + \mathrm{P}_2(t) + \mathrm{P}_3(t) \quad \text{… Equ. 3.7}$$

$$\frac{dCan}{dt} = \sum_{n=1}^{3} \mathrm{P}_n(t) \quad \text{… Equ. 3.8}$$

3.2.3 Determination of the initial estimates of the nonlinear parameters

The initial estimates of the nonlinear parameters were determined by taking the maxima (3 maxima) from the curve obtained by calculating the mean of the anaerobic powers (*i.e.* rate of change of anaerobic energy) for all athletes over each 10-m interval, and it is illustrated in Figure 3.1. It is important to find these first estimates to minimise computational time, and prevent divergence from solutions (Boutayeb and Darouach, 1995; Chen and Fassois, 1992). The computational program, as shown and summarized in flowchart diagram (See Appendix B, section B.3), was run repeatedly until convergence is reached or until the error (ε) which is the difference between the computed anaerobic power, and the total of the anaerobic subsystem powers at each distance interval for each athlete was minimal. At first, the 10-m split times, the total anaerobic power and the estimated values of the nonlinear parameters (β) were initial inputs to the computational program to find an estimate of the individual subsystem anaerobic powers. The norm function (norm) was used to find the residual error so that the amplification errors were kept minimum (Kariya and Kurata, 2004; Wolberg, 2005). In addition, the pseudo-inverse function (pinv) was used, in this case, especially for a non-square matrix (6 variables representing the anaerobic subsystem powers and the rate parameters x 10 equations representing the 10 split times) and this function works well when the number of equations are greater than the number of variables (Campbell and Meyer, 1991; Zheng and Bapat, 2004). In this particular case, the initial estimates of the time constants obtained from Figure 3.1, were determined as τ_1= 1.1 s; τ_2= 3.9 s; τ_3= 7.9 s, and are used subsequently to estimate the rate parameters β_1, β_2 and β_3 by taking the inverse of the respective time constants. In Figure 3.1, the variables $\mathrm{m1}$, $\mathrm{m2}$ and $\mathrm{m3}$ represent the three consecutive maxima of the anaerobic power curve.

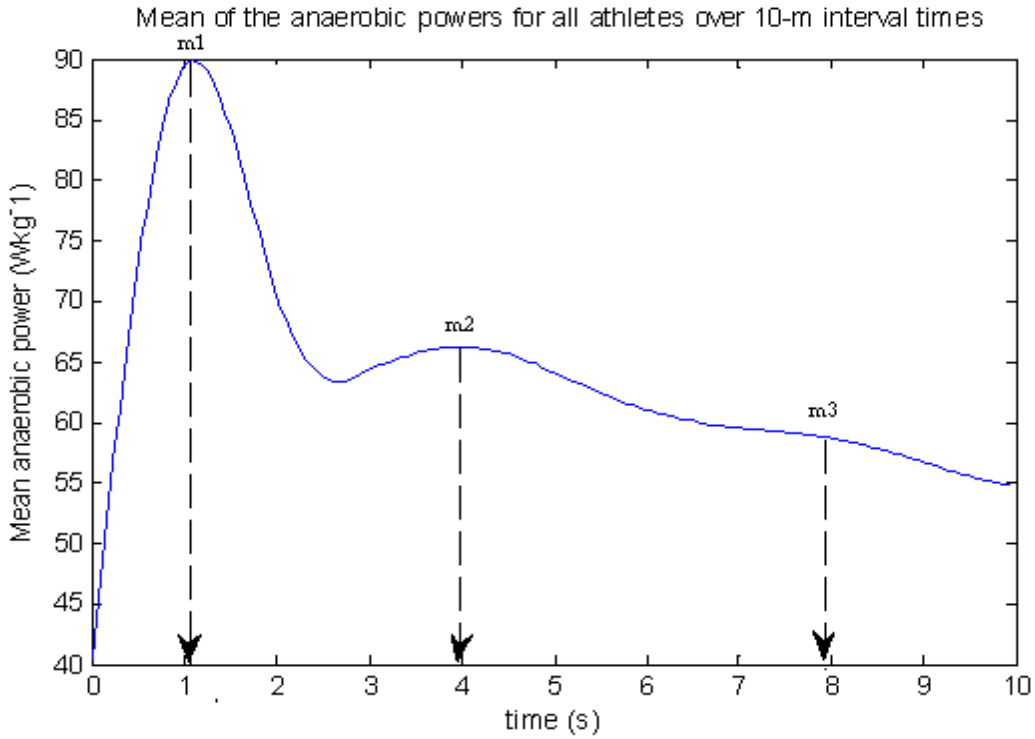

Figure 3.1: Initial estimates of the nonlinear variables

3.2.4 Validation of model

The validation of the mathematical modelling was assessed in respect to the root mean square error (RMSE), in determining the total anaerobic powers derived for each athlete. The percentage root mean square error was calculated to find the error between the exact calculated total anaerobic power at each discrete time from the sum of the simulated individual anaerobic subsystem powers at these discrete times. The calculated total anaerobic power was the difference between the total aerobic power and the power lost due to mechanical work and heat. Figure 3.2 summarises the energy processes involved to mathematically model and analyse the chemical energy produced from the anaerobic energy system pathway. The chemical energy produced from both the anaerobic and aerobic metabolisms was converted into heat energy and mechanical energy.

Consequently, from this relationship, the energy from the anaerobic process can be determined, and then subsequently compared to the sum of the energy produced from the three corresponding anaerobic subsystems (Laurent and Locatelli, 2002; Ward-Smith and Radford, 2000).

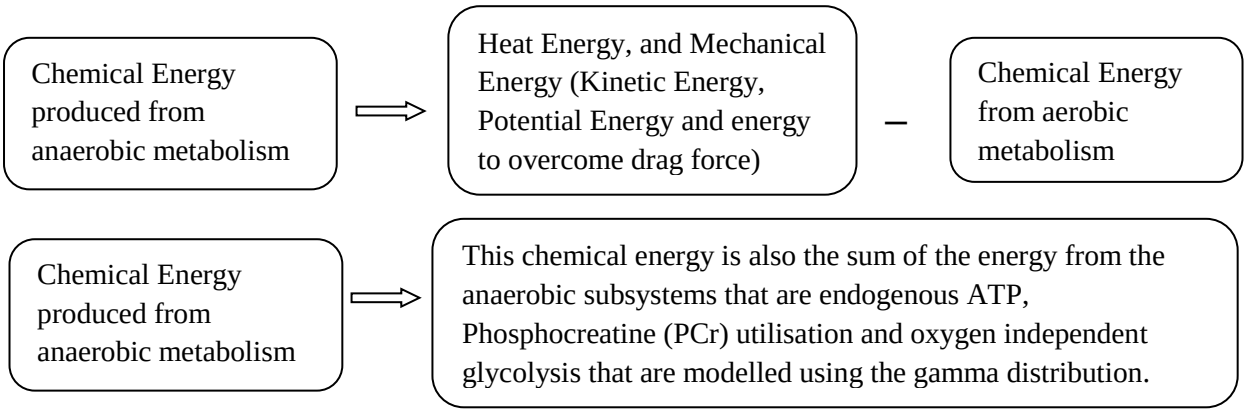

Figure 3.2: The flowchart diagrams summarise the mathematical model in simulating the various anaerobic energy subsystems (Laurent and Locatelli, 2002; Ward-Smith and Radford, 2000).

3.3 Results

The velocity-time graph (Fig. 3.3) of the elite sprinters showed clearly the increase in speed from 0 ms^{-1} to a maximum speed where, during this period, the acceleration was maximal as shown by the steep slope of the velocity-time curve during the first 2 seconds. Subsequently, around 5 to 8 seconds, the sprinters started to decelerate slowly which continued in the same trend till the completion of this sprinting race.

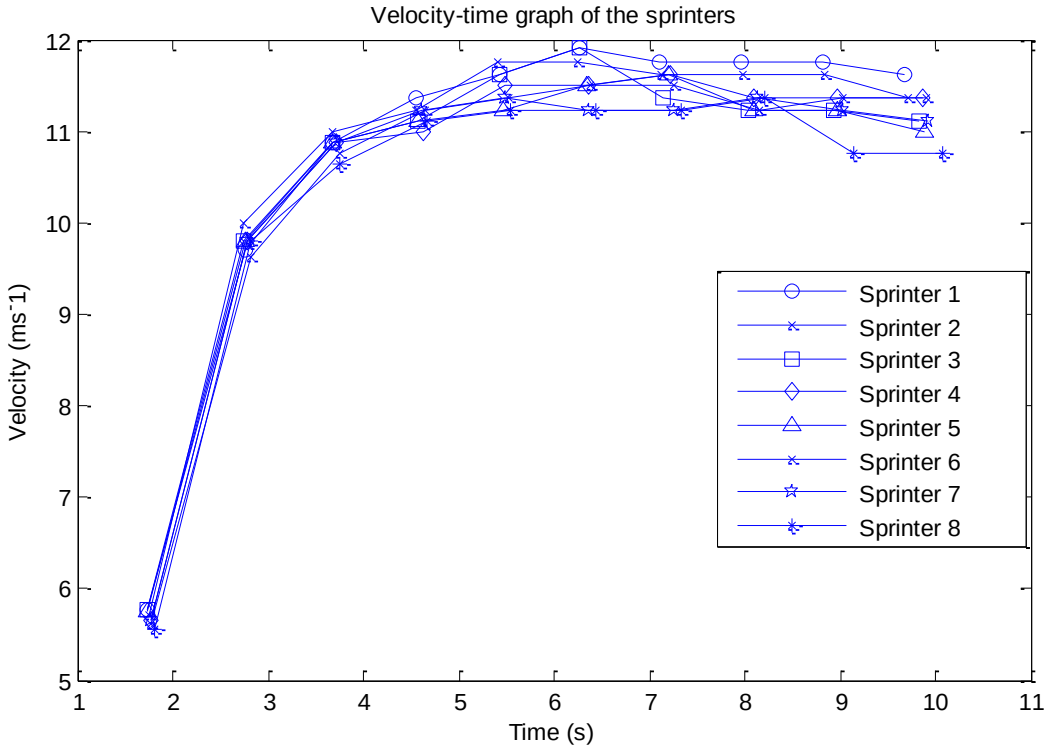

Figure 3.3: Velocity of all the 100m-dash elite sprinters (n = 8)

3.3.1 Anaerobic and aerobic power contributions

The total power, anaerobic power and aerobic power per unit body mass for all sprinters were determined (See Figure 3.4). Respective measured reaction time for each sprinter was excluded from the respective finishing time since during this brief period of about 0.141 (±0.01)s, the sprinters were still at rest, and hence equations 3.1 and 3.3 do not apply as the rate of change of heat energy and mechanical energy were assumed to be zero at time t = 0. It was found that the anaerobic power contributed to approximately 95% of the total power for this 100-m sprint.

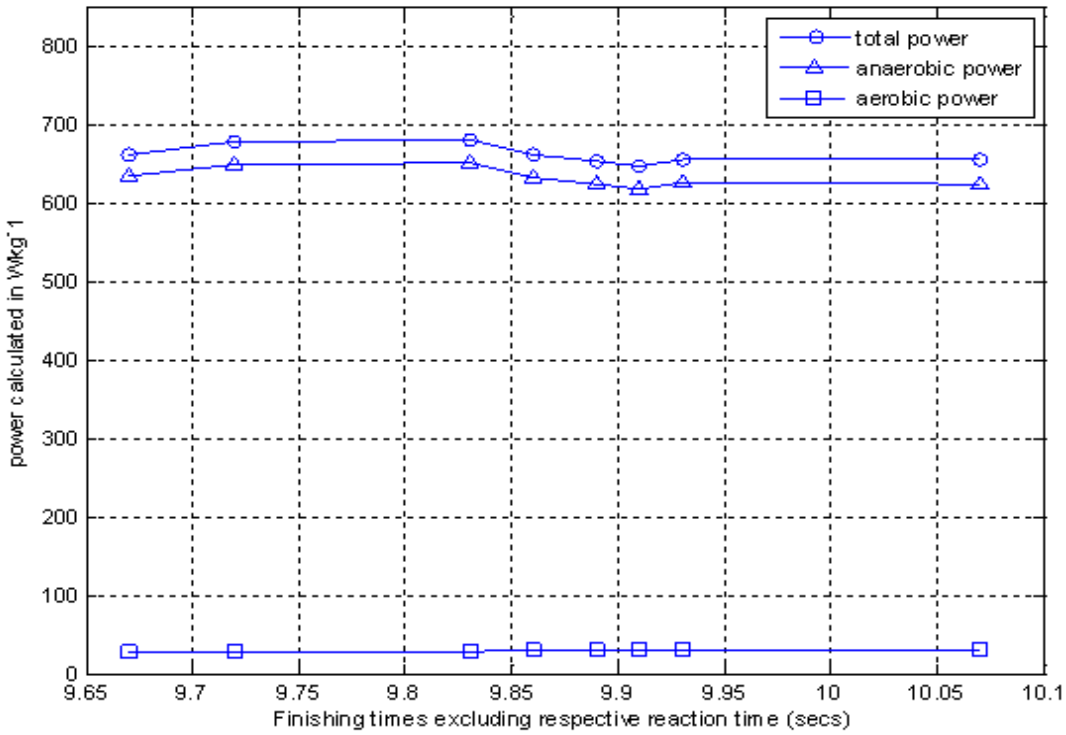

Figure 3.4: Total power, anaerobic power and aerobic power for all sprinters (n=8) are plotted vs. finishing times excluding measured reaction times.

3.3.2 Anaerobic subsystems (ATP endogenous, PCr utilisation and oxygen independent glycolysis)

Figure 3.5 shows the normalised maximum rate of energy production for each subsystem for the anaerobic metabolism for a particular athlete to illustrate the difference among the anaerobic subsystems. The time T1 (Figure 3.5) represents the time when the ATP endogenous curve intersects the oxygen independent glycolysis energy curve measured as 2.71 s, and T2 represents the time when there is intersection between the phosphocreatine (PCr) utilisation and oxygen-independent glycolysis energy curves measured as 5.17 s. Furthermore, the mean and standard deviation of the power variables (watts per kilogram) P_1, P_2 and P_3 were 6.6±1.78 Wkg^{-1}, 40.5±2.97 Wkg^{-1} and 9.98±1.04 Wkg^{-1} respectively and the nonlinear parameters (β_1, β_2, β_3) representing the rate parameters of the

anaerobic subsystems were 0.94±0.05 s^{-1}, 0.31±0.015 $s^{-1}$and 0.11±0.004 s^{-1} respectively. As shown in Figure 3.5, the endogenous ATP concentrations decreased rapidly at the start of the race and contributed to most energy during the first 2 to 3 seconds of this 100-m sprint race. Then, Phosphocreatine (PCr) utilisation process buffered the drop in ATP for another 5 to 8 seconds during which the PCr utilisation curve reached its maximum much before the oxygen independent glycolysis energy-curve reached its maximum at about 9.1 seconds.

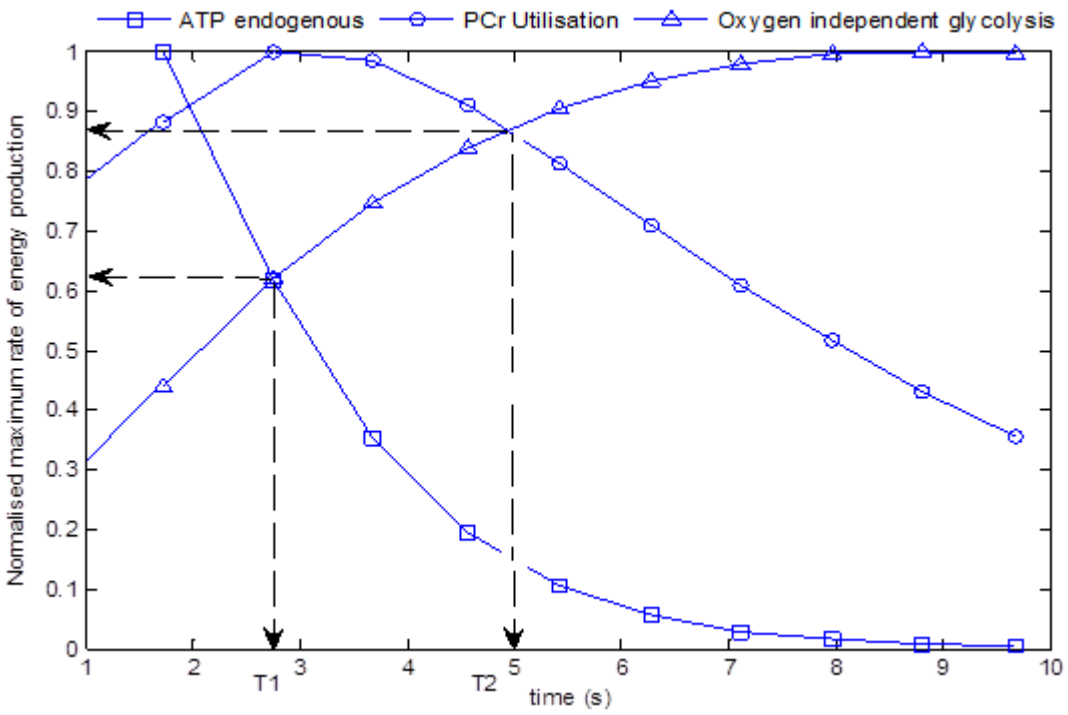

Figure 3.5: Normalised maximum rate of energy production of first rank sprinter (Maurice Greene) for each subsystem of the anaerobic metabolism vs. time excluding reaction times. The arrows represent the x and y coordinates of the points of intersection of the anaerobic subsystem curves.

By extrapolating the mathematical results, the effect of increasing the percentage of energy released from the PCr utilisation anaerobic subsystem was investigated using the computed anaerobic subsystem powers and the rate parameters for the first rank sprinter (See Appendix B, Table B.1). The mathematical model predicted

that if the percentage of energy released from the PCr Utilisation was increased to 110%, the finishing time of the first rank sprinter would have been 9.27 s, and if the percentage of energy contribution from this particular anaerobic subsystem was increased further to 120%, the finishing time would have been 8.88 s and these results are shown in Figure 3.6.

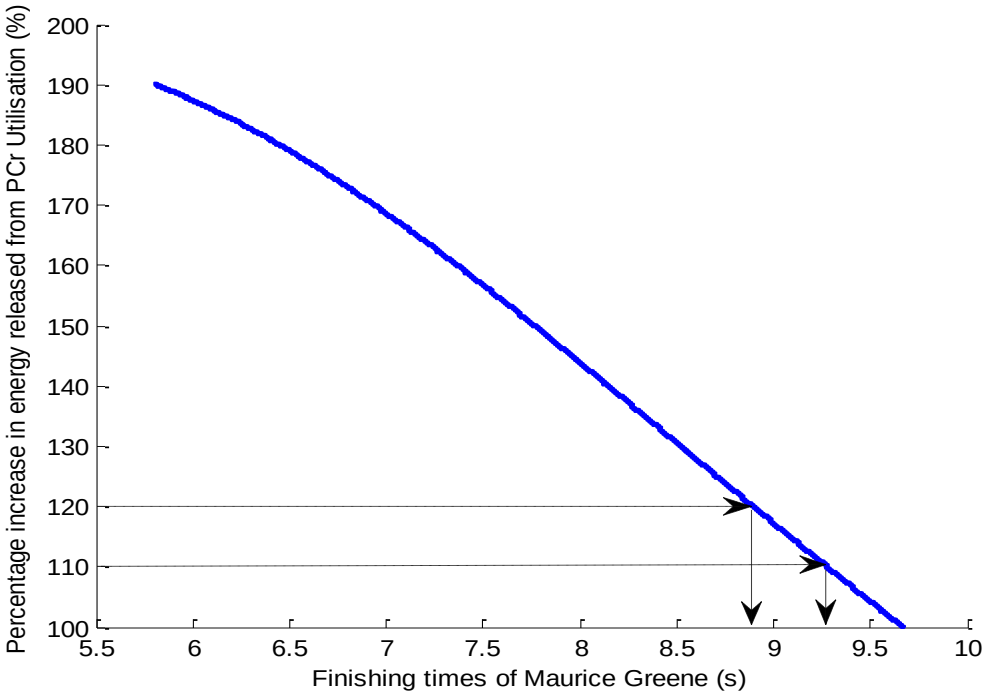

Figure 3.6 The effect of increasing the percentage of energy released from the PCr utilisation anaerobic subsystem for the first rank sprinter. The arrows represent the effect of increasing the percentage of energy produced from the PCr anaerobic subsystem and the expected finishing times for Maurice Greene.

3.3.3 Root mean square error (RMSE) of mathematical model

The percentage root mean square error was calculated to determine the error between the exact calculated total anaerobic power at each discrete time from the sum of the simulated individual anaerobic subsystem powers at each discrete time (Figure 3.7). This total anaerobic power is the difference between the total aerobic power and power lost due to mechanical work and heat. The minimum percentage

root mean square error was 0.0022 and the maximum percentage root mean square error was 0.018. The variability of the percentage errors were caused by the distinct kinematics as well as the distinct weights, heights and reaction times of the elite sprinters in finding convergent solutions to the variables.

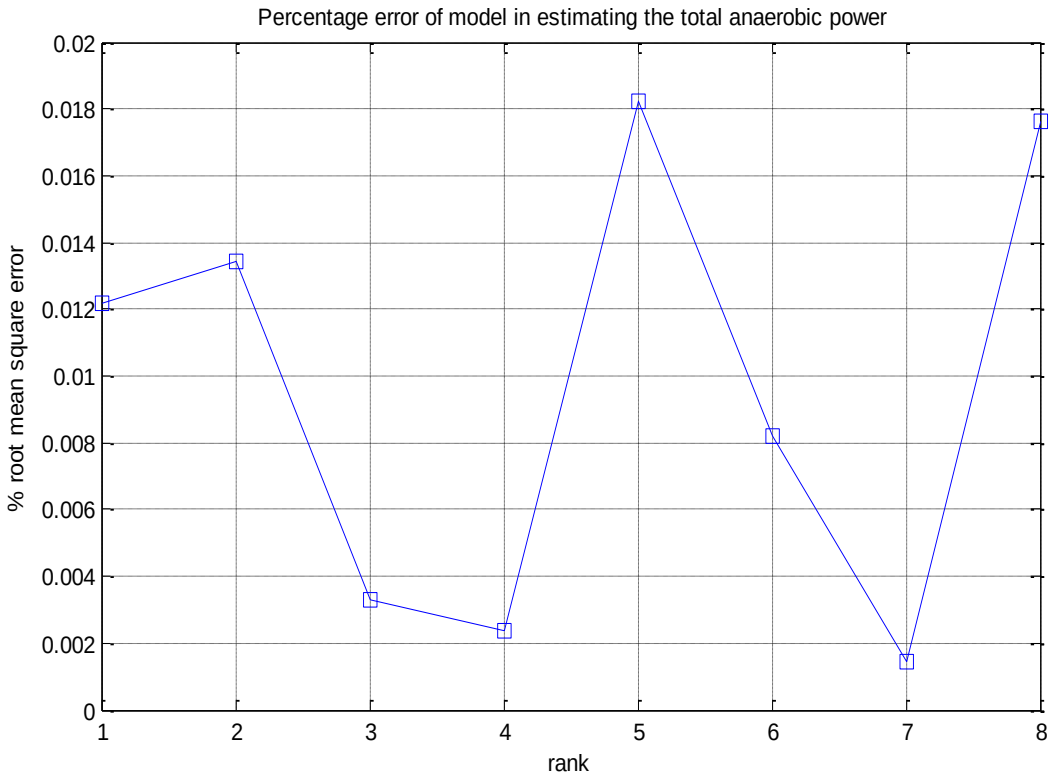

Figure 3.7: Percentage root mean square error (RMSE) in estimating the total anaerobic power for all athletes ($n = 8$). The average value of RMSE was 0.009W.

3.4 Discussion

3.4.1 Model validation

The average value of the root mean square error (RMSE) for this mathematical model in determining the total anaerobic powers for all athletes was 0.009W which indicates a good model (Ward-Smith and Radford, 2000; Wargon et al., 2009) in determining the total anaerobic power for this data, under these physical and environmental conditions. Furthermore, muscle biopsy studies by Gaitanos et al. (1993) and Bogdanis (1996) found that, in maximal 6 seconds and 10 seconds

cycling sprints, that power output was supported by energy derived mainly from PCr degradation (the concentration of which decreased by 57%) and there was also a causal relationship between the percentage of PCr and speed which affected sprint performance.

3.4.2 Aerobic and anaerobic metabolisms

It was observed that the percentage of chemical energy derived from the anaerobic process was 95%, compared to literature where they found mathematically that 92% of chemical energy during the 100m sprint running was produced from anaerobic sources (Peronnet and Thibault, 1989; Ward-Smith, 1985). Therefore, the calculated percentage of energy production from anaerobic process as compared to literature also suggests that mathematical modelling may be a reliable tool in assessing the anaerobic and aerobic energy system pathways. The difference in percentage of the energy derived from the anaerobic process between literature and the mathematical model may be related to decreasing finishing times of the 100-m sprint running over the last decades (IAAF, 2008). Nevertheless, the values obtained by mathematical modelling and previous related studies (Peronnet and Thibault, 1989; Laurent and Locatelli, 2002) indicate that it is possible to model physiological systems accurately by mathematical models.

3.4.3 PCr utilisation anaerobic subsystem

The speed of all the athletes started to decrease at around 5 to 7 seconds through the sprint race as well as in previous studies (Hirvonen et al., 1987), and it is shown (Figure 3.3) that this decrease in speed coincided with the highest rate of decay of the Phosphocreatine (PCr) utilisation energy curve (Figure 3.5). The energy contribution from the PCr system was found to be 12.8% higher than the

energy contribution derived from oxygen independent glycolysis over the total sprint duration. Bogdanis et al. (1996), who had examined the contribution of phosphocreatine (PCr) during repeated bouts of cycle ergometer sprints (10 to 30-s), established that there was a high correlation (r) between the percentage of PCr and the percentage of restoration of mean power output (MPO) as well as speed during the initial 10 seconds of the sprints ($r = +0.84$ and $r = +0.91$). Couple with that, Bogdanis et al. (1996), did not find any correlation between power output recovery and concentration of any other metabolites (lactate, hydrogen and dihydrogen phosphate ions). Furthermore, there was no observed correlation between the percentage of PCr and MPO during the last 20-s of the sprint (Bogdanis et al., 1996). In addition to Bogdanis' observations, an independent study by Hirvonen et al. (1987) demonstrated, in a series of maximal cycling sprints (40-100 m), that skeletal PCr stores were severely depleted after 5 to 7-s. Interestingly, it was found that elite sprinters used more of their available PCr stores over the first 5 to 7-s than sprinters of slightly less ability. Furthermore, the rate constant for the PCr anaerobic metabolic energy process ($0.31s^{-1}$) as determined in this mathematical modelling was found to be greater than that of the oxygen-independent glycolysis metabolic process ($0.11s^{-1}$). This observed metabolic behaviour can be explained because it takes more time for the ATP to be produced from the oxygen independent glycolysis process than that from the PCr utilisation energy process (Baechle and Earle, 2000; Wilmore and Costill, 2005).

3.5 Summary

In this study, the energy produced from the anaerobic and aerobic capacity was investigated using nonlinear mathematical modelling. It was found that anaerobic energy contributes to 95% of the energy needed to complete the 100m sprint. In addition, investigation on the anaerobic subsystems showed that the phosphocreatine utilisation energy was found to be 12.8% higher than the energy contribution from oxygen independent glycolysis for the total duration of the sprint. Furthermore, it was found that, for any particular anaerobic subsystem, that the rate parameters for all the elite sprinters were similar and that both the rate parameter and the maximum power achieved, for any particular anaerobic subsystem, affected sprint performance. It was shown in this study through mathematical modelling together with dietary-based as well as muscle biopsy studies (Baechle and Earle, 2000; Bogdanis et al, 1996; Wilmore and Costill, 2005) that this phosphocreatine utilisation anaerobic subsystem affects the performance of athletes during high-intensity short duration exercise. The next experimental case study, chapter four, investigated the energy produced from the anaerobic and aerobic systems for an endurance physical activity.

CHAPTER FOUR
Study Two

Optimal management of resources during various pacing trials for a 20-km cycling time trial

According to the literature review of the physiological models of exercise fatigue, the type of physical activity (Weir et al., 2006), the failure of the energy metabolic pathways (Noakes, 2000; Shulman and Rothman, 2001), the conservation of energy via the energy systems (Shulman and Rothman, 2001), the increase in blood lactate concentration (Brooks et al., 2005; Hill et al., 1923; Hill, 1924) may cause power output reduction during endurance exercise, the increase in perception of effort during endurance exercise (Behm and St-Pierre, 1997; Sandow, 1952) altogether affect sports performance. Therefore, this experimental case study was conducted in the hope to validate these physiological control model hypotheses by, firstly, analyzing the effect of different pacing on the performance of an endurance time trial exercise by modelling and analysing the energy production from the energy systems; And secondly by analyzing the homeostatic disturbance that these different pacing might cause to the human organism and finding any relationship between blood lactate concentration and the rating of perceived exertion (RPE).

4.1 Introduction

In the first study, the amount of energy derived from the energy systems in maximal exercise of short duration were investigated, and in this second experimental case study, the effect of pacing on the energy expenditure from the

aerobic and anaerobic systems was investigated for a 20-km cycling time trial exercise. Furthermore, this second study endeavoured to find the degree of homeostatic disturbance that different pacing time trials (self pace, even pace and variable pace) might cause on the human organism together with any association between RPE and blood lactate concentration.

In athletic competition, pacing is important so that the available metabolic resources are utilised effectively to finish a physical activity in the minimum possible time, and to maintain enough metabolic resources to complete that task successfully (Ulmer, 1996). One way to analyse pacing is through time trial exercise and these can be assessed in terms of energy efficiency. Few studies have investigated the energy production from anaerobic and aerobic metabolisms during prolonged exercise bouts (Hettinga et al., 2006; Hettinga et al., 2007). In this study, therefore, the energy produced from aerobic and anaerobic metabolic processes are investigated for various types of pacing, and assessed with respect to work rate and energy expenditure for a 20-km cycling time trial.

4.2 Method

4.2.1 Participants

Ten healthy and well-trained male cyclists participated in this study. The mean (± standard deviation) height, body mass index (BMI), the measured maximum oxygen uptake of the cyclists ($\dot{V}O_{2\max}$) and their known associated work capacity were 1.77 (±0.06) m, 24.2 (±1.8) kg·m^{-2}, 4.89 (±0.32) L·min^{-1}, 353 (±30) W respectively. The age of the participants ranged from 25.5 to 40.1 years.

4.2.2 Study protocol

This research study was ethically approved by the School of Life Sciences Ethics Committee, University of Northumbria at Newcastle. The healthy and well-trained participants were required to complete a 20-km cycling exercise bout in the minimum possible time employing different pacing trials. These were: self pace (cycle as hard as they felt they could at any moment in time), even pace using the mean output from their self pace cycling time trial, and a variable pace based on 70% and 140% of the subject's respective self pace average power output (de Koning et al., 2011; Palmer et al., 1999). The participants completed these three different pacing time trials on separate occasions, in the physiology lab of the School of Life Sciences in Northumbria University, with at least one week rest in-between the trials for them recovery purposes and prevent a training effect (Flynn et al., 1994).

4.2.3 Data collection

Physiological data including heart rate (BPM) were recorded using a data acquisition system (Powerlab, ADI Instruments, Australia), and volume of oxygen consumption ($\dot{V}O_2$/L·min^{-1}) was measured using an online gas analyser (Cortex Metalyser, Cortex Biophysik, Germany). Power outputs were recorded at a frequency rate of 11 Hz using Velotron 3D software which was interfaced with the Cycle Ergometer (VelotronPRO, RacerMate Inc., USA) that was used for all cycling time trials. The rating of Perceived Exertion (RPE) was used as a subjective measure for the sensation of fatigue the cyclists felt during the 20-km cycling time trial, and these RPE scores were obtained at every 2-km interval while blood samples were collected for every 4 km interval to determine blood lactate concentration (mmolL^{-1}). The data were collected by the research team of School

of Life Sciences, and these data were then analysed using Matlab software platform version R2008a. SPSS v17 software was used for subsequent statistical analysis. All the collected and computed data were tested for parametricity using Kolmogorov-Smirnov (K-S) test to find out whether they follow a normal distribution so as to ensure the appropriate statistical tests were identified for comparison purposes (Fasano and Franceschini, 1987; Lopes et al., 2007).

4.2.4 Data analysis

In order to analyse the energy expenditure for the different pacing trials, the metabolic aerobic power (P_{met}) was determined from the measured volume of oxygen consumption ($\dot{V}O_2$), and the respiratory exchange ratio (RER) which is the ratio of the amount of carbon dioxide produced to the amount of oxygen consumed by the cyclist. The metabolic aerobic power (Hettinga et al., 2006) was determined based on the volume of oxygen consumption and RER (Equ. 4.1). Then, the Gross Mechanical Efficiency (GME) was calculated from the ratio of the power output (P_{tot}) to metabolic aerobic power (P_{met}); and the anaerobic mechanical power (P_{an}) during the time-trial was computed as the difference between the power output and the aerobic mechanical power P_{ae} as shown in Equ. 4.2.

$$P_{met}\ (W) = \dot{V}O_2\ (L \cdot min^{-1}) \times \{(4940 \cdot RER + 16040)/60\} \quad \ldots \text{Equ. 4.1}$$

$$P_{an} = P_{tot} - P_{ae} \quad \ldots \text{Equ. 4.2}$$

In this analysis, it was assumed that respiratory exchange ratios (RER) in excess of 1.00 happened due to the buffering of lactate by bicarbonate (Hettinga et al., 2006; Hettinga et al., 2007). The lactate is the body's buffering agent that neutralizes the acid that accumulates in the working muscles (Brooks, 2001; Robergs et al., 2004).

Moreover, the Hazard Score index (de Koning et al., 2011), which is the product of the momentary rating of perceived exertion (RPE) and the fraction of the remaining distance, has the ability to depict the likelihood that the cyclists would change their power outputs or their velocities during the cycling time-trial. This hazard score would also show indirectly how the power outputs of the cyclists were regulated. The calculated anaerobic and aerobic powers for the various pacing trials were then tested for parametricity using Kolmogorov-Smirnov (K-S) test, and then One Way Analysis of Variance (ANOVA) with repeated measures was used to compare the mean data between the time trials (Field, 2009). When significant F ratios were found ($p < 0.05$) in the statistical analysis, the means of the tested variables were subsequently compared using a Tukey's post-hoc test. When testing for statistical significance, the p-value represents the probability of reaching the test statistic value (*e.g.* the mean of a population sample) as extreme as the observation value (Berger and Casella, 2001). The F-ratio (See Appendix C, section C.1) is a test statistic in finding whether the difference between two or more independent variables is statistically significant or stable by computing the ratio of the variance between groups and the variance within groups (Lomax, 2007; Sawilowsky, 2002). The product moment correlation coefficient (r) was used to analyse relationship (if any) between the variables (See Appendix C, section C.2 for its mathematical formula).

4.3 Result

4.3.1 Total work done by all the cyclists for all the pacing time-trials

The mean (± standard deviation) of total work done by all the cyclists for self pace, even pace and variable pace were 5.14 (±0.01) MJ, 5.15 (±0.01) MJ and 5.13 (±0.01) MJ respectively (See Table 4.1).

Table 4.1: A summary of the total work (W_T), work through anaerobic capacity (W_{Anae}) and work through aerobic capacity (W_{Ae}) are displayed for each pacing trial. There was no significant difference between W_T ($p > 0.05$). However, there were significant differences among anaerobic capacities, and aerobic capacities for all pacing trials ($p < 0.01$). Each row of the table (from the 3rd row onwards) corresponds to the work done by a cyclist in increasing order of time performance except for the last row (bold) which represents the **average** of these variables.

Self Pace (x 10^5 / J)			**Even Pace (x 10^5 / J)**			**Variable Pace (x 10^5 / J)**		
W_T	W_{Anae}	W_{Ae}	W_T	W_{Anae}	W_{Ae}	W_T	W_{Anae}	W_{Ae}
5.5694	0.9786	4.5909	5.5590	0.6373	4.9217	5.5689	2.0467	3.5222
5.5684	0.9994	4.5690	5.5680	0.8807	4.6873	5.5714	2.1433	3.4281
5.5657	1.5923	3.9733	5.5710	0.5777	4.9933	5.5366	2.0519	3.4847
5.3173	0.8023	4.5150	5.3233	0.5591	4.7642	5.2821	1.9936	3.2885
5.1568	1.0666	4.0901	5.1490	0.5264	4.6225	5.0718	1.9452	3.1266
5.1085	0.7840	4.3245	5.1052	0.6652	4.4400	5.1047	1.8907	3.2139
5.0100	1.2439	3.7661	5.0069	0.8631	4.1437	5.0038	2.0832	2.9206
5.0132	1.1701	3.8431	5.0145	0.7242	4.2903	5.0089	2.0278	2.9810
4.7312	1.0714	3.6598	4.7332	0.7877	3.9455	4.7346	2.0332	2.7013
4.3988	0.8725	3.5262	4.4339	0.6643	3.7696	4.3699	1.7514	2.6184
5.14393	**1.05811**	**4.0858**	**5.1464**	**0.68857**	**4.4578**	**5.1252**	**1.9967**	**3.1285**

As expected, these results confirmed that there was no significant difference ($p > 0.05$) in the total work done by all the cyclists for each of the three different pacing trials. Furthermore, there was no significant difference ($p > 0.05$) in the total power which is the sum of the aerobic and anaerobic powers for each athlete for each pacing trial. Furthermore, the total energy used by each cyclist for each pacing trial was practically constant despite the large variation in the amount of energy produced via the anaerobic energy system and the aerobic energy system for each pacing trial.

4.3.2 Anaerobic power, aerobic power and total power

Figure 4.1 shows the total power output together with the corresponding computed aerobic powers and anaerobic powers for the first rank cyclist for the self pace trial depicting the exponential growth and decay respectively of the energy produced from each energy pathway. Figure 4.2 shows the mean total power of all the cyclists for 5-km cycling interval. The average even pace power was about 265 W which was constant throughout the time trial while both self pace power and variable pace power decreased between 5 km to 15 km followed by endspurts (increase in power outputs) between 15 km to 20 km of the cycling time trial. Moreover, the mean gross mechanical efficiencies for self pace, even pace and variable pace were 17.9±1%, 18.1±0.9% and 18.3±1.1% respectively. In addition, it was found that there was no significant difference ($p > 0.05$) in the gross mechanical efficiency between the different pacing time-trials.

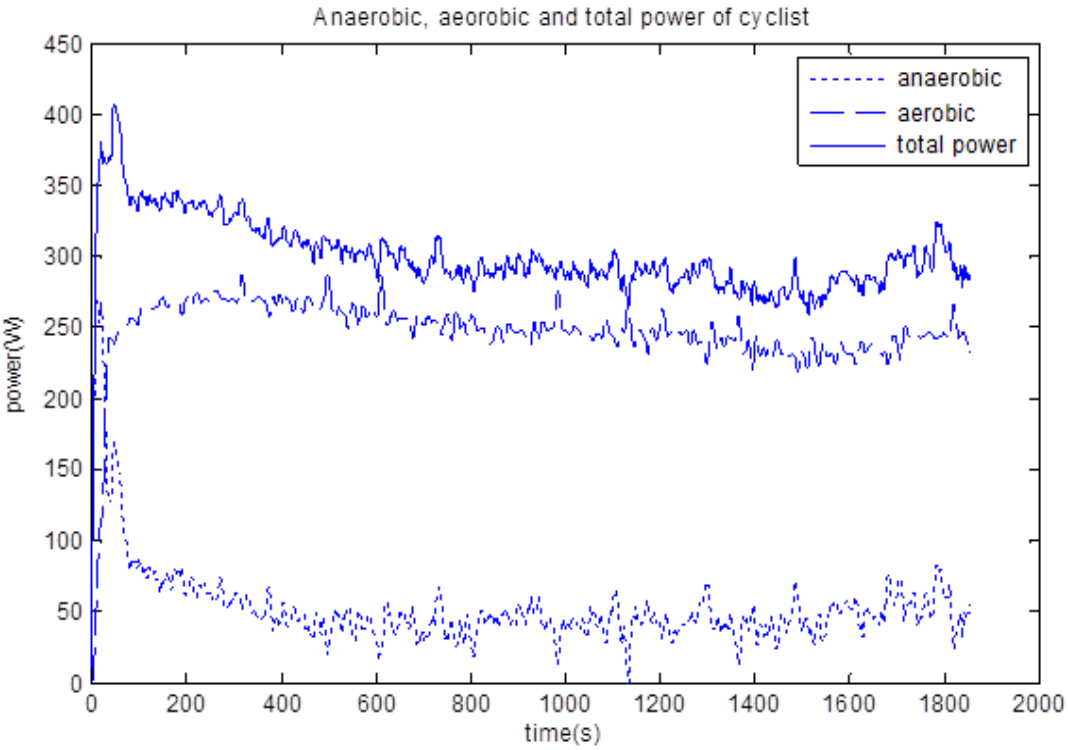

Figure 4.1: Anaerobic power, aerobic power and total power (W) distribution vs. time (s) for a particular cyclist (ranked first) in terms of performance time for a 20-km cycling self pace time-trial.

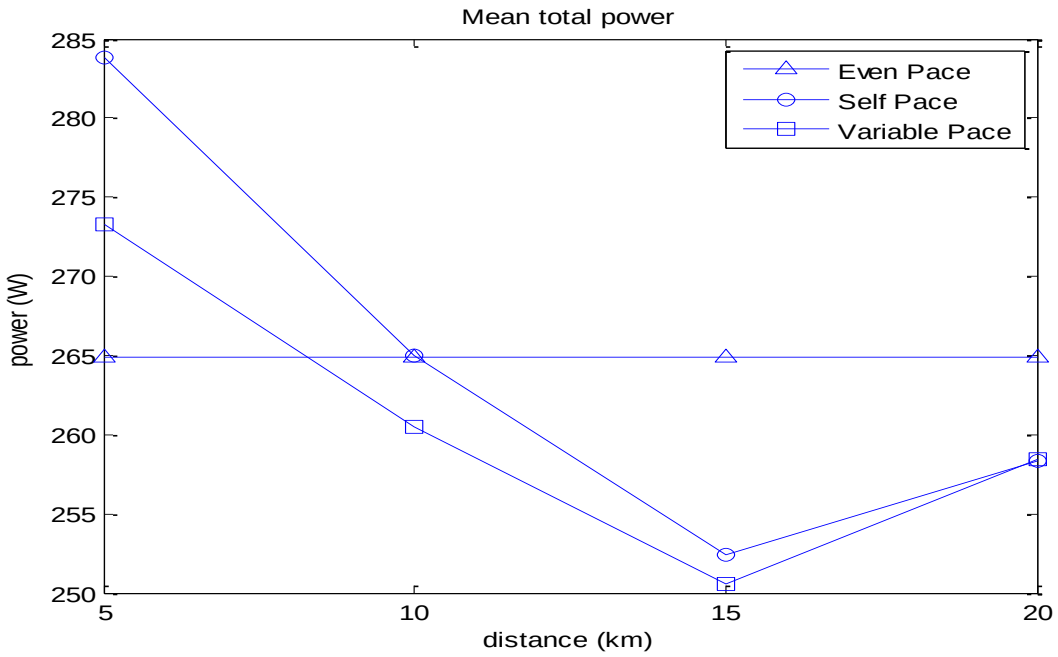

Figure 4.2: The average total power (W) for all the cyclists for 5-km interval for even pace, self pace and variable pace trials.

4.3.3 Mean aerobic and anaerobic powers at 5-km cycling interval

The aerobic and anaerobic powers for all cyclists were determined for each 5-km interval, and for each type of pacing (Figures 4.3, 4.4). The data in Figure 4.3, showed that for self pace trial, the mean aerobic power increased to a peak until the mid-point of the time trial (*i.e.* 10-km), then decreased slowly from 10 km towards the end of the cycling race. In even pace and variable pace time trials, however, the mean aerobic power for all athletes increased monotically to a "plateau". Interestingly, it was found that there was a significant difference ($p < 0.01$) between the estimated aerobic powers for all pacing trials. In Figure 4.3, the difference in the absolute values for each pacing aerobic power occurred because there was a large variability in the energy produced by the aerobic system from the ten cyclists.

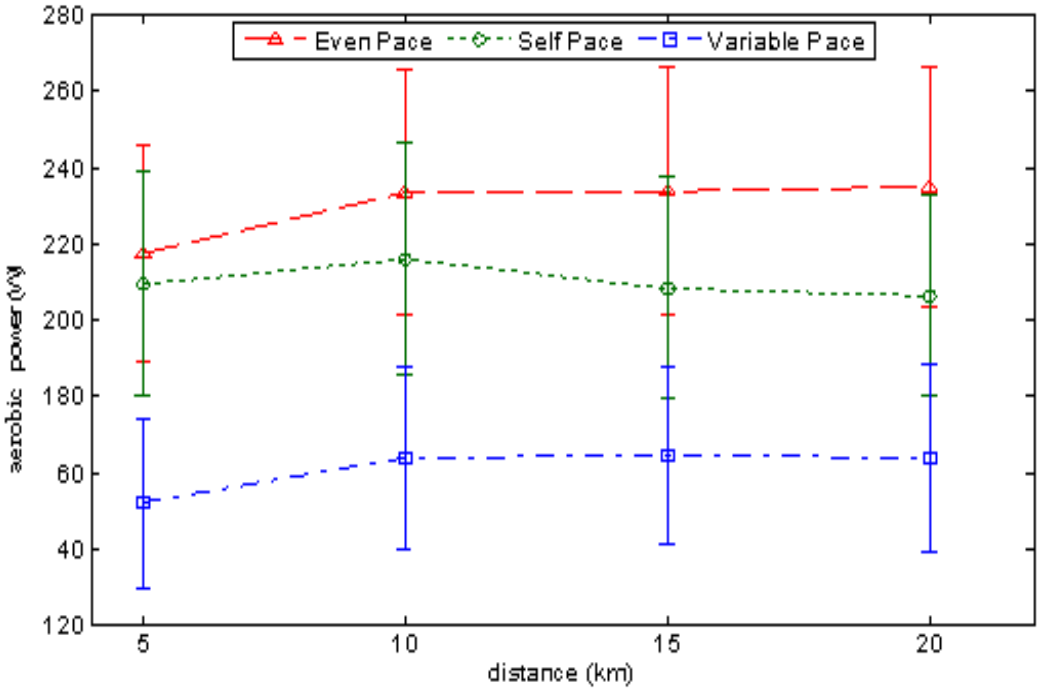

Figure 4.3: Mean aerobic power for the subjects ($n = 10$) for the 20-km cycling time trials for even pace, self pace and variable pace ($p < 0.01$).

Figure 4.4 describes the mean anaerobic power vs. distance (km) for the participants for the 20-km cycling time trial for even pace, self pace and variable pace at 5-km interval. It was found that there was also significant difference ($p < 0.01$) between the anaerobic powers for all pacing trials. Furthermore, the endspurts were clearly observed by the increase in the anaerobic powers between the 15-km to the 20-km of the cycling race for both variable pace and even pace trials. In Figure 4.4, the difference in the absolute values for each pacing anaerobic power occurred because there was a large variability in the energy produced by the anaerobic system from all the ten cyclists.

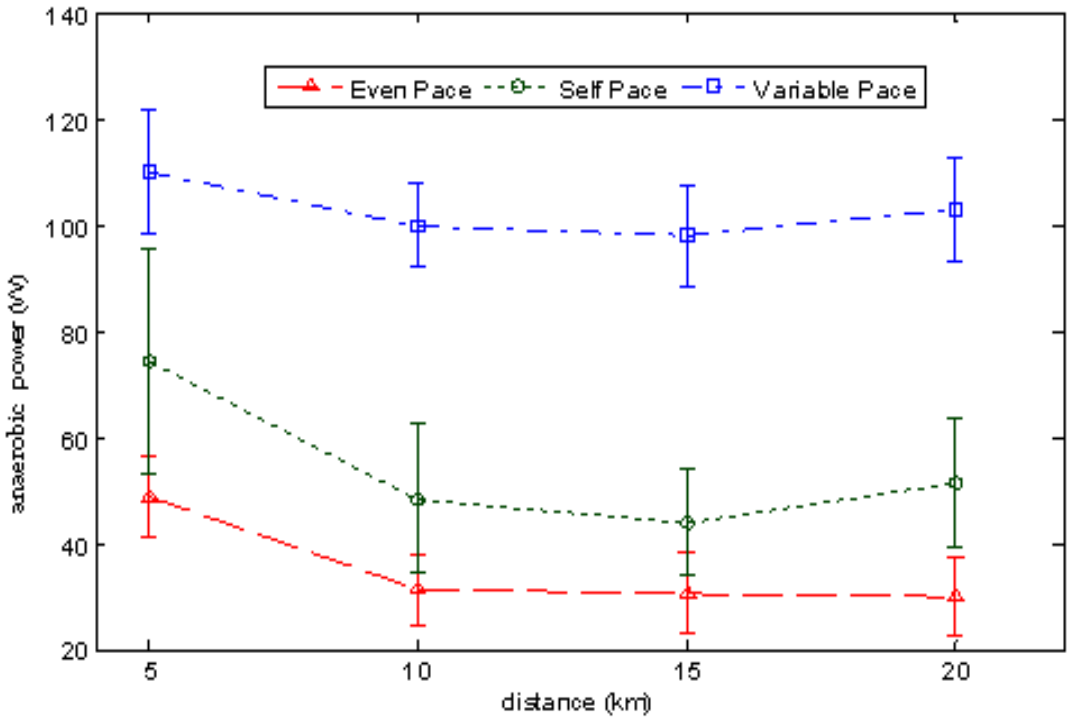

Figure 4.4: Mean anaerobic power of the cyclists ($n = 10$) for the 20-km cycling time trials for even pace, self pace and variable pace at 5-km interval ($p < 0.01$).

4.3.4 Hazard score index of the cyclists for various pacing time-trials

It was observed that the cyclists obtained the highest hazard score for the variable pace and least in self pace. Hence, variable pace caused greatest homeostatic disturbances in the physiological systems of the cyclists. In addition, all the hazard score graphs increased to a maximum and then decrease to zero resembling the arousal state inverted-U model or behaviour of the cyclists at a particular distance.

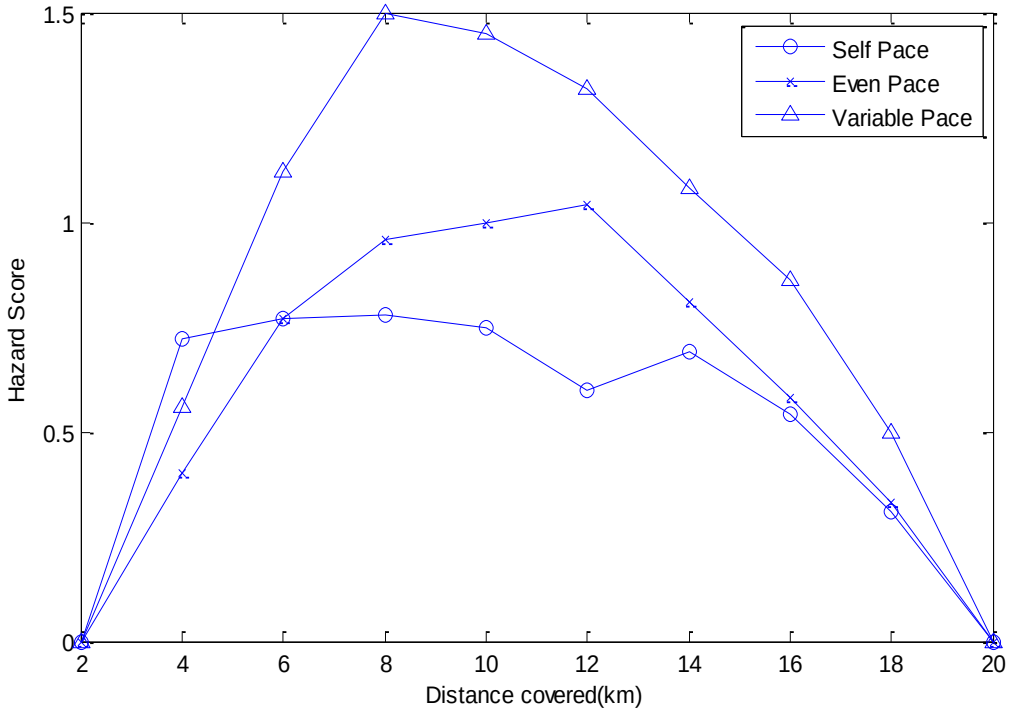

Figure 4.5: Hazard Score of the cyclists for each pacing time-trial vs. distance covered (Hazard score = momentary RPE x Percentage of remaining distance).

4.3.5 Rating of perceived exertion and blood lactate concentration

The mean ratings of perceived exertion (RPE) for self pace, even pace and variable pace for that time trial endurance exercise were 15.4 (±1.3), 13.9 (±1.1) and 15.2 (±1.2) respectively. In addition, the mean blood lactate concentration was

highest in the variable pace time trial with 5.8 (± 1.7) $mmolL^{-1}$ followed by self pace (5.0±1.7) $mmolL^{-1}$ and least in even pace time trial with a concentration of (4.1 ±1.3) $mmolL^{-1}$. The association between blood lactate concentration and ratings of perceived exertion was found using the product moment correlation coefficient (r) which was +0.681. This means that there was a strong positive relationship (Cohen, 2002) between blood lactate concentration and the ratings of perceived exertion.

4.4 Discussion

4.4.1 Mean gross mechanical efficiency

The effect of three types of pacing on the energy expenditure during a 20-km cycling time trial was investigated. The mean gross mechanical efficiency of 18.1±1 % determined in this study corresponded to previous data obtained for nonprofessional cyclists (Chavarren, and Calbet 1999; Moseley and Jeukendrup, 2001).

4.4.2 Total work done by cyclists

The total work done by all the cyclists was lowest for variable pace trial and highest for self pace trial, but there were no significant difference in the total work done among the three pacing trials. These results are in agreement of what was found in certain research studies that investigated shorter cycling distances (Hettinga, et al., 2006; Hettinga et al., 2007) where they found also that even pace was found to be the preferred type of pacing. For a particular type pacing to be employed for a prolonged or endurance exercise, therefore, depends on the distance of the race. As shown in Figure 4.3, it was observed that the energy derived from the aerobic energy system pathway for even pace as greatest and least for variable pace. In contrast, it was observed that the energy derived

through anaerobic metabolism for variable pace was greatest and least in even pace even though there was no significant difference in the total energy used by the cyclists for each pacing (Figure 4.4). Therefore, pacing did not necessarily favour a total economy of resources but rather influenced the way that energy was produced from the energy system pathways and it also helped to establish a suitable internal environment for the completion of physical activity.

4.4.3 Hazard score Index, RPE and blood lactate concentration

There is the tendency of the athletes to change pace (even in self pace time-trial) during competitive simulations which is partially related to how they feel at the moment (RPE) and to how much proportion of the event remains. The computation of a simple index by merging these two predictors produced the Hazard Score that represents the hazard of a competitively catastrophic collapse faced by the cyclist in a competition or a race, which seems to a good prediction of subsequent behaviour. It was found that the variable pace time trial caused the highest homeostatic disturbance, and least in self pace time trial as part of a conservatory nature or behaviour of the human organism. Interestingly, apart from the positive linear relationship between the rating of perceived exertion and blood lactate concentration, both measures were highest for variable pace as compared to the other pacing time trials.

4.5 Summary

In this study, the energy produced from anaerobic and aerobic metabolisms for various types of pacing for a 20-km cycling time trial were investigated. It can be deduced that even pace was aerobic energy system dependent, and variable pace was anaerobic energy system dependent. Hence, one of the questions that arises

is whether a particular type of pacing favours a particular metabolic energy process (*i.e.* anaerobic or aerobic) and, if this is so, then there is a need to understand whether the human body system favours a particular energy system and optimizes performance accordingly. Furthermore, the hazard score index showed that the variable pace time-trial caused the greatest homeostatic disturbance as compared to the other pacing time-trials (self pace and even pace), and there was a correlation between blood lactate concentration and ratings of perceived exertion. As such, mathematical modelling and analysis was able to probe into the workings of the energy systems, and also attempted to predict the effect of pacing on the physical behaviour and performance from mathematical measures.

CHAPTER FIVE
Study Three

Nature and characteristics of the system control mechanisms underlying physiological data.

According to the literature review of the physiological control models, there is the presence of a control mechanism which optimises physical performance (Ulmer, 1996), and some physiological control models posit that the biological signals are complex but yet deterministic in nature (Lambert et al., 2004; Weir et al., 2006). Furthermore, St Clair Gibson and Noakes (2004) suggested that the neural integration of the afferent information from the various peripheral systems leads to an oscillatory power output and physiological responses during physical activity. The time constant (how fast a system returns back to baseline after a perturbation) of the extent to which a particular physiological system deviates from a metabolic setpoint (baseline), during exercise, may be associated to the homeostatic control of the interlinked physiological systems (Koeslag et al., 1997; St Clair Gibson and Noakes, 2004). Therefore, this experimental case study was conducted to investigate the hypotheses of these control models of exercise-induced fatigue using various mathematical methods.

5.1 Introduction

The changes in power output during exercise, previously associated with fatigue, has been recently been suggested to be related to a complex integrative control which involves the continuous interaction between all the physiological peripheral systems and central nervous system in a deterministic way (Lambert et al., 2004). The continuous change in the metabolic and physiological variables at rest and

during exercise, the deviation of a particular biological system variable from its metabolic setpoint value (*i.e.* normal state at rest for a healthy individual) and the speed at which this variable returns to its baseline point, are important factors in regulating homeostasis in any physiological system (St Clair Gibson, 2005). However, few studies (Atkinson et al., 2007; Tucker et al., 2006) have been conducted to investigate these physiological system control mechanisms despite the great need to investigate their nature and characteristics to better understand how these biological mechanisms regulate homeostasis, control human behaviour and physical activity (Atkinson et al., 2007; Lambert et al., 2004; Noakes et al., 2004; St Clair Gibson et al., 2005; Tucker et al., 2006) at rest and during exercise. From the second research study, the presence of a defensive mechanism was observed and a particular energy system was favoured at the expense of the other energy metabolic systems depending on the intensity and duration of the physical activity. Here, the aim of this study was to investigate the nature and characteristics of the system control mechanisms underlying physiological data using mathematical analysis to understand how these mechanisms control physical activity during exercise, and hence improve athletic performance accordingly, without any risk of physiological system failure.

5.2 Methods

5.2.1 Participants

Ten healthy and well-trained male cyclists participated in this study and this research study was approved by the Ethics Committee of the School of Life Sciences at Northumbria University at Newcastle.

5.2.2 Data Collection

The data collected from Study 2 was used to investigate the system control mechanisms underlying physiological data and these data included volume of oxygen consumption ($\dot{V}O_2$), heart rate (BPM) and power output (W). Apart from physiological changes during self pace, variable pace and even pace cycling time trial, blood lactate concentration was monitored at 4 kilometre interval while rating of perceived exertion (RPE) using Borg scale (Borg, 1998) was measured at 2 kilometre distance interval.

5.2.3 Data Analysis

5.2.3.1 Distribution of power output during the self pace 20-km cycling time trial

Frequency histogram power distributions (See Figure 5.1) for all cyclists for self pace 20-km cycling time trials were analysed to assess how the system control mechanisms control behaviour and activity using indirect measurements as described below. The frequency of a constant pacing is zero and as for the variable pace is mono-frequency that depends on the period of the pulse-wave variable power output signal.

5.2.3.2 Presence or manifestation of the system control mechanisms underlying physiological data

The power spectral densities of the self pace power outputs of the cyclists were computed to determine how spectral power varies with normalized increasing frequency which is the ratio of the frequency components to the maximum frequency component of the power output signal. Depending on the spectral power behaviour of the physiological signal, this would determine the presence or absence of system control mechanisms in the biological data (Tucker et al., 2006).

5.2.3.3 Nature of metabolic setpoint function using fractal analysis

Normally, for healthy individuals, the biological processes that occur within a physiological system set the level of metabolic activity whether at rest or during exercise. In order to investigate the nature of the metabolic setpoint function (St Clair Gibson et al., 2005), in terms of similarity or redundancy, fractal analysis was applied to the power output of the various pacing trials as well as the physiological data (*i.e.* heart rate and volume of oxygen consumption) for all pacing trials that were generated for the 20-km cycling time trial (as described in section 4.2). The fractal dimension was determined using Higuchi's algorithm theorem (Higuchi, 1988) and tested using Weierstrass synthetic function of known dimension (Mandelbrot, 1982; Mandelbrot, 1983). The fractal dimension (FD) is a means to measure the complexity of a signal, and the more complex a signal is, the higher the FD value. The hypothesis was based upon whether the fractal dimensions for all the different types of physiological data were similar for all athletes so as to observe any redundancy in the system control mechanisms that influenced the metabolic setpoint function (Koegslag et al., 1997; Lambert et al., 2004).

5.2.3.4 Characteristics of the complex system control mechanism using recurrence analysis

In order to determine the characteristics of the complex system control mechanisms, recurrence analysis was used to locate rhythms and patterns in the data (Trulla et al., 1996; Zbilut et al., 1995). For example, if the current state (value) of a signal for time t_1 is the same as a future time t_2, then it is called a recurrence in time, and this recurrence is represented by a black dot, and these black dots form patterns. Then, from this type of plot, quantitative measures (as

described in section 2.7.4) such as the recurrence rate (RR), the determinism (DET), and the Trapping Time (TT) were used to find the characteristics for a particular physiological system (Zbilut et al., 1995; Zbilut et al., 1992).

5.2.3.5 Data and statistical analysis

These data were analysed using Matlab software platform version R2008a and SPSS software was used for statistical analysis. All the measured and computed data derived in this study were tested for parametricity using Kolmogorov-Smirnov (K-S) test (Fasano and Franceschini, 1987). A One-Way Analysis of Variance (ANOVA) with repeated measures was used to compare the means of these computed variables and then Tukey's HSD was used as post-hoc test to find whether the difference was significant or not (Field, 2009). A significant difference occurred when statistical p was less than 0.05.

5.3 Result

5.3.1 Distribution of power output during the self pace 20-km cycling time trial

Figure 5.1 represents the frequency histogram distribution of self pace power output (W) for each cyclist with increasing rank order in terms of finishing time from top (Figure 5.1a) to bottom (Figure 5.1j). It was shown that the self pace power distributions of 90% of all cyclists displayed a negative skewness of mean value -1.29 (±0.75) which means that the mass of the power distribution is concentrated on the right of the frequency histogram mean, and the average kurtosis or "peakedness" of the self pace power distribution curves was 14.8 (±9.6). Apart from the skewness and peakedness of the self pace power distributions, it was also observed that the density of the self pace power distributions varied among the ten cyclists which meant that some self pace power

distributions appeared to be discrete continuous (a small cluster of similar power outputs) as shown in figures 5.1(f), 5.1(i) and 5.1(j). Most of the frequency histogram self pace power distributions showed simple inverted bell-shaped distributions.

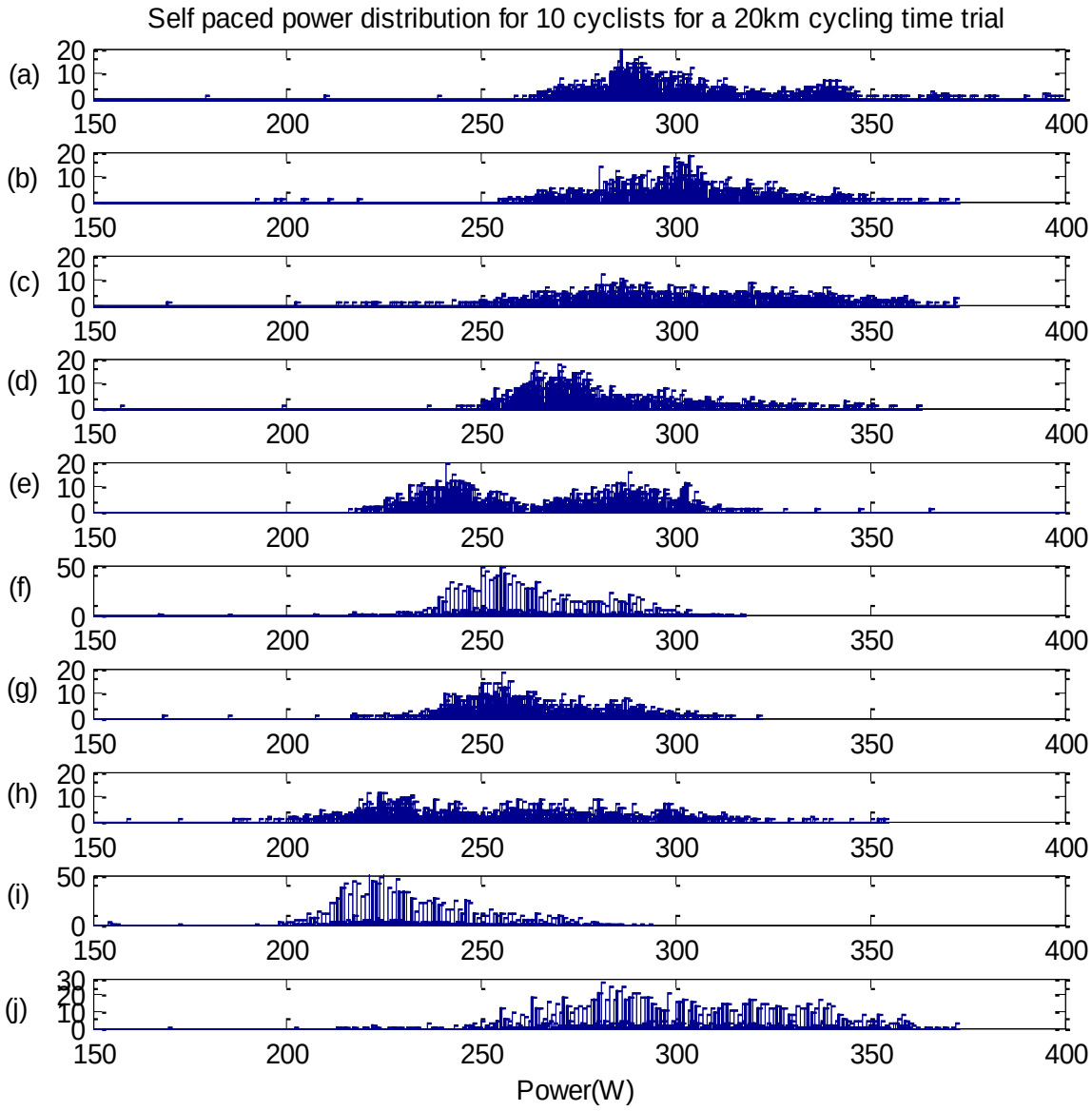

Figure 5.1: Frequency histogram distribution for self pace power output for all cyclists (n=10) with increasing rank order from top figure 5.1(a) to bottom figure 5.1(j). Some power output distributions were discrete continuous 5.1(f), 5.1(i) and 5.1(j) and the other power output distributions spanned over certain range.

5.3.2 Presence of the system control mechanisms underlying physiological data

In Figure 5.2, the power spectral densities of the self pace power outputs of various rank cyclists showed clearly the $1/f$ – scaling factor (where f is frequency) or the inverse proportionality of spectral power with increasing normalised frequency. This means that the self pace power output distributions for all cyclists with increasing frequency showed the same trend. The spectral density (power of signal vs. frequency) or the spectrum of a signal captures the frequency content of the physiological signals and helps in identifying periodicities (*i.e.* occurrences at regular intervals) but temporal information is lost (Davenport and Root, 1987).

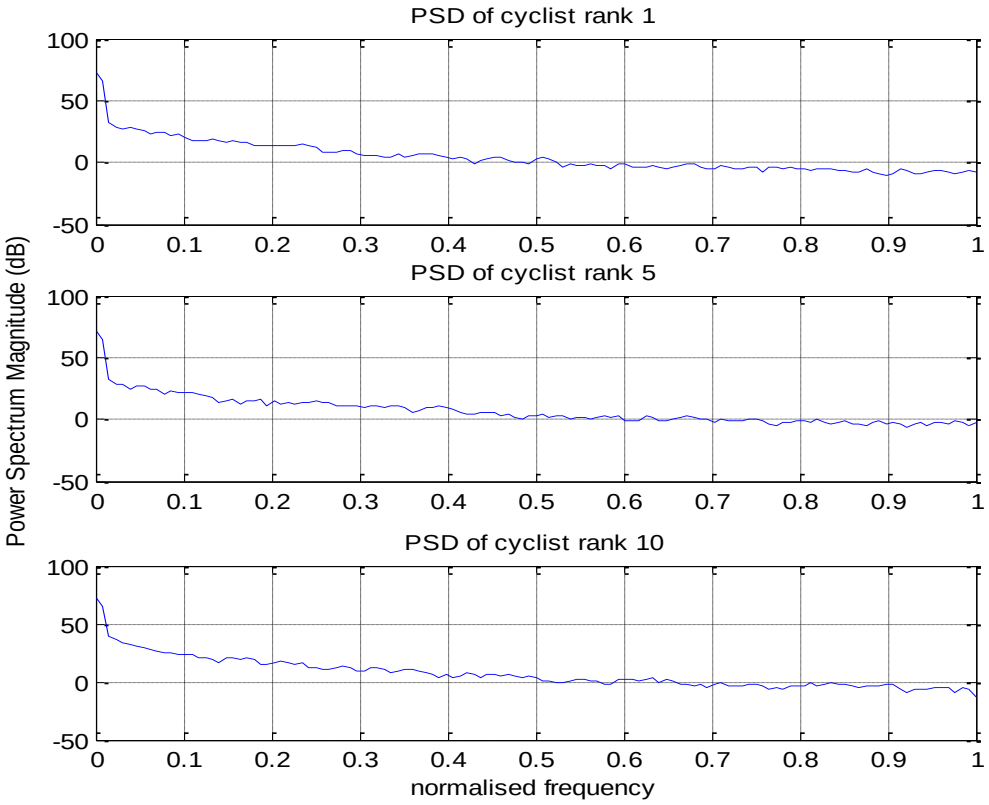

Figure 5.2: Power Spectral Density vs. normalized frequency (Hz) for the self pace power output for cyclists ranked 1st , 5th and 10th from top to bottom for 20km cycling time trial. The power spectrum magnitudes (dB) were negative after the normalised frequency 0.5 because they were close to zero.

5.3.3 Nature of metabolic setpoint function using fractal analysis

For even pace trial (Figure 5.3), the fractal dimension was constant with value 1 followed by the self pace trial and variable pace trial with mean values 1.33 (±0.03) and 1.38 (±0.01) respectively.

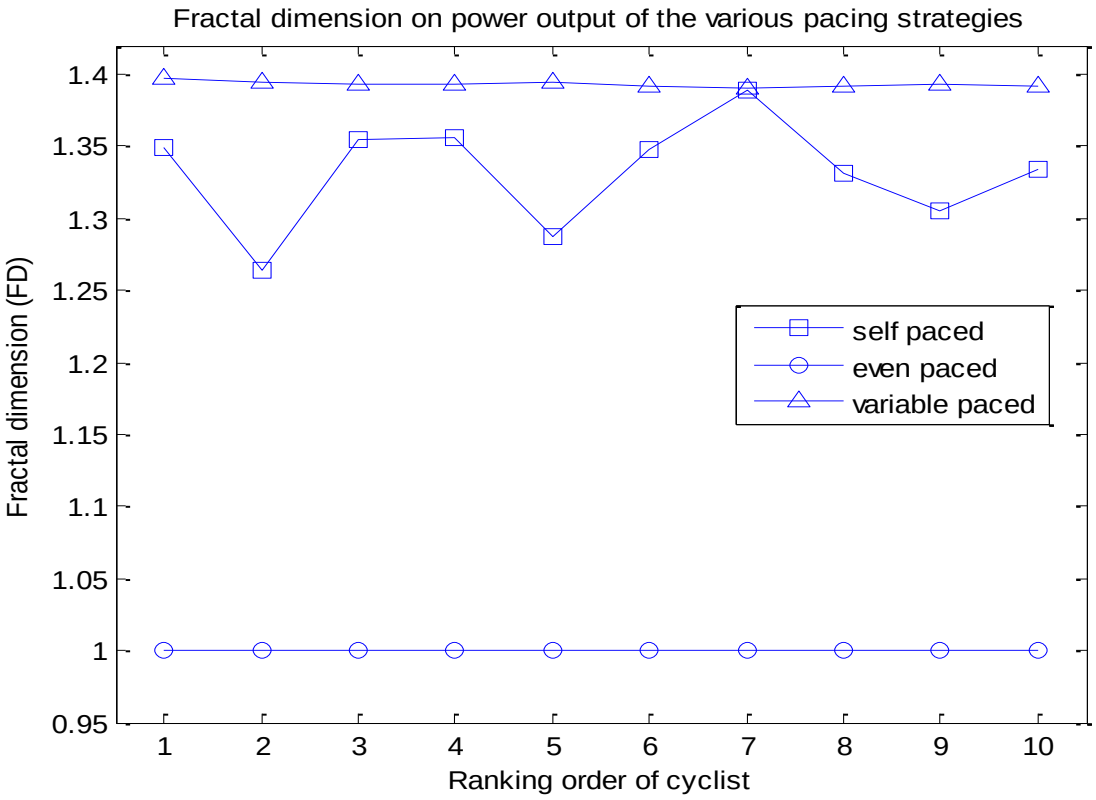

Figure 5.3: Fractal dimension for power output for all pacing time-trials and for all the cyclists (n=10).

In addition, the mean fractal dimension for the volume of oxygen consumption for all individuals for all pacing trials was 1.16 (±0.03), and that for heart rate was 1.38 (±0.08). This means that the complexity of the biological activities which occurred, for any particular physiological system, was similar for different human organisms. However, there was a significant difference between the fractal dimension

between heart rate and volume of oxygen consumption ($p < 0.05$) which means that each physiological system had different complexity.

5.3.4 Characteristics of the system control mechanism using recurrence analysis

The recurrence plots depicting the patterns for variable pace, even pace and self pace are shown in Figure 5.4. The left hand side figures represent the one dimensional view of the variable pace, even pace and self pace power output signals, and the right hand side figures are their corresponding recurrence plots from top to bottom respectively.

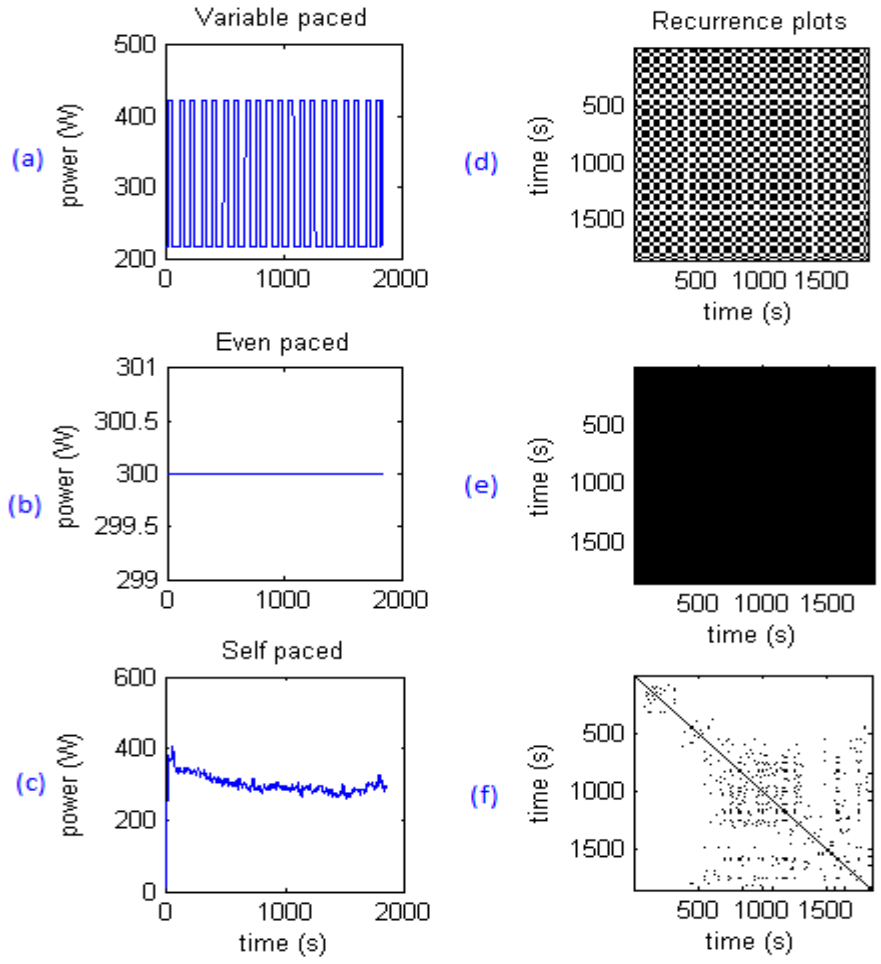

Figure 5.4: Recurrence plot depicting the patterns for variable pace, even pace and self pace power outputs for one particular cyclist (figures 5.4(d), 5.4(e) and 5.4(f)) and its corresponding one dimensional view (figures 5.4(a), 5.4(b) and 5.4(c)).

The pattern of the variable pace power output resembled a checker board (periodic change), that of even paced was homogeneous (no change) and that of self pace was complex as shown by the cluster of points that form small-scale structures (including single dots, diagonal line) and large-scale structure (representing general changes). All these were further quantified and interpreted using recurrence quantitative analysis measures to find out about the determinism, trapping time and recurrence rate (or probability that a particular physiological state happen at a time) of the physiological signals.

Table 5.1: The mean (± standard deviation) Recurrence Quantitative Analysis (RQA) measures for each pacing time-trial for the volume of oxygen consumption ($\dot{V}O_2$/L·min^{-1}) for all cyclists (n = 10).

RQA measures	**Self Pace**	**Even Pace**	**Variable Pace**
Recurrence Rate (%)	8.5 ± 0.9	10 ± 1.6	8.3 ± 0.9
Determinism (DET)	0.29 ± 0.04	0.31 ± 0.06	0.30 ± 0.05
Trapping Time (TT/s)	2.5 ± 0.1	2.5 ± 0.2	2.5 ± 0.1

Table 5.2: The mean (± standard deviation) Recurrence Quantitative Analysis (RQA) measures for each type of pacing for heart rate (BPM) for all cyclists (n = 10, the asterisk symbol * represents statistical significance with statistical $p < 0.01$).

RQA measures	**Self Paced**	**Even Paced**	**Variable Paced**
Recurrence Rate (%)	11.2 ± 0.05	11.2 ± 0.06	8.5 ± 0.03
Determinism (DET)	0.89 ± 0.18	0.92 ± 0.11	0.86 ± 0.18
Trapping Time* (TT/s)	7.0 ± 3.9[a]	10.9 ± 8.8	6.7 ± 3.2[b]

The superscript **a** denotes significant difference between even pace trial and self pace trial; superscript **b** denotes significant difference between even pace trial and variable pace trial.

5.4 Discussion

5.4.1 Distribution of power output during the self pace 20-km cycling time trial

All self pace power distribution curves had high and positive kurtosis of mean +14.8 where values above 3 are considered a sharp peak (Dodge, 2003). Therefore, this means that the cyclists varied their cycling power output in a specific and narrow range based on the very sharp peak of their power output frequency distribution. Moreover, their self pace mean power output was lower than their corresponding median power output. This accounted for the negative

skewness of their power histogram distribution curves.

5.4.2 *Presence of the system control mechanisms underlying physiological data*

The power spectral densities of the self pace power outputs showed the $1/f$ scaling factor, where f is the frequency which was in agreement with the previous studies by Tucker et al. (2006) who emphasized that this could not be a consequence of noise, since noise would have a broad and constant spectrum for any particular frequency (f). It was also observed that for all cyclists the highest spectral power occurred at a very low frequency followed by small ripples or peaks in a $1/f$ -scaling manner. The presence of this inverse frequency scaling factor and multiple frequency peaks suggest that the system control mechanisms appeared similar in different human organisms (Tucker et al., 2006). These were investigated further in the following section using fractal analysis, and in Study 4 using wavelet analysis to understand what was happening at the low and high frequency bands.

5.4.3 Nature of metabolic setpoint function using fractal analysis

To verify how far these system control mechanisms, present in the human body physiological systems, are similar, fractal analysis was used. In Figure 5.3, it was observed that the complexity of a self pace power output signal lies in between the fractal dimension (FD) values obtained for variable paced power output and that of even paced power output. The percentage error in estimating the fractal dimensions was 3% since fractal dimension cannot be derived exactly (Dubuc et al., 1989). The complexity of the system control mechanisms, for a particular physiological system, depends on the number of independent sources controlling that system (Hoyer et al., 2007; Pajunen, 1998). As such, for the complexity of a signal to increase, there should be an increase in the number of independent

control centres modulating a physiological system. This results in an increase in the information processing load between the central system (*e.g.* the brain or the brain stem) and the peripheral systems that may be the cause for the sensation of fatigue (Okamura, 2007; Okogbaa, 1994; St Clair Gibson et al., 2006) which was experienced by the cyclists as shown by their high RPE values (see results in section 4.3.5). Furthermore, the difference in complexity of the biological system activities may be attributed to the notion that when the system is more complex, the more robust it might be, or it does not allow the system to collapse completely even if a single control mechanism fails (Lambert et al., 2004; Pincus, 1994).

5.4.4 Characteristics of the system control mechanisms using recurrence analysis

A recurrence quantitative analysis (RQA) was applied to the physiological data to determine the characteristics of the system control mechanisms underlying those physiological data. It was found for the volume of oxygen consumption physiological variable that there was no significant difference between the RQA measures for all cyclists performing the different pacing trials ($p > 0.05$). The mean recurrence rate (RR) was 9%, mean determinism (DET) was 0.29, and trapping time (TT) was 2.5 s, and there was no significant difference ($p = 0.03$) between the RQA measures for heart rate for all pacing trials, and the mean values of RR was 10%, DET was 0.89, and TT was 8.2 s. However, the difference in the trapping times between the respiratory system and the cardiovascular system biological activities implied that heart rate activities stayed longer in a particular state than that of the physiological activities of the respiratory system according to the trapping time values. Interestingly, the heart rate activities were more deterministic than the respiratory system physiological activities. This suggests that future

activities of the heart could be predicted much more easily than the more stochastic process of the physiological data produced from the respiratory system despite both recurrence rates (RR) being low. Moreover, the trapping times for the heart rate activities were significantly different ($p < 0.01$) for each pacing trial. There was a tendency for the imposed pacing trials (even pace and variable pace) to force that physical system to mimic its behaviour (Table 5.2) as reflected by the duration of the mean trapping time, to remain in a physiological state was highest in even pace (10.9 s), and shortest in variable pace (6.7 s).

5.5 Summary

In this study, it was observed that the 1/f-scaling factor was present in the spectral power outputs suggesting that the presence of system control mechanisms in the physiological activities. Fractal analysis of power outputs showed that an even pace trial had the smallest fractal dimension value as compared to self pace and variable pace, and hence it can be useful tool in distinguishing particular pacing trial for optimising performance. Moreover, each physiological organ system had different fractal dimensions which suggest that the complexity of the homeostatic control mechanisms regulating these systems are different, and this may be associated to the robustness of the system to physiological failure. For specific duration of a race, finally, it was observed that each physiological system had its own characteristics based on the different recurrence quantitative measures and this means the system control mechanisms controlling any particular physiological system are different in nature. Henceforth, recurrence analysis can be used as an important graphical and quantitative tool to determine the stability (*i.e.* in terms of trapping time and recurrence rate), and predictability (*i.e.* in terms of determinism) of a physiological system during exercise.

Chapter Six

Study Four

Mathematical analysis of the system control mechanisms to investigate indirectly how a central regulator paces the human body during a 20-km cycling time trial

In exercise physiology, there is an increasing need to assess the various complex physiological signals to verify the theories of certain physiological models (Lambert et al., 2004; St Clair Gibson and Noakes, 2004; St Clair Gibson et al., 2006). These exercise physiology models posited that physical exercise is modulated by a central regulator in the CNS, and the human body works as a complex integrative system. From the previous experimental case studies, it was found that there was the presence of a control and that the physiological system activities had different characteristics but not much was known about how the system control mechanisms sustain homeostasis in any physiological system especially during physical activity. Therefore, this study utilised a different mathematical method to investigate how the physiological systems are regulated.

6.1 Introduction

In order to find how the physiological systems are controlled, a mathematical method was needed to assess the biological activities both in time and frequency. However, time-based and frequency-based mathematical analyses are not suitable for the exploration of the irregular and non-stationary patterns of the complex biological signals (Mallat, 1989). Therefore, the continuous wavelet transform (CWT) was utilised to conduct time-scale analysis of the real-time signals which occur at every scale and time-position unlike the Discrete Wavelet

Transform (Rioul and Vetterli, 1991). The advantage of CWT is that it enables any changes at different frequency bands of the physiological signals to be observed in order to provide an indirect assessment of corresponding brain functions (David et al., 2007).

6.2 Method

6.2.1 Participants

Ten healthy and well-trained male cyclists took part in this research study, and it was approved by the Ethics Committee of the School of Life Sciences at Northumbria University. Their mean (± standard deviation) height and BMI were 1.77(±0.06) m, and 24.2 (±1.8) $kg \cdot m^{-2}$ respectively. The age of the participants ranged from 25.5 to 40.1 years. The data as described in the previous two chapters were used to investigate how the system control mechanisms modulated physical activity.

6.2.2 Data analysis

The Matlab software platform version R2008a and Wavelet Toolbox™ 4 were used for this research study. The continuous wavelet transform (CWT), using Morlet wavelet, was applied to physiological signals including volume of oxygen consumption, heart rate and power outputs (which integrate all the physiological activities of the various physiological systems) to obtain continuous wavelet spectrum coefficients. These coefficients were then subdivided into regions or bands that were Ultra Low Frequency (ULF), Low Frequency (LF) and High Frequency (HF) bands. The observed frequency regions were then classified in frequency bands (Addison, 2005; Yamaguchi, 2003) based on the wavelet transform scales (n) where integer variable n ranges from 1 to 256: the scales ranging from 1 to 8 were classified as high frequency; scales 9 to 64 were

classified as low frequency; and scales 65 to 256 were classified as ultra-low frequency (Lu et al., 2006; Pichot et al., 1999). The inverse relationship between the pseudofrequency (Hz) and the scale factor using Morlet wavelet is depicted in Figure 6.1. In this way the respective mean wavelet normalised powers (Indiradevi et al., 2007; Latka et al., 2003) were determined for each frequency band (Equ. 6.1) to investigate the frequency changes (if any) and monitor the respective duration of these events at various scales or frequencies of the physiological signals to determine how a central regulator regulates these physiological systems. The mean normalised wavelet spectrum power was found from equation 6.1 where the variable i represents the time events at every second of the physiological signal up to m which represents the total duration of the physiological activity whereas the variable j represents the scale number, and finally, Coefs (i, j) represents the continuous wavelet transform coefficients at time i and scale number j with limits n and m representing the scale number and time respectively.

$$P = \left\{ \frac{1}{max\ (Coefs(i,j))} \sum_{j=1}^{n} \sum_{i=1}^{m} Coefs\ (i,j) \right\} \quad \ldots \text{ Equ. 6.1}$$

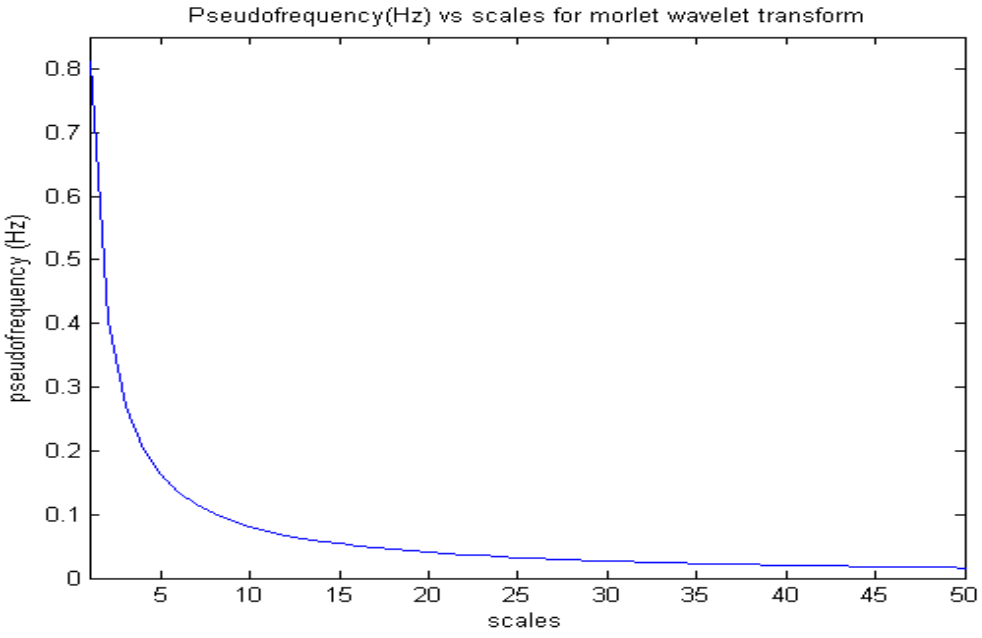

Figure 6.1: Relationship between pseudofrequency (Hz) and scales

6.2.3 Statistical analysis

The wavelet powers that were determined for self pace, even pace and variable pace trials were tested for parametricity using Kolmogorov-Smirnov test (Fasano and Franceschini, 1987). In addition a 3x3 (frequency band x pacing trial) factorial ANOVA with repeated measures was used to compare the means of the various frequency bands and any significant difference occurred when statistical p was less than 0.05 (Berger and Casella, 2001). Then, Tukey's HSD post-hoc test was used following the ANOVA to find any significant difference in the analysed variables (Field, 2009). If significance occurred, relationships between variables were then examined by calculating the product moment correlation coefficient r. Results were then presented as means ± standard deviation (S.D).

6.3 Result

6.3.1 Continuous wavelet transform on physiological data

Figure 6.2 displays the wavelet spectrum analysis profiles of the self pace power output for a particular cyclist together with the associated physiological data that include volume of oxygen consumption and heart rate activities during the 20-km cycling time trial. In these wavelet spectrums, the shift from a dark region (low) to light coloured region (high) represents a transition in the signal, or the occurrence of an event. For example, for the heart rate data (Figure 6.2), a dark region suggests that the heart rate activity is homogenous (*i.e.* there is no large fluctuation) whereas when a light coloured region is observed there is an abrupt change in heart rate activity. The higher amplitudes or change in transition are shown as lighter or brighter areas of the continuous wavelet spectrum. Using the two dimensional view of the signal, the general (large-scale structure) and local

(small-scale structure) behaviour and characteristics of the signal in time are clearly shown, which are not obvious from the one dimensional view of the raw physiological signals.

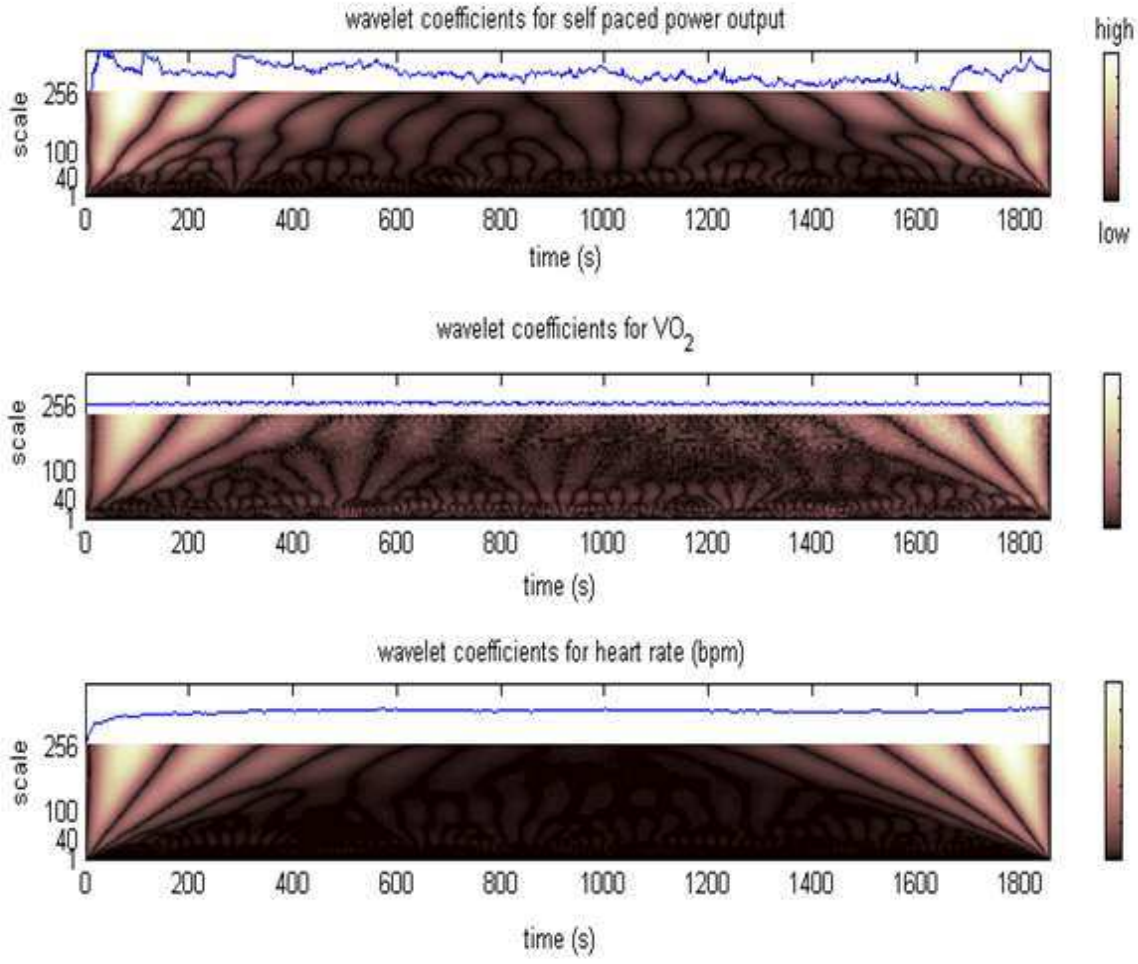

Figure 6.2: Continuous Wavelet Transform (Scale vs. time) on self pace power output, volume of oxygen consumption and heart rate for one particular cyclist who ranked 2nd.

In Figure 6.2, the x-axis represents time (in seconds) and y-axis represents the scale n which varies from 1 to 256. There were more abrupt changes at low frequencies than at high frequencies of the spectrum for all physiological signals, and there were more changes but less abrupt at high frequencies. By abrupt, it is meant that there is a big transition such as moving from a white region to a dark

region of the wavelet spectrum. So using the example of the heart rate data, it was observed that heart rate activity increased abruptly at the beginning of the race and at the near end of the race (about three minutes before the end of the race which represents the endspurt) depicted by the light coloured regions. Moreover, during the race there were frequent small changes in heart rate activities as shown by the dark regions. In this manner the bright colour (Figure 6.2) was classified as high transition (change in amplitude) whilst the dark colour was classified as low transition.

In addition to that, Figure 6.3 shows the variation in amplitude and frequency of the self pace power output profiles obtained after the wavelet transform was applied at three different scales 16, 128 and 200 to show the happenings or events in these regions. The y-axis of Figure 6.3 represents changes in the amplitudes of the power output signal for three chosen scales as drawn with a white line on the continuous wavelet transform figure and presented subsequently on three time-series figures.

It was clearly observed that there were high peaks at the start and end of the cycling time trial. By moving to the higher data capture rate or frequency, recurring changes at specific intervals about 200 seconds can be observed by the small peaks on scale 16 and positions in time as compared to the broader small ripple peaks depicted in the scales 128 and 200 between the time 200 seconds to 1400 seconds. Therefore, by moving to higher capture rate, it was possible to know precisely the happening of an event in time, as well as its corresponding pseudofrequency.

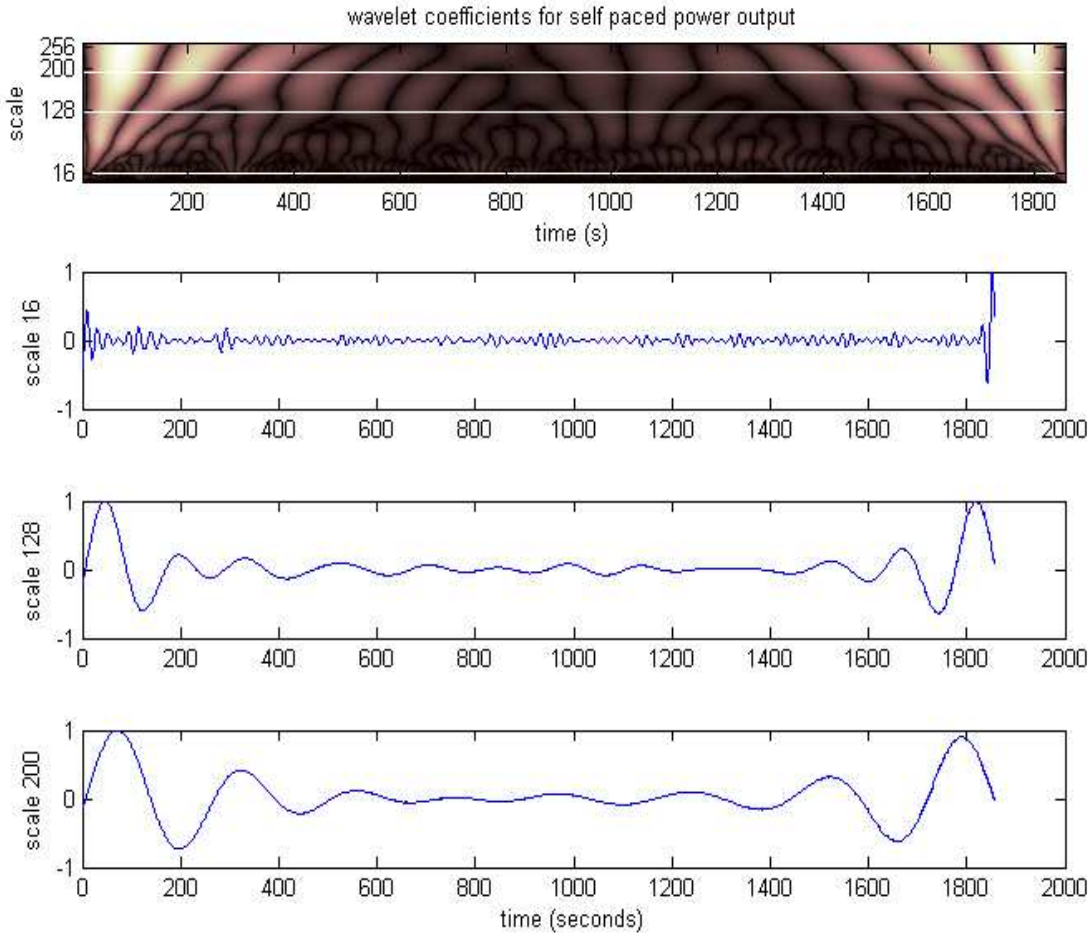

Figure 6.3: One dimensional view at specific scales (16, 128 and 200 from top to bottom respectively) of the wavelet coefficients obtained after CWT has been applied to self pace power output.

6.3.2 Wavelet band powers for volume of oxygen consumption for all cyclists

For the volume of oxygen consumption physiological activities, it was found that there was a significant difference ($p < 0.01$) between the ULF wavelet power as compared to both HF and LF wavelet powers that were determined for each type of pacing (Table 6.1). However, there was no significant difference ($p > 0.05$) between HF and LF wavelet powers (See Appendix D, Figure D.1). In addition a small decrease in ULF band power with increasing performance times of the

cyclists was also observed whereby more prominent decreases in ULF band power were evident for self pace (r = -0.77), and even pace (r = -0.66) trials than in variable pace trial(r = -0.16).

Variables	Self pace	Even pace	Variable pace
HF band power	0.033 ± 0.009	0.030 ± 0.012	0.025 ± 0.011
LF band power	0.024 ± 0.007	0.024 ± 0.008	0.024 ± 0.008
ULF band power	0.068 ± 0.004*	0.066 ± 0.003*	0.073 ± 0.005*

Table 6.1: This table represents the mean normalised power of the wavelet coefficients together with the standard deviation for volume of oxygen consumption ($\dot{V}O_2$) for each pacing and for each frequency band (HF, LF and ULF). The symbol * means there was a significant difference between that frequency band power and the other frequency bands with statistical $p < 0.05$.

6.3.3 Wavelet band powers for heart rate for all cyclists

Both ULF and LF band wavelet powers were not significant with mean values 0.06 (±0.4%) and 0.012 (±0.1%) respectively for all cyclists and for all pacing time trials (Table 6.2). For any particular pacing time-trial, there was no significant difference ($p > 0.05$) between HF and ULF ($p > 0.05$) but there was a significant difference between HF and LF bands ($p < 0.01$). Furthermore, there was a small positive correlation between the HF band power of heart rate physiological activities and performance times ($r = 0.3$; $p = 0.03$) (See Appendix D, Figure D.2).

Variables	Self pace	Even pace	Variable pace
HF band power	0.1658 ± 0.1805*	0.1452 ± 0.1585*	0.1339 ± 0.1331*
LF band power	0.0128 ± 0.001	0.022 ± 0.027	0.0132 ± 0.001
ULF band power	0.066 ± 0.005	0.079 ± 0.004	0.066 ± 0.004

Table 6.2: This table represents the mean normalised power of the wavelet coefficients together with the associated standard deviation for heart rate (HR) for each pacing and for each frequency band (HF, LF and ULF). The symbol * means there was a significant difference between that frequency band power and the other frequency bands with statistical $p < 0.05$.

6.4 Discussion

In order to analyse the physiological data to assess how a central control paces the body or the peripheral systems during exercise, a continuous wavelet transform was applied to these data to split these complex biological signals into specific scales and hence frequency bands (Mallat, 1989). For the self pace trial, there were sudden changes at low frequencies in the power output and physiological data especially at the start and at the end of the race (endspurt). These abrupt changes at low frequencies coincided with the acceleration at the beginning and at the end of the race (endspurt) and were consistent with common observations during a time-trial exercise (Ansley et al., 2004; St Clair Gibson et al., 2004; Tucker et al., 2006a; Tucker et al., 2006b). Furthermore, smoother frequent changes occurred at high frequencies for self pace power output. The factors that govern the power output are the force applied at the pedal by the cyclist as well as

the velocity (or cadence) at which the cyclist is moving (Gordon and Papadopoulos, 2004). These factors depend on the number and type of muscle fibres that are activated or recruited to generate the required force and velocity. According to McComas (1996), small motoneurones fire slowly and continually (observed as small changes in amplitude) and they innervate motor units that are resistant to fatigue as compared to large motoneurones which fire rapidly (the changes are for short duration) and in bursts (as shown by large amplitudes) that innervate motor units that are fatigable. This was perhaps why there were sudden changes in low frequency band as large motoneurones were triggered especially at the start and at the end of the race in contrast to slow and continual firing rates of small motoneurones that occurred in the low frequency bands during the race.

6.4.1 Wavelet band powers for volume of oxygen consumption for all cyclists

For volume of oxygen consumption, the ULF band wavelet power was highest as compared to the other frequency bands for the whole duration of the race. Therefore, this might be the frequency band where there was interactive communication between the central regulator to this particular physiological system. According to Sherwood (2005), it is the brain stem that consists of the respiratory control centers and generates the periodic pattern of breathing (Sherwood, 2005). In addition, there was a slight decrease in ULF band wavelet power with increasing cyclists' performance times, and this suggests that this control centre used this frequency band to regulate this particular physiological system (via feedforward and feedback information) which subsequently affected the sport performance of the cyclists.

6.4.2 Wavelet band powers for heart rate for all cyclists

As for the observed heart rate activities, both LF and ULF band powers were almost constant for all cyclists. In addition, the significant difference in the HF band power as compared to the other frequency bands, however, means that there was some external drive or controller (Lu et al., 2006; Pichot et al., 1999; Xu et al., 1998) which was using this frequency band, or specific range of frequencies, to control this particular physiological activities of that particular athlete despite the poor correlation between HF band power and increasing performance times.

6.5 Summary

In this study, the system control mechanisms underlying physiological data were investigated to see how a central regulator within the central nervous system paces the human body during exercise. It was found that the ULF band power for volume of oxygen consumption was highest for all cyclists and this ULF power decreases with increasing cyclists' performance times. Moreover, there was a significant difference in the HF wavelet band power as compared to other frequency bands (*i.e.* HF was highest for heart rate activities for all cyclists). As such, there may be a regulator that paces the human body which uses specific frequency bands to control and communicate with the different physiological peripheral systems simultaneously so as the physical activity is completed without homeostatic failure. The strength in the wavelet power in the ULF band for respiratory system and HF band for heart rate activities suggest that these frequencies in fact depicted the behaviour of the sympathetic or parasympathetic drive which means that these system control mechanisms role were to reduce or increase such physiological system activities to complete a race or competition without catastrophic physiological system failure.

CHAPTER SEVEN

Study 5

The effect of an exhausting exercise bout on cognitive performance

According to certain theoretical control models, exercise-induced fatigue is not always peripheral, and it is the brain that causes the sensation of fatigue owing to a decrease of metabolic resources to and from the brain, or to a central activation process that regulates behaviour and physical performance (Davis and Bailey, 1997; Fowles et al., 2002; Gonzalez-Alanso et al., 1999; Lambert et al., 2004; Nielsen and Clausen, 2000; Parkin et al., 1999; Nybo and Nielsen, 2001; St Clair Gibson and Noakes., 2004). Therefore, this experimental case study was conducted to assess whether there was finite level of metabolic energy resources in the brain, by performing both mental and physical tasks to exhaustion. In so doing, it was hoped to be able to observe the effect of these exhausting tasks on cognitive performance.

7.1 Introduction

Up to now, very little is known about the psychophysiological mechanisms that underlie mental fatigue and the cognitive functions (Cox, 1994). Specifically within athletics, the ability to allocate and maintain attention during sporting competition can be as much taxing as the physical exertion associated with the sporting activity for successful performance (Nideffer, 1993). However, minimal research has evolved in exploring cognitive fatigue as a possible performance mediator within athletics and hence, the influence of fatigue on attention and performance is

still unclear. The studies that examined the impact of either acute or long-term exercise on cognition are equivocal, and it was suggested that physical exercise has a possible small positive effect on cognition (Etnier, 1997). Further studies are, therefore, needed to investigate the effects of exercise on cognitive performances (Etnier, 1997; Tomporowski, 2002). Based on the principle that processing in the brain is competitive, and it has finite metabolic resources, a new mechanistic explanation for the effect of exercise on the brain function, called the "transient hypofrontality hypothesis" was developed (Dietrich, 2003). This hypothesis states that during physical exercise the extensive neural activation which is needed to run motor patterns, assimilate sensory inputs, and coordinate autonomic regulation causes a decrease in brain activity (Dietrich, 2003; Dietrich and Sparling, 2004).

In addition, the limitation of most studies related to the effect of exercise on cognition used only reaction time as the sole measure of cognition even in studies employing complex behavioural tasks (Chmura et al., 1994). Those relatively few studies (Delignières and Brisswalter, 1995; Paas and Adam, 1991) that included response accuracy in their analysis, reported that there was either no change or increased accuracy with exercise. Therefore, by conducting this cognitive fatigue study, it was hoped to observe the response of cortical activities to an exhausting physical exercise, and how physical activity affects cognitive performance in terms of reaction time and accuracy as they seemed to reflect important aspects of cognition. The next section describes the methodology used to conduct this experimental case study.

7.2 Methods

7.2.1 Participants Details

This research study was approved by the Ethics Committee of the School of Life sciences, Northumbria University at Newcastle. Twelve healthy and right-handed participants (6 males and 6 females) were recruited and their mean (± standard deviation) height was 1.73 (±0.08) m. The participants' age ranged from 25.9 to 33.3 years, and their body mass ranged from 56.6 kg to 82.8 kg. All the participants came to the neurophysiology laboratory on three separate occasions with at least one week apart so as to reduce any learning effect (Lord et al., 1998; Wright, 1936). In addition, these volunteers were moderately to highly mentally and physically active as they were required to complete a series of cognitive tasks lasting for half an hour on two occasions, and also on one occasion they had to cycle as hard as they could for about half an hour or until they felt they could not continue this physical activity anymore.

7.2.2 Description of the cognitive tasks

The rapid visual information processing (RVIP) and modified stroop (MST) tasks were used in this research study, and the psychological strain which was placed by these tasks on the participants was mostly cognitive (Capuron et al., 2005; Coull et al., 1998). Hence, they needed to sustain attention for half an hour for them to be accurate and quick in their responses during the cognitive trials. The cognitive tasks are described as follows.

For the modified stroop task (MST), the participants had to respond to the colour of the word appearing at the centre of a computer screen (Red, Blue, Yellow and Green) by pressing respectively and quickly the numerical keys (1, 2, 8 and 9) on

the keyboard. Moreover, if the word that appeared on the screen was written in grey, they were required to respond to the word. For instance, if the word YELLOW was written in grey colour, then the participants would need to press the numerical key '8' which was associated to yellow colour. In addition, during the modified stroop task, they had to count mentally the number of white squares (size of 9 cm by 9 cm) and white circles (diameter size was 9 cm) that appeared randomly and sparsely at the centre of the screen. The duration of this type of cognitive task was 5 minutes.

As for the rapid visual information processing (RVIP) task, the participants were required to respond to a specific sequence (odd or even) of integer numbers from 0 to 9 which appeared one at a time every 600 ms on the computer screen. For example, when they noticed three consecutive odd numbers (*e.g.* 3, 5, 7) or three consecutive even numbers (*e.g.* 2, 8, 6), they had to press the 'spacebar' on the keyboard as quickly and accurately as they could. The duration of this cognitive task was also 5 minutes. And then, these cognitive tasks were alternately presented to the participants for a period of 30 minutes so that there were in all three RVIPs and three MSTs which represented the cognitive battery test.

7.2.3 Hardware and software resources

The material resources that were used for this study comprised of Research Powerlab, Dual and Octal Bio Amp systems (Powerlab, ADInstruments, Australia) for the recording of the electrocardiogram (ECG) and electroencephalogram (EEG) physiological data. The electro-caps (the electro-cap size can be small, medium or large) consist of Ag/AgCl electrodes embedded in the elastic electro-cap fabric to record EEG activities from the scalp, and these physiological data

was transmitted to the powerlab systems via an electro-cap interface (Electro-Cap International, Inc., USA). Moreover, the ECI electro-gel was used to reduce the resistance between the EEG electrodes and the scalp, and a digital multimeter (Draper 52320, UK) was used to measure this impedance. In addition, a '0-volt' potential 8-lines cable was built and used as a reference baseline voltage to measure the EEG potentials. Couple with that, a parallel communication interface port was built and tested to send 8-bit parallel data from the Research Powerlab to the installed E-Prime software workstation. This communication interface was used to send 8-bit data to 'timestamp' the responses of the participants while responding to the visual stimuli on the computer screen. These 8-bit data actually represented 2-digit numbers called digital bytes that were assigned 'comment texts' (*i.e.* blue, red, green, yellow, square, circle, odd and even) to display on the real-time physiological data upon the trigger of the corresponding visual stimuli. These would help to convert the continuous EEG data into data epochs time locked to specified event types for analysis purposes. Finally, the software that were used in this experimental case study were Chart 5 for Windows (Research Powerlab) to record and process the physiological signals, the E-Prime software version 2.0 to implement and conduct the mental fatigue tasks, and Matlab software 7.0 for data analysis.

7.2.4 Study protocol and procedures

On the first visit to the physiology laboratory, the participants completed each a screening health questionnaire to determine their eligibility for taking part in this research study and then, they were each assigned an identification number to protect their anonymity. The right size electro cap was identified using the electro-cap head tape measure (Electro-Cap International, Inc., USA) for each participant,

and then they were given each a practice session on the cognitive tasks that they would need to complete during their second and third visits. Moreover, this first visit allowed the participants to familiarise themselves with the laboratory environment and the procedures.

During the second visit (also named the 'control' experiment), the participants completed first the Multidimensional Fatigue Inventory (MFI-20) questionnaire which comprised of 20 items assessing the general fatigue, physical fatigue, mental fatigue, reduced activity and reduced motivation. Before starting the cognitive battery test, the participants sat comfortably facing the computer monitor at a distance of about 60 cm (Corr, 2002) and the appropriate EEG electro-cap was fitted onto the participant's scalp according to the manufacturer's instructions (Electro-Cap International Inc., USA). Next, a blunted needle was used to fill each electro-cap electrode, relevant to this study, with the ECI electro-gel to ensure the impedance between the EEG electrode and the scalp was less than 5000 Ω using the digital multimeter. Then, EEG activities at the frontal midline (F_z), central midline (C_z), and parietal midline (P_z) were recorded while the reference Ag/AgCl electrodes were attached to A_1 representing the left ear lobe (Uetake and Murata, 2000), and the ground electrode was located at AF_z representing the Anterior Frontal of the scalp (Boksem et al.,2006). Furthermore, the ECG electrodes were attached to the arms and wrist of each participant according to the manufacturer's specifications (Powerlab SPB08c, 2004), and connected to the powerlab systems to record the heart rate activities and the corresponding beat-to-beat intervals. The sampling frequency was set at 400 Hz which was sufficient to capture the EEG and ECG activities based on Nyquist's criterion (Shannon, 1949).

Then, after each 5 min-block of cognitive task (either RVIP or MST), they had to complete the visual analogue scales (VAS) so that their mental fatigue, physical fatigue and concentration were quickly and easily monitored during the trial. For these visual analogue scales, the participants were required to mark in-between the horizontal scales that consisted of two extreme marks '0' and '10' representing low and high respectively. Afterwards, at the end of the cognitive experiment, they had to fill again the MFI-20 questionnaire to compare any changes in the subjective feeling measures between pre and post the cognitive battery experiment. As a preliminary procedure, a pilot study was conducted to test the reliability of the MFI questionnaire items using Cronbach's alpha (Cronbach, 1951; Schmitt, 1996). The value of the Cronbach's alpha was found to be 0.82 which showed a reliable tool for assessing the internal consistency of a psychometric test in representing subjectively the fatigue felt by these participants for this type of cognitive experiment (Cronbach, 1951; Schmitt, 1996).

On their third visit (also named as the 'exercise' experiment), the participants each performed, first of all, an exhausting cycling exercise bout for about half an hour. They wore comfortable clothes and footwear to perform this tiring cycling bout on the Velotron (VelotronPRO, RacerMate Inc., USA) whereby they were instructed to cycle as hard as they could till they could not continue this physical activity anymore. After completing this physical activity, they had to complete the MFI-20 questionnaire, and then, during the cognitive trial, they were asked to complete the series of RVIP and Modified Stroop tasks similar to the second visit, while physiological data that were EEG and ECG were recorded following the same procedure as described for the second visit. Finally, after completing the cognitive

battery test, they filled again the MFI-20 questionnaire. As precautions, the participants were requested to try not to blink while responding to the visual cues, during the cognitive experiments, to reduce interference of the electrooculogram activities (Erfanian, and Mahmoudi, 2005) to the measured EEG signals. Apart from the visual cues on the screen, there were neither other visual stimuli nor auditory stimuli that would distract the participants from the cognitive trials.

7.2.5 Data analysis

Both EEG and ECG activities were recorded while the participants performed the series of cognitive tasks in both experimental conditions (control and exercise). The heart rate activities could provide information about the instantaneous intensity of the physical and mental exercise but it could not measure the additive effect of the physical and mental stress that lasted for a period of time (Pichot et al., 2002). As recent research (Pichot et al., 2002) focused on heart rate variability (HRV) in evaluating cumulative fatigue subjected to a physical activity, therefore, both beat-to-beat intervals and heart rate were included to find whether these variables could show any physiological difference among the participants subjected to these two experimental conditions (control vs. exercise). The following subsections explain in more details the recording and analysis of the EEG activities as well as the event related potentials (ERP) data.

7.2.5.1 EEG analysis

EEG activities were recorded continuously from the midline placements F_z, C_z and P_z according to the international 10-20 system electrode placement using the Ag/AgCl electrodes embedded in the elastic electro cap fabric (Figure 7.1). The cortical EEG activities were amplified, digitized, sampled at a frequency rate of

400Hz, and online filtered using a pass band of 0.1 to 100Hz using the powerlab systems (Lorist et al., 2005). Then, the EEG signals were digitally low pass filtered with a cut-off frequency of 30 Hz, and online reduced to a sample frequency of 100 Hz to analyse the EEG frequency bands of interest (Boksem et al., 2006). Moreover, artefacts such as blinking and fast eye movements were removed from the recorded signals based on any amplitude greater than ±70 µV (Holm et al., 2009). Then, these processed signals were used for the EEG and ERP analysis.

For the EEG analysis, the average power in the theta band (4 – 8 Hz), and alpha band (8 – 12 Hz) were computed at the frontal and parietal electrodes F_z and P_z respectively. Next, the ratio of these two powers was determined, and named the 'cognitive ratio'. Moreover, the entropy (*i.e.* the amount of information flow or content) of the EEG signals (F_z, C_z and P_z) were computed to represent an additional mathematical measure to compare the cognitive performance of the participants for each experimental condition (*i.e.* control vs. exercise cognitive trials).

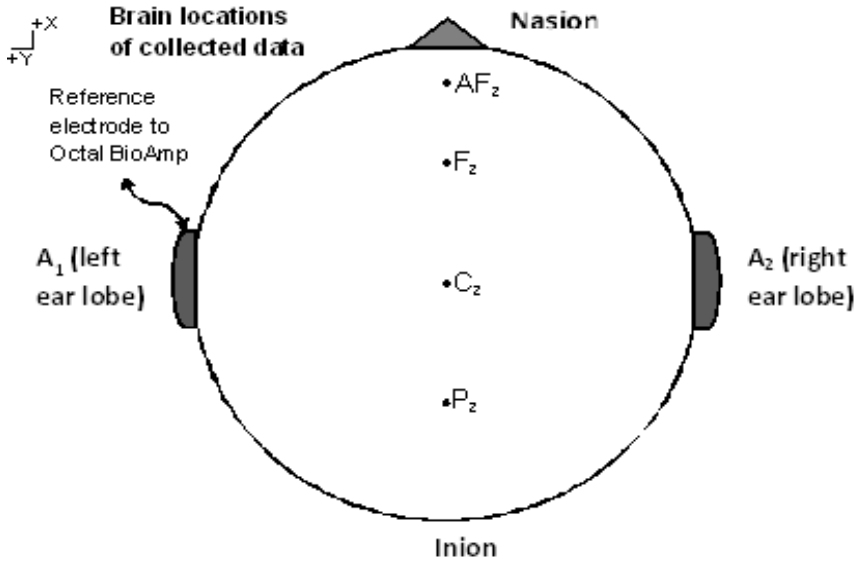

Figure 7.1: The 10-20 international system electrode placement showing the EEG electrode placement, the reference electrode (A_1 - left earlobe) and the ground

electrode (AF_z), F_z (Frontal midline electrode), C_z (central midline electrode) and P_z (parietal midline electrode).

7.2.5.2 ERP analysis

The Event Related Potential (ERP) data were at first processed following the same procedure used for the EEG analysis, then segmented into stimulus-locked (*i.e.* the visual stimulus) EEG epochs between -100 ms to +600 ms. The EEG epochs of the trials with omitted or miss responses were not included in the stimulus-locked ERP (Boksem et al., 2008) as they represent biased responses for the subsequent EEG analysis. And then, three types of ERP components (N100, P200 and P300) were analysed in this research study based on their associated cognitive function properties. As described in the literature review, the N100 ERP component ranges in-between 80 to 120 ms after a stimulus is triggered and is linked to a person's arousal and selective attention (Hillyard et al., 1973; Nash and Williams, 1982). Moreover, the P200 appears in-between 150 ms to 275 ms after the onset of an external stimulus and is related to the higher-order perceptual processing which is regulated by attention and visual cognition (Freunberger et al., 2007; Furutsuka, 1989; Luck and Hillyard, 1994). In addition, the P300 is an evoked potential associated to engagement of attention and it is linked to an individual's reaction time to an external stimulus as well as it can be used to measure how demanding a task is on the cognitive workload (Polich, 2003; Polich, 2007).

The averaged evoked related potentials were then used to measure the latencies and amplitudes of the ERP components N100, P200 and P300 respectively. Latencies were peak latencies, and were determined based on the visual

examination of the spatial distribution of the ERP components while amplitudes were mean amplitudes, and these were calculated as the average amplitudes in a time window of ±50ms (Walhovd and Fjell, 2002) around the peak latency.

7.2.6 Statistical Analysis

Firstly, all recorded and computed data were tested for normality using Kolmogorov-Smirnov (K-S) test (Fasano and Franceschini, 1987; Lopes et al., 2007). Then, a two-way factorial ANOVA (Analysis of Variance) was used (Quintana and Maxwell, 1994) to investigate the effects of time (6 intervals of 5 minutes) on various variables (*e.g.* reaction time, accuracy, cognitive ratio, heart rate, RR-interval and subjective measures of fatigue) for two experimental conditions (control and exercise) for same group of participants. When the main analysis indicated a significant interaction ($p < 0.05$) between the factors, follow-up analysis were achieved, adjusting error rates according to Bonferroni correction (Boksem et al., 2005). Furthermore, paired t-test was used to compare the means of any dependent variable subjected to these two experimental conditions. (Also see Appendix C, section C.1 for more details on F-ratio and degrees of freedom)

7.3 Results

The results are divided into several sections that are namely the subjective measures that consisted of the visual analogue scales (mental fatigue, physical fatigue and concentration) and the MFI-20 questionnaire that assessed the general fatigue, physical fatigue, mental fatigue, reduced motivation, reduced activity pre and post the cognitive battery test; the cognitive performance of the participants in terms of both reaction time and percentage accuracy of responses to visual cues followed by the ECG, EEG and ERP analysis results.

7.3.1 Subjective measures of fatigue

A two-way (2 experimental conditions x 6 time intervals) factorial ANOVA with repeated measures was conducted on the visual analogue scale data to determine whether there was a statistical significance in the means of dependent variables that were mental fatigue, physical fatigue and concentration between the two experimental conditions (control vs. exercise) but on the same group of individuals. The within-subject variable was the time-on-task repeated measures that were denoted as time5, time10, time15, time20, time25, and time30 (*i.e.* time5 means 5 minutes of the cognitive task had elapsed, time10 means 10 minutes of the cognitive task had elapsed, and so on till completion of the cognitive task). The model assumptions in terms of normality using Kolgomorov-Smirnov (K-S) test (Fasano and Franceschini, 1987; Lopes et al., 2007), and homogeneity of covariance using Box's test (Anderson, 1958; Seber, 1984) were evaluated and met in this statistical analysis. Furthermore, the MFI-20 subjective measures were analysed using a 2 (two experimental conditions) x 2 (pre and post cognitive tasks) factorial ANOVA with repeated measures, and the statistical results of these subjective measures of fatigue are as follows in the subsequent subsections.

7.3.1.1 Visual analogue scales (VAS) subjective measures

As shown in Figure 7.2, it was found that there was a statistically significant interaction in the percentage of mental fatigue between the condition type and time-on-task factor times ($F_{(6, 22)} = 492.19$, $p < 0.001$) as well as there was a significant main effect of time-on-task (time5 to time30) ($F_{(5, 22)} = 463.794$, $p < 0.001$). In addition, there was also a significant main effect in the condition type ($F_{(1, 22)} = 713.133$, $p < 0.001$) which represented a large effect size. For the physical fatigue subjective measure, there was a significant difference between the two

experimental conditions ($p < 0.001$), and within the subject test times ($p < 0.001$). However, there was no significant difference between the means of the concentration visual analogue scale for these two experimental conditions ($p = 0.057$) despite a significant difference ($p < 0.001$) in the time-on-task repeated measures.

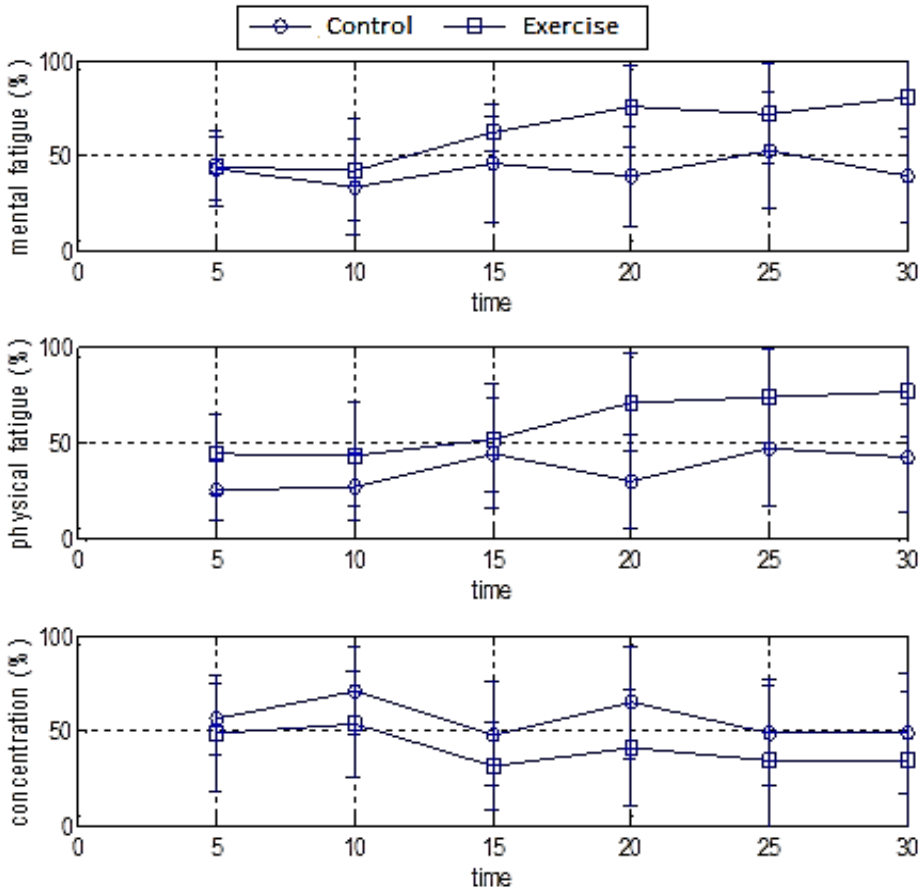

Figure 7.2: Energy-VAS subjective measures for the participants (n=12) under two conditions (control and exercise involved cognitive task).

7.3.1.2 Multi-Fatigue Inventory (MFI) subjective measures

A summary of results is shown in Table 7.1 depicting the mean value together with the respective standard deviation for each subjective measure, and for each experimental condition (control vs. exercise-involved) at the start and at the end of

the cognitive tasks. There was a significant difference between the mean of each dependent variable subjected for both experimental conditions and also between pre and post each experimental condition ($p < 0.05$).

Type of Fatigue / Type of Trial	General Fatigue	Physical fatigue	Reduced Activity	Reduced Motivation	Mental fatigue
Control (Pre)	5.5±1.8**	5.4±2.2**	5.3±1.9**	4.5±1.7**	4.3±1.2**
Control (Post)	10.6±2.6*	7.3±2.9**	9.0±2.9**	9.6±3.2*	13.3±1.7*
Exercise (Pre)	11.6±3.1**	11.8±2.6**	13.4±4.0**	9.6±2.7**	8.4±2.6**
Exercise (Post)	15.4±2.2*	15.5±2.3**	14.9±2.7**	14.0±4.2*	15.0±1.9*

Table 7.1: Summary of the MFI measures obtained from the participants ($n = 12$) under these two conditions (control and exercise involved cognitive tasks). The double asterisk (**) denotes a statistical significance at $p < 0.01$ between the means of the subjective measures for pre-control and pre-exercise whereas the single asterisk (*) denotes a statistical significance between the means for post-control and post-exercise experimental condition with statistical significance $p < 0.05$.

7.3.2 Cognitive performance (Reaction Time and Accuracy)

The cognitive performance, in terms of reaction time of the participants in choosing the correct responses by pressing the appropriate key as well as the mean percentage of accuracy for all the participants subjected to these two experimental conditions, was evaluated for each 5 minutes time-on-task interval. The dependent variables that were reaction time and accuracy were analysed using two-way (2 experimental conditions x 6 time intervals) factorial ANOVA with repeated measures.

7.3.2.1 Reaction time performance

The mean reaction time (Figure 7.3) of the participants in responding to the visual cues over the whole duration of the cognitive task for the control experiment was 475 ± 19.2 ms, and that for the exercise-involved experiment was 410.6 ± 16.6 ms (See Appendix E, Table E.2).

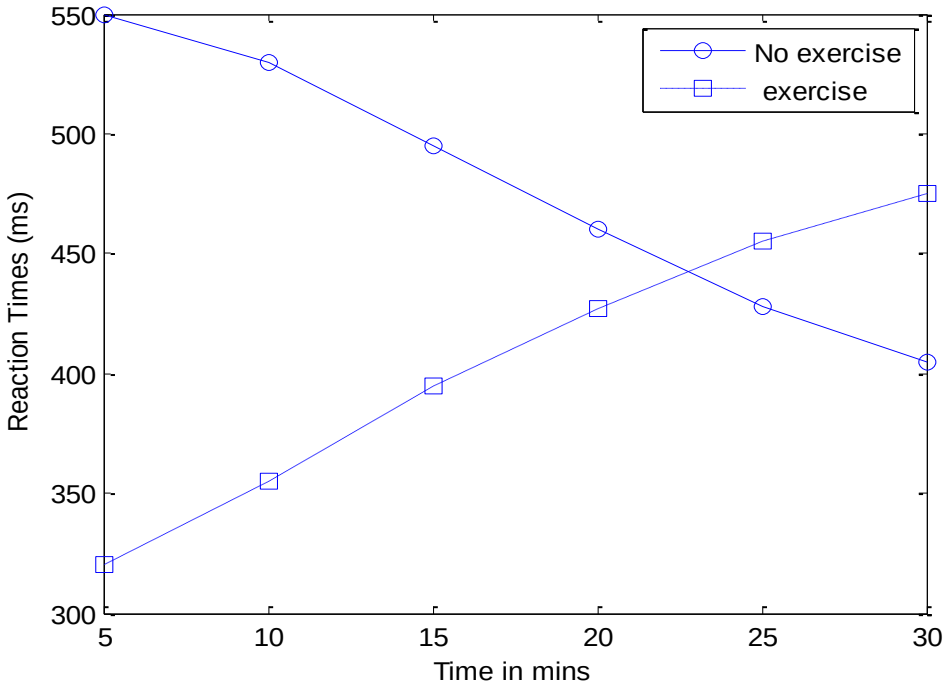

Figure 7.3: The mean (± S.D) reaction times of the participants (n = 12) for control and exercise-involved cognitive tasks were 475 (±19.2) ms and 410 (±16.6) ms respectively).

It was observed that the reaction times decreased linearly with increasing time-on-task for the control experiment with reaction times at time5 (5 minutes elapsed) of cognitive task was 550 ± 20 ms and at time30 (30 minutes elapsed) was 405 ± 20 ms ($p < 0.01$), and the contrary was found for the exercise-involved task whereby reaction times increased linearly till to the completion of the cognitive experiment

(time5 was 320 ± 10 ms vs. time30 was 470 ± 20 ms, $p < 0.01$). It was found that there was also a significant interactive effect between the repeated measures of time-on-task and experimental conditions ($F_{(6, 22)} = 1418.8$, $p < 0.001$).

7.3.2.2 Accuracy performance

As shown in Figure 7.4, the participants performed significantly better ($F_{(1, 22)} = 93.875$, $p < 0.01$) in the control trial with a mean percentage accuracy of (91.3 ± 1.2)% as compared to their accuracy of response performance for the exercise-involved cognitive trial which was (89.1 ± 1.4)%. However, there was no significant interactive effect between time-on-task and the two experimental conditions ($p = 0.236$, non-significant).

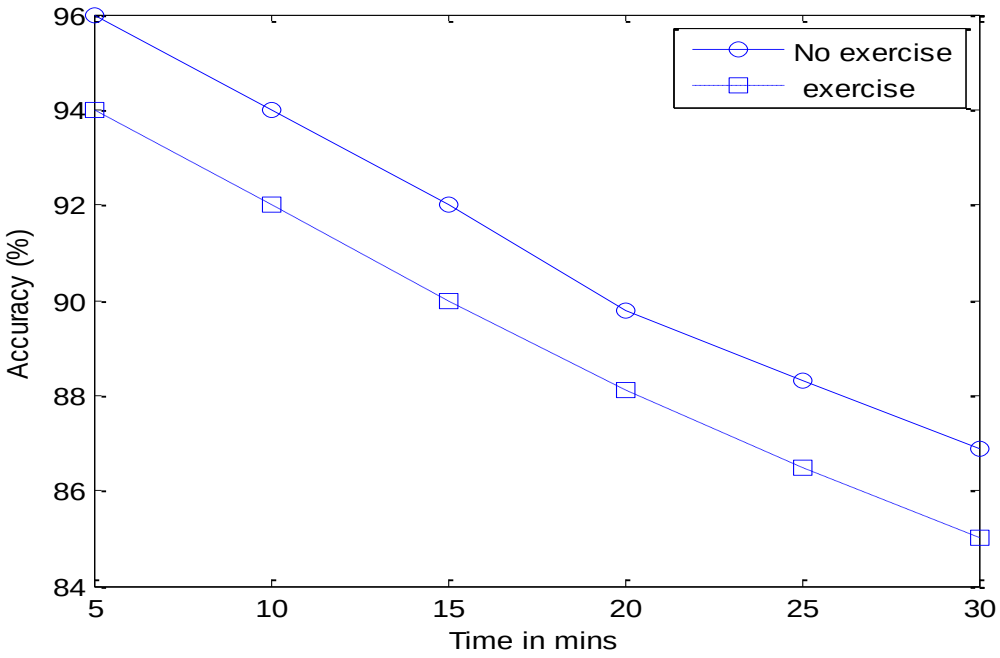

Figure 7.4: The mean (± S.D) percentage accuracy of the participants ($n=12$) for no-exercise (control) and exercise conditions were 91.3 (±1.2) % vs. 89 (±1.4) % respectively.

In both cognitive trials, it was found that the percentage accuracy of the responses decreased linearly based on the 6 data time intervals from time5 to time30 (See also Appendix E, Table E.1). Furthermore, there was no significant difference ($p > 0.05$) in the percentage of accuracy of the responses of the participants for the control and exercise experimental conditions in counting mentally the low probability target visual cues that were the white squares and white circles randomly shown up on the screen for the Modified Stroop task.

7.3.3 ECG Analysis (heart rate/bpm and RR – Interval/ seconds)

First of all, as shown in Figure 7.5, there was a significant difference at 95% confidence interval in the time-on-task factors (*i.e.* there was a significant difference in the mean of the heart rate activities for the various time intervals from time5 to time30, $F_{(5,\ 22)} = 4.455$, $p = 0.046$). There was also an interactive significant effect (time-on-task factors x experimental conditions, $p < 0.001$) and a significant difference between the mean heart rate activities (Figure 7.5) for each cognitive trial ($p = 0.039$). For the control trial, heart rate activities decreased from an average of 79 bpm in the first interval (time5) to 77 bpm in the last interval (time30) of the cognitive tasks, and for the exercise involved trial, heart rate activities fluctuated more often than that of the control experiment by increasing and decreasing through time.

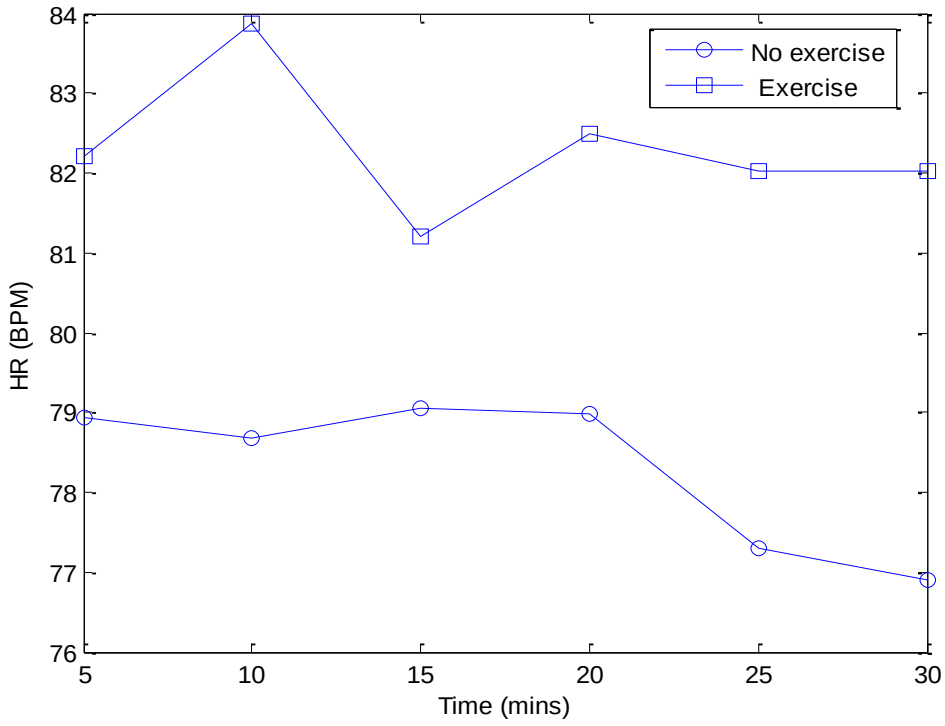

Figure 7.5: The mean heart rate activities (HR/BPM) for the twelve participants for each cognitive task session (control was 78.3 ± 1 bpm vs. exercise was 82.3 ± 2 bpm) at 5 minutes time-on-task intervals.

Secondly, as shown in Figure 7.6 there was a significant difference in the mean of the beat-to-beat interval for the two experimental conditions ($p < 0.001$) which was 0.776 seconds for the control experiment, and 0.740 seconds for the exercise-involved experiment. In addition, there was also a significant interactive effect for the within-subject factor (time-on-task) and the between-subject factor (conditions) with significant high F ratios of $F_{(6, 22)} = 27.720$, and statistical $p < 0.001$.

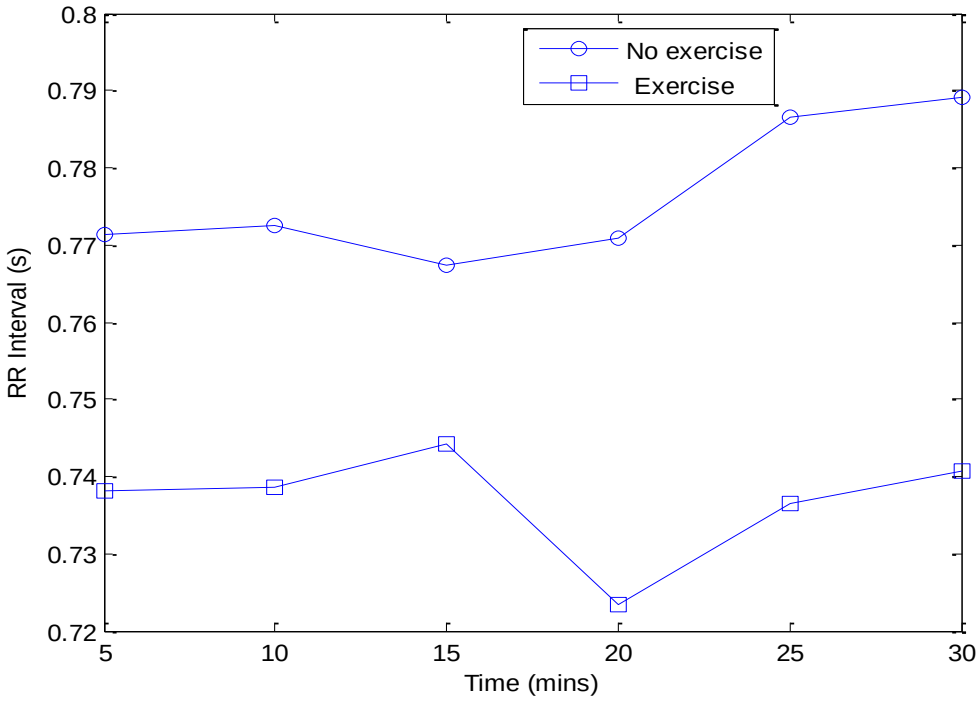

Figure 7.6: The mean beat-to-beat (RR interval/seconds) for the twelve participants for each cognitive task session (control vs. exercise) at 5 minutes time-on-task interval. The RR-interval for the control experiment was (0.776 ± 0.02) s vs. exercise involved experiment was (0.740 ± 0.01) s.

7.3.4 EEG Analysis and Entropy

In Figure 7.7, the notation Fzθ represents the theta band power at the frontal midline, the notation Pzα represents the alpha band power at the parietal midline of the brain and the cognitive ratio is the ratio of the frontal theta band power to the parietal alpha band power. For the whole duration of the cognitive trial, the mean (± standard deviation) of the theta band power, at the fronto-midline (F_z) for all the participants, was 6.76 (±1.74) μV^2 for the exercise-involved cognitive trial vs. 6.59 (±1.68) μV^2 for the control cognitive trial. Whilst the mean (± standard deviation) of the alpha band power of the participants at the parieto-midline (P_z) was 4.25 (±0.928) μV^2 for the exercise experiment as compared to 4.37 (±0.725) μV^2 for the

control experiment. From the fronto-parietal network data analysis, it was found that there was no significant difference between the cognitive ratios computed for both exercise and control cognitive trials ($F_{(1, 22)} = 3.140$; $p = 0.09$) even though the cognitive ratios were slightly higher for the exercise-involved cognitive trial than that of the control trial. However, there was a significant effect of the within subject factor time-on-task ($p < 0.01$), and there was also a significant difference in the cognitive ratio between the first 10 minutes (2.82 ± 0.31) of the cognitive trial as compared to that for the last 20 minutes (1.14 ± 0.16) of the cognitive trial for both experimental conditions (control vs. exercise-involved cognitive task trials, $p < 0.01$). (See also Appendix E, Table E.3).

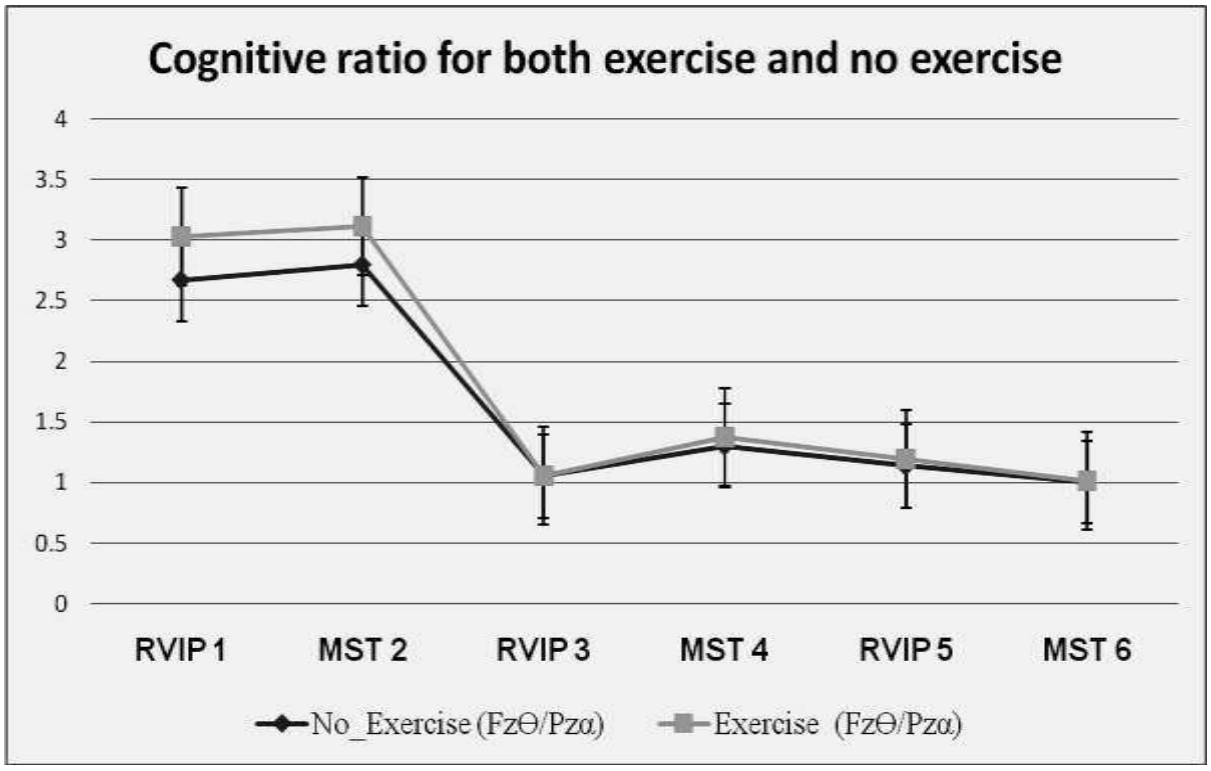

Figure 7.7: Cognitive ratio for both exercise-involved and no exercise (control) involved cognitive tasks ($n = 12$).

A further investigation was conducted by analysing the entropy (Section 2.7.7) of the EEG signals in order to find out the amount of information flowing in the frontal

and parietal regions in both conditions (Table 7.2). A 2-way factorial ANOVA with repeated measures (2 experimental conditions x 3 locations in the brain) was applied to the entropy data, and the statistical analysis revealed that there was a significant difference in the mean entropy between the frontal region (F_z) and the parietal (P_z) region ($p < 0.05$). In fact, the ratio of the entropy of the EEG signals at F_z to that of P_z for the control experiment was 0.867, and the ratio of the entropy of the EEG signals at F_z region to that of P_z for the exercise-involved cognitive task trial was 1.25.

Descriptive statistics for entropy				
Brain Locations	Conditions	Mean	Range	N
Cz	Control	2.0325	0.23542	12
	Exercise	2.0392	0.57619	12
	Total	2.0358	0.43046	24
Fz	Control	1.9958*	1.49913	12
	Exercise	2.3033*	0.92830	12
	Total	2.1496	1.22949	24
Pz	Control	2.3008*	1.31144	12
	Exercise	1.8408*	0.43025	12
	Total	2.0708	0.98300	24

Table 7.2: Summary of the entropy results of the EEG signals for the three brain regions (C_z, F_z and P_z) analysed in two experimental conditions (control vs. exercise) for 12 participants. The asterisk symbol * represents a statistical significance between the means of the entropy of EEG activities at P_z and F_z for both conditions with statistical $p < 0.05$.

7.3.5 ERP analysis results

In this section, the results from the ERP analysis are described and this ERP analysis was conducted to find out if there were any associations between the ERP components (N100, P200 and P300) and cognitive functions. Figure 7.8 illustrates the average ERP profile of a particular participant at the frontal midline region (F_z) for both exercise and control trials. This figure depicts clearly the ERP components after the onset of a stimulus. The coordinates **A** represents the N100 ERP component which occurred at 123.5 ms, and coordinates **B** represents the P300 ERP component which occurred at 304 ms.

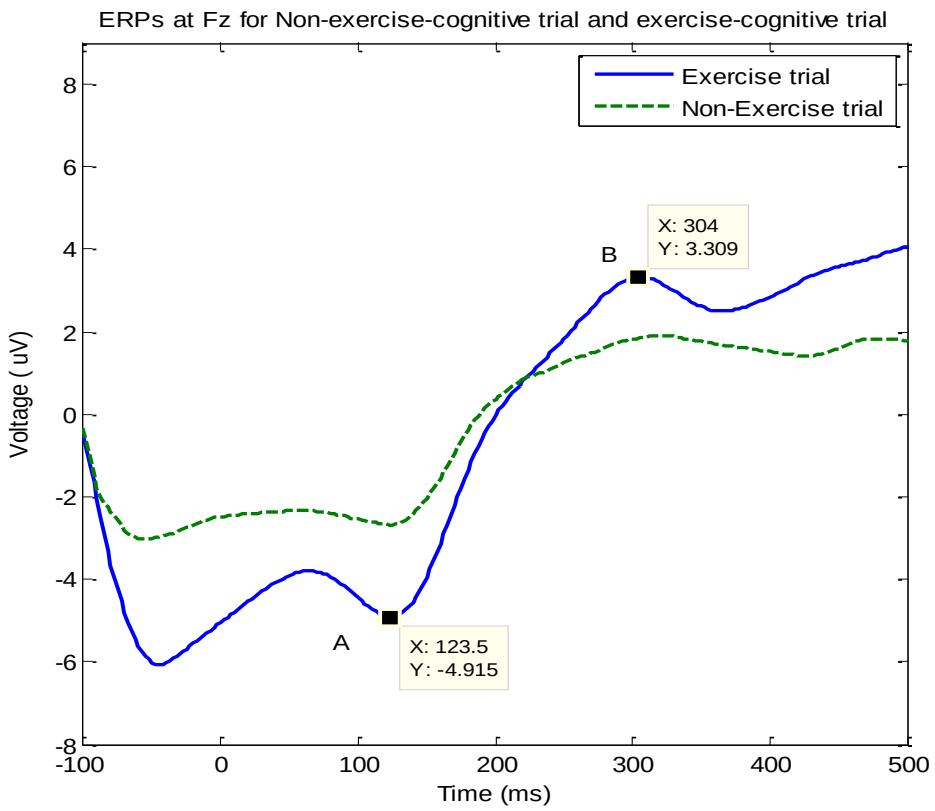

Figure 7.8: The average event related potential for one particular participant at the frontal midline region (F_z) for both cognitive trials.

The results of the ERP analysis are shown in Table 7.3 that summarizes the mean amplitude and latency of the various ERP components (N100, P200 and P300) for all the participants subjected to both experimental conditions that were the control and exercise-involved cognitive tasks.

Brain Locations	Conditions	ERP Components	Amplitude (μV) Mean (± Range)	Latency (ms) Mean (± Range)
F_z	Control	N100	-1.42 (±0.60)	108 (±25)
		P200	2.10 (±0.70)*	238 (±32)
		P300	4.25 (±2.80)	383 (±74)
	Exercise	N100	-1.87 (±0.80)	71.6 (±33)
		P200	3.84 (±1.40)*	228 (±55)
		P300	6.20 (±3.10)	369 (±63)
C_z	Control	N100	-0.135 (±2.0)	89.3 (±18)
		P200	4.22 (±1.30)	193 (±29)
		P300	3.04 (±1.5)*	406 (±76)
	Exercise	N100	-2.11 (±1.73)	89.7 (±23)
		P200	4.20 (±1.40)	232 (±37)
		P300	6.80 (±2.60)*	395 (±87)
P_z	Control	N100	-0.99 (±0.60)	118 (±31)
		P200	2.40 (±0.90)	226 (±37)
		P300	1.61(±1.10)**	432 (±65)**
	Exercise	N100	-1.42 (±0.82)	103 (±36)
		P200	2.90 (±1.10)	227 (±17)
		P300	6.13(±1.70)**	372 (±58)**

Table 7.3: Summary of statistical analysis of the mean amplitude (μV) and latency (ms) ERP components (N100, P200 and P300) for both experimental conditions for all participants ($n = 12$) (the single asterisk * represents $p < 0.05$ and the double asterisk symbol ** represents statistical significance $p < 0.01$).

Statistical analysis at the brain location F_z revealed that there was only a significant difference between the means of the amplitude of the P200 ERP component ($p < 0.05$) for the control and exercise cognitive trial. Moreover, there was a significant difference in the means of the P300 amplitude ($p < 0.05$) between these two experimental conditions at C_z; and also a significant difference ($p < 0.01$) between the means for both P300 amplitude at P_z (1.61 ± 1.10 μV for control vs. 6.13 ± 1.70 μV for exercise) and P300 latency at P_z (432 ± 65 ms for control vs. 372 ± 58 ms for exercise).

7.4 Discussions

7.4.1 Questionnaire analysis (Fatigue-related subjective measures)

The visual analogue scales subjective measures showed that the participants felt more fatigued mentally and physically in the exercise-involved cognitive trial than the control experiment. Furthermore, there was an increase in the physical fatigue and mental fatigue as well as a decrease in concentration with increasing cognitive time-on-task trials for both experimental conditions. In the same line of thought, certain researchers (Dureman and Bodén, 1972; Matthew 2004) showed that the subjective measures of fatigue were significantly greater than initial fatigue ratings as time-on-task increased.

From the MFI results, both mental and physical fatigue subjective measures were significantly greater in the exercise-involved cognitive experiment than that of the control experiment. Furthermore, the participants felt more reduced motivation and reduced activity for the exercise-involved cognitive experiment than the control experiment as well as they felt more reduced motivation and reduced activity at the end than at the start of the experimental conditions. In addition, McMorris and Graydon (1996) stated that motivation would not increase with increasing time-on-

task as the task demands were maintained same across trial blocks. In fact, for the exercise-involved cognitive trial, the author observed that most of the participants around time20 (20 minutes of the cognitive task had elapsed), started to feel very sleepy or tired (initiation of yawning processes) (Hermanowicz, 2007; LeWinter, 2007), and they stated as post-study comments that they needed a 'good nap or sleep'.

7.4.2 Cognitive performance (Reaction time and Accuracy of responses)

Interestingly, the participants subjected to the exercise-involved cognitive experiment had overall a lower mean reaction time (or faster speed) in pressing the numerical keys when presented with a particular visual stimulus or when the chunk of odd or even numbers was found. However, the reaction time of the participants in the exercise-involved cognitive task increased exponentially with increasing time on task. In contrast to that, the reaction time of the participants for the control experiment decreases exponentially, and hence they performed at faster speed during the last time interval (time30) of the cognitive trial. Several researchers indicated a relationship between cognitive task length and response time. Firstly, Levitt and Gutin (1971) found a non-monotonic effect on reaction time while Macchi et al. (2002) found a positive relationship between response time and cognitive task length; Furthermore, Boksem et al. (2005) found a mixed relationship based on post error responses which was negative and post correct responses which was positive after either a medium or long-haul physical task performance.

In this research study, the participants were more accurate in their responses during the control experiment than during the exercise-involved cognitive experiment. Couple with that, the accuracy of the responses to the visual cues

decreased significantly for both experimental conditions as time-on-task increased. Lorist et al. (2002) emphasized a negative relationship between task duration and performance as response accuracy decreased significantly across the participants with increasing time-on-task. Another subjective report of fatigue by Lorist et al. (2000) showed that as fatigue increased, response accuracy decreased independent of time-on-task. Certain researchers, Williams et al. (1999) and Williams (2000), demonstrated that when the subjective reports of fatigue increased, the number of visual fixations increased as more fixations were needed to extract sufficient information from the visual scene which might contribute to a greater sensation of fatigue. Following a physical and a series of mental fatiguing task, the participants' arousal decreased based on the subjective measures of fatigue where they seemed to disengage from the task even though that the cognitive task demand was same across the cognitive block trials. Overall, the participants subjected to the control experiment performed better in terms of higher percentage accuracy of their responses but with slower reaction times than in the exercise-involved cognitive experiment.

7.4.3 ECG analysis (Heart rate and RR-Interval)

Heart rate activities were higher for the exercise session and the beat-to-beat interval was higher for the control session. For the exercise session, there were more fluctuations in the mean heart rate activities with increasing time-on-tasks whereas for the control session, the mean heart rate activities of the participants started to decrease from time20 (20 minutes elapsed). The frequency of fluctuations in the heart rate activities (for the exercise session) showed the frequent fluctuating physical efforts while combatting fatigue (Dureman and Bodén, 1972; Matthew, 2004). The decreasing heart rate activities of the participants in

the control showed that the arousal levels of the participants started to decrease by taking into account the decreasing percentage accuracy of their responses (Steriade, 1996; Yerkes and Dodson, 1908). The decrease in heart rate activities and corresponding increase in beat-to-beat intervals for the exercise-involved cognitive trial were associated to an increase in fatigue due to an increase in the parasympathetic control or activity of the nervous system (Hancock and Meshkati, 1988; Jouanin et al., 2004). The next section discusses the results from the EEG and ERP analysis.

7.4.4 EEG analysis (cognitive ratio and entropy)

There was no difference in the means of alpha band power at P_z and theta band power at F_z subjected to both experimental conditions. Moreover, there was no difference in the cognitive ratio (frontal theta band power to parietal alpha band power) between the two experimental conditions. However, there was a difference in the cognitive ratio with increasing time-on-task including an abrupt reduction in cognitive ratio after 10 minutes of the cognitive trial had elapsed for both control and exercise cognitive experiments. The decrements in cognitive function (cognitive ratio and poor accuracy) that were observed during the sustained mental work can be regarded as cognitive fatigue which subsequently prevented the alert participants to continue high mental performance (Hockey et al., 1997; Montgomery et al., 1995). In addition, Makeig and Inlow (1993) found a progressive increase in the EEG power in the frequency range of 4 Hz to 14 Hz as alertness decreased and error rates increased in a vigilance mental task. Cheng et al. (2007) found that there was a significant difference between the theta band and alpha band frequency power before a mental fatigued 3-hour visual display task session as compared to post session. When people feel fatigued during or after

prolonged periods of cognitive activity, they have the tendency to lose concentration and cannot focus their attention on the tasks they are performing (Boksem et al., 2006). Furthermore, some researchers found that when arousal level dropped, EEG activities changed from fast and low amplitude waves to slow and high amplitude waves, and this decrease in arousal brought about a corresponding increase in low-frequency alpha and theta activities (Klimesch, 1999; Lafrance and Dumont, 2000; Oken and Salinsky, 1992), which might be reflecting a decrease in cortical activation (Cook et al., 1998; Laufs et al., 2003). Hence, the amount of alpha and theta power can provide an indication of the level of fatigue which one experiences during mental fatiguing tasks (Boksem, 2005).

As the cognitive ratio is based on a particular frequency range principle, the advantage of using the entropy mathematical method was that it considers the total information content of the EEG signal across the brain regions under investigation (Viertio-Oja et al., 2004). It was found that the mean entropy was significantly different between the fronto-midline and the parieto-midline regions of the brain, and the ratio of the entropies of these two brain areas was higher during the exercise-involved experiment than in the control experiment. Therefore, it appeared that there was an "overload" of information that prevented the participants in performing well, and remarkably, the cognitive ratio (as described in the previous section) was also slightly higher during exercise than that of the control session. During competitive sporting environments, Mathews and Desmond (2002) stated that high task difficulty, prolonged task exposure and multiple task demands could induce a great level of information processing which subsequently increased the mental workload. Such increments in mental workloads caused a depletion of the cognitive system’s resources that were

available for task completion and consequently promoted the development of fatigue (Matthews et al., 2000; Mathews and Desmond, 2002). According to activation theory stated by Lindsley (1951) and elaborated by Hebb (1955), the continuum ranging from low activation (*e.g.* sleep) to high activation (e.g. excited states) is a function of cortical bombardment by the ascending reticular activating system (Magoun, 1952; Steriade, 1996), and the relationship between activation and level of performance is represented by the inverted U curve (Yerkes and Dodson, 1908). This means that, with an increasing activation level, the level of performance increases monotically but after exceeding an optimal point, the relation becomes nonmonotonic which implies that further increase in activation level beyond this optimal point decreases the level of performance; this reduction in performance is related to the amount of increase in the level of activation. Therefore, the overflow of information as shown by the entropy ratio prevented the participants to focus properly their attention which might contribute to the reason why their response accuracy decreased specifically during the exercise-involved cognitive trial.

7.4.5 ERP analysis (N100, P200, P300 ERP components)

The N100 ERP component mean amplitude was relatively greater, and its corresponding latency was smaller during the exercise session than in the control session for all three brain locations. This insinuates the participants reached a faster and higher arousal state in the exercise-involved cognitive trial than in the control experiment (Nash and Williams, 1982). The higher amplitude of the ERP component P200 at the frontal lobe showed that the participants achieved a greater intensity of higher-order perceptual processing and visual cognition. However, the higher arousal state, and higher-order perceptual processing and

visual cognition were impaired by cognitive fatigue as shown by the decreasing cognitive ratio. One possible reason was that there might be too much information flow (as shown by the entropy values) that caused a mental stress on the participants post exercise which affected the accuracy of their responses that was lower than the control session (Freunberger et al., 2007; Luck and Hillyard, 1994).

This greater amount of information flow was caused by a greater of amount of activation at cortical level to such a point that the participants surpassed the optimal level of arousal for optimal performance. Even though the participants subjected to the exercise-involved experiment had faster reaction time (reflected by their lower P300 latencies), their response accuracies were poorer hence resulting poorer cognitive performance. On the other hand, the control session results showed that the participants improved their reaction times in the last intervals of the cognitive tasks but they failed to improve their accuracy in responding to the visual cues with increasing time-on-task because they felt more fatigued in the last intervals of the cognitive tasks than at the start according to their subjective measures of fatigue. Moreover, the P300 amplitude increased acutely following an aerobic exercise (Nakamura et al., 1999) whereas other researchers found that P300 amplitude significantly increased after a 3-hour VDT experimental task where the subjects appeared mentally fatigued (Doppelmayr et al., 2007). Therefore, both the latency and amplitude of the P300 ERP component seemed to be influenced following both physical and cognitive activities and hence representing promising tool for measuring cognitive performance.

7.5 Summary

The participants felt physically and mentally more fatigued during the exercise-involved cognitive trial than during the control experiment. Moreover, there was the

apparent decreasing engagement from the cognitive task based on the decreasing accuracy performance of responses together with decreasing cognitive ratios with increasing time-on-task cognitive trial for both experimental conditions. This cognitive fatigue and the changes in heart rate activities as well as beat-to-beat intervals across the cognitive trials showed that the development of fatigue did not manifest only centrally but also peripherally. In addition, sustaining attention during the cognitive task is closely related to arousal according to the differences in the event-related potentials which subsequently influence the cognitive performance of the participants. The circular relationship between fatigue, performance and arousal state implies that one should work 'harder' to sustain performance levels which would require consequently more energy resources, thereby intensifying the development and consequences of fatigue. Therefore, the increase in perceived mental workloads and decrease cognitive performance following an exercise-involved cognitive experiment as well as between pre and post both experimental conditions demonstrated that there appeared limited amount of energy resources in the central systems that are reflected in their cognitive behaviours and cognitive performance.

CHAPTER EIGHT

General Discussion

8.1 General discussion of the physiological control models of exercise fatigue

The aim of this thesis was to use mathematical modelling and analysis techniques to explore the nature and cause of exercise fatigue to optimise performance during physical activity. A set of objectives, in the form of experimental case studies were devised to investigate, firstly, adenosine triphosphate production from the energy system pathways during physical activity (*i.e.* during maximal exercise of short duration or endurance exercise using different types of pacing) (Hill et al., 1923; Shulman and Rothman, 2001; Weir et al., 2006); And secondly to investigate the presence, complexity and the characteristics of the system control mechanisms which regulate physical behaviour and activity, and sustain homeostasis in the physiological systems (Davis and Bailey, 1997; Lambert et al., 2004; St Clair Gibson and Noakes., 2004; Ulmer, 1996). Therefore, by understanding the physiological principles that are responsible for the control of exercise-induced fatigue, through well-known mathematical techniques to biology and medicine together with the existing biological theories, it was hoped to be able to evaluate and develop these physiological theories in order to improve sports performance. Various theoretical control models of fatigue have been proposed to explain the cause of exercise fatigue, and these physiological models have somehow similar or different rules underlying their developments.

For instance, some biological control models (Brooks et al., 2005; Hill et al., 1923; Hill, 1924) state that physical activity is limited only by changes in the substrates or metabolites found in the working skeletal muscles. These changes can represent either too much accumulation of metabolites (such as blood lactate) or depletion of important substrates (such as adenosine triphosphate) that can impede the proper functioning of the exercising muscles which impair the capacity of these muscles to produce force or power. Another control model of exercise fatigue (Davis and Bailey, 1997; Fowles et al., 2002; Nielsen and Clausen, 2000) states, however, that only chemical changes within the brain modify the cerebral function that reduces the capacity to maintain central motor drive to the working muscles (central fatigue).

Furthermore, some non-catastrophic control models (Lambert et al., 2004; St Clair Gibson and Noakes., 2004) posit that exercise performance is regulated by the central nervous system to maintain homeostasis in all physiological systems so as to prevent any physiological systems failure as opposed to the catastrophic central fatigue or peripheral fatigue conceptual models (Davis and Bailey, 1997; Gonzalez-Alanso et al., 1999; Hill et al., 1923; Roberts et al., 1997). In fact, all the biological control models revolve around the idea that either exogenous or endogenous factors affect the physical performance of the athletes, and hence are related to the fatigue which occurred during physical activity. Following these popular theories in explaining the cause of fatigue, this thesis attempted, together with the existing physiological control models, to unearth the essence and cause of exercise-induced fatigue through the use of various mathematical modelling and analysis techniques in various physical conditions at rest and during exercise. The following sections critically evaluate the mathematical results in light of the

physiological models of the exercise fatigue, present the corresponding implications for the cause of exercise fatigue, and describe the limitations of the experimental case studies.

8.2 General discussion of the physiological models based on the results of the experimental case study one

In this research, there was a growing need to understand how the human body system modulates the amount and the rate of ATP utilisation during high-intensity exercise of short duration to delay the onset of fatigue and hence, improve sprint performance. According to the results from experimental case study one, the energy produced from the oxygen-independent glycolysis anaerobic subsystem seemed to undergo a more physical demanding metabolic process (*i.e*. the time constant of the oxygen-independent glycolysis metabolic energy process was highest) as compared to the energy produced from the other anaerobic subsystems (ATP-endogenous and PCr utilisation). In addition, the PCr anaerobic subsystem contributed to most energy needed to complete that physical activity and the depletion of this anaerobic subsystem affects sprint performance. The energy production from the various energy systems seemed to be controlled following the timely metabolic process (the maximal metabolic process of the anaerobic subsystems occurred at specific times) as well as the metabolic setting rate of ATP production from each energy system. Furthermore, it was observed that the underlying mechanisms responsible for this timely metabolic process and metabolic rate, for any particular anaerobic subsystem, appeared similar as the value of the corresponding time constant was practically same for all sprinters.

The results obtained from study one support one particular hypothesis predicted by the cardiovascular/anaerobic/catastrophic model which states that the depletion of substrates (such as ATP) may affect the working muscles (Edwards, 1983; Hill et al., 1923). However, these mathematical results did not implicate that the exercise fatigue which affected their sprinting performance was only peripheral because there appeared to be some common controlling mechanisms among all sprinters that affected the rate of ATP production and depletion for the various anaerobic energy subsystems. These common controlling mechanisms can originate from the peripheral or central systems, or both.

These mathematical results are also in agreement with the energy supply/energy depletion model (Shulman and Rothman, 2001) whereby fatigue during exercise is associated to the failure of the metabolic pathways to produce sufficient amount of energy (ATP) to the active muscles. This particular physiological theory and the mathematical modelling result show that if the sprinters' body systems can produce more energy from the PCr utilisation metabolic process, or store a larger amount of ATP endogenously, or initiate a faster metabolic process of the oxygen-independent glycolysis, their sprinting performance could have been better.

Furthermore, the energy supply/energy depletion model predicts the importance of conserving energy production through energy pathways (*i.e.* the anaerobic subsystems and the aerobic system) which is thought to affect performance. This insinuates that by depleting any particular energy system pathway, this may cause a stress on the human organism where in this research, it was demonstrated that the highest rate of expenditure of ATP via the PCr system caused a simultaneous decrease in maximum speed whereby the sprinters failed to sustain the maximal speed performance. From these results, certain physiological control model

theories (Edwards, 1983; Hill et al., 1923; Shulman and Rothman, 2001) support the mathematical findings but they do not provide answers to certain mathematical observations such as the common controlling mechanisms that were observed peripherally for all sprinters which affected their sprint performance.

8.3 General discussion of the physiological models based on the experimental results of case study two

To continue evaluating the principles underlying the physiological control models, there was also a great need to investigate how pacing affects sport performance during a time-trial exercise in terms of energy expenditure and physiological homeostatic disturbance (Ansley et al., 2004; de Koning et al., 2011; Noakes, 2000; Shulman and Rothman, 2001; Tucker et al., 2006; Ulmer, 1996). One common and important observation in athletic competition (Ansley et al., 2004; Tucker et al., 2006; Ulmer, 1996), especially in time-trial exercise, is that athletes have a tendency to vary their pacing during the race. In so doing, they either let their competitors surpass them (as part of a foreseen plan) or accelerate in the middle of the time-trial race or at the near end of the race (the latter is normally observed as an endspurt). These observations show that muscular power output seems to be controlled in an anticipatory manner to avoid unexpectedly large (and uncontrolled) homeostatic disturbances in the physiological systems. Therefore three common types of pacing (de Koning et al., 2011) were considered (self pace, even pace and variable pace) for this experimental case study for comparison purposes of the metabolic resources from the energy systems, and the effect of pacing on physiological systems. The mathematical modelling and analysis results showed that pacing affected considerably the amount of energy produced from the aerobic and the anaerobic energy systems as it was found that a particular type of pacing can be either anaerobic energy system dependent or aerobic energy

system dependent. Therefore, by adopting a pace, it is just to optimise these energy systems accordingly. Interestingly, for any particular cyclist, the total work done for completing each pacing trial was not significantly different. This indicates that pacing does not necessarily economise the total metabolic energy resources found in the human body system but rather creates a desirable internal environment for the biological metabolic processes to take place so that the physical activity can be completed successfully. Moreover, the hazard score index predicted the amount of homeostatic disturbance that pacing may inflict on the physiological systems by taking into consideration the momentary ratings of perceived exertion (a subjective measure of fatigue) and the percentage distance that remains to be covered.

As expected, both self pace and even pace time trials caused less homeostatic disturbance (with low hazard score index) to the physiological systems throughout the time trials as compared to the variable pace trial. The mathematical results as well as other research studies (Hettinga et al., 2006) showed that there was a constant reduction in power output during the endurance time trial exercise, and this acted as a defensive mechanism to prevent the cyclist (human organism) from maintaining same high pace or power output to avoid any catastrophic physiological system failure or any irreversible damage. This observation also showed that muscular power output appeared to be controlled in an anticipatory manner to avoid large (and uncontrolled) homeostatic disturbances. Furthermore, blood lactate concentration correlated with RPE values whereby it was found that variable pace was the most tiring type of pacing with highest blood lactate concentration and rating of perceived exertion. Therefore, these mathematical findings are in partial agreement with certain principles of the

cardiovascular/anaerobic/catastrophic physiological model which posits that when the rate of production of blood lactate concentration is higher than its rate of removal, this may cause exercise-induced fatigue. In addition, the mathematical results (case study two) demonstrated that the energy supply/energy depletion model predicted well by stating that the insufficient amount of energy from a particular metabolic pathway may inhibit sport performance as variable pace was the most anaerobic energy system dependent where mean RPE and blood lactate concentration over the whole time-trial were highest. Moreover, the experimental results also support another important prediction of this energy supply/energy depletion model which postulates that energy production from the energy pathways may affect physical activity performance and a proper strategy is needed to use the available metabolic energy resources efficiently.

Moreover, the mathematical results also favour one particular hypothesis of the neuromuscular fatigue model which states that there is a reduction in force or power output despite the fact that the perception of effort increases (which is believed to be caused by a reduction of muscle activation by the central nervous system) as was observed with increasing RPE specifically during the mid-portion of the self pace time-trial. The biomechanical model predicts that the muscles can be regarded as elastic energy systems where the more elastic the muscle is, the less torque this muscle is required to produce and this increases efficiency (Pennisi 1997, Roberts et al., 1997; Noakes, 2000). During the variable pace, the cyclists utilised fast twitch muscle fibres more often (based on the very high intensity of 140% of their mean pace power output) (Pascoe and Gladden, 1996) that consequently made the blood lactate concentration to increase (McMahon 1984; Wasserman et al., 1986) and they felt most fatigued by adopting this

variable pace. The mathematical modelling results together with the ratings of perceived exertion and the blood lactate concentration did not support the notion that the more elastic muscle one utilised, this would decrease the rate of substrates accumulation as posited by the biomechanical model (Pennisi, 1997). In addition, implicit to the task dependency model, the physiological mechanisms of exercise-induced fatigue in fact varied with the different pacing time-trials where the cyclists felt most fatigued while performing a variable pace and least fatigued for the even pace trial even though the cycling distance to be covered was kept constant. The presence of the endspurt that occurred at the end of the cycling race demonstrated that there was the inherent conscious effort which influenced exercise performance as predicted by the psychological/motivational model and also performance was sub-optimal specifically throughout the self pace time-trial. Therefore, it was shown based on the mathematical modelling and analysis results as well as the conceptual model theories that pacing affected the amount of energy produced from the aerobic and anaerobic energy systems and this caused different degrees of homeostatic disturbance which subsequently brought about different levels of the sensation of exercise-induced fatigue. During self pace trial, there was the conservatory nature of the cyclists of not pushing themselves to the physical limit that influence their exercise behaviour without disregarding the fact that they were consciously changing their physical effort, in an anticipatory manner, as shown by the endspurts when reaching the end of the race and frequent changes in power output throughout the cycling time-trial race.

8.4 General discussion of the physiological models based on the experimental results of study three

Following a thorough mathematical analysis of the effect of pacing trial on energy expenditure, results from the third experimental case study confirmed the presence of control mechanisms in the physiological system activities. By taking into account the hypotheses of certain physiological control models (Lambert et al., 2004; St Clair Gibson and Noakes., 2004), mathematical results also showed that the complexity and characteristics of the physiological system control mechanisms were different so as to maintain the proper functioning of these biological organ systems during physical activity. The inverse relationship of power to increasing frequency as well as the multiple frequency peaks demonstrated the presence of control mechanisms in the physiological systems. However, for any particular organ system, the complexity and characteristics (predictability and resilience to change) of the system control mechanisms responsible for regulating the various physiological systems were different.

Furthermore, the fractal dimension predicted the complexity of the pacing trial, and the complexity of the physical activity influenced the development of fatigue which consequently affected the physical performance of the cyclists as posited by the task dependency model (Weir et al., 2006). For a control to occur there should be the presence of a frequency or a rate inherent in the communication or interaction among the physiological systems. The existence of such type of control system that was showed to be existent in this research supports the prediction of Ulmer (1996) in his teleoanticipation model which stated a control is important to optimise performance during a physical activity. The difference in complexity of the system control mechanisms demonstrated that the human body works as a complex

system (as hypothesized by the Central Integrative Regulator model) and this complexity may be due to the complex interaction of the biological processes that occurred between the central systems and the peripheral systems (Lambert et al., 2004; St Clair Gibson and Noakes., 2004). The behavioural characteristics of the physiological systems in terms of resilience to change subjected to a physical activity (a particular type of pacing) and predictability (in terms of determinism and recurrence rate) form an important part of the possible features of the control mechanisms that influence the onset of fatigue that are supported by the predictions of the task dependency model. Some physiological systems are more stringently protected by this control system as per the high trapping time of the biological activities of the cardiovascular system as compared to that of respiratory system. This is one of the expectations of a more robust system (with higher time constant) as suggested by (Lambert et al., 2004; Pincus, 1994). The predictability of the behaviour of the physiological activities of the biological organ systems as supported by the task dependency model may also contribute to the onset of exercise-induce fatigue and affect sport performance accordingly.

Therefore, certain principles of the existing biological control models of exercise fatigue and the mathematical results from this research support the idea there is the presence of a control system which is important to regulate the internal environment, the interactive behaviour of the physiological systems and the whole human organism subjected to a physical activity.

8.5 General discussion of the physiological models based on the experimental results of study four

Some physiological models (Lambert et al., 2004; St Clair Gibson and Noakes., 2004) postulated that there may be a potential central regulator that paces the human organism during physical activity by interacting with the various

physiological organ systems. Based on the mathematical analysis of the physiological system activities, it was observed that the system control mechanisms used specific frequency bands simultaneously to interact with the physiological systems. For there to be the simultaneous allocation of frequencies to modulate the physiological activities of the organ systems (See Sections 6.3.2 and section 6.3.3), there should be a higher level of control to these organ systems. As such, this control may be present in the central systems in regulating the peripheral systems (Anthea et al., 1993). In addition, an increase or decrease in the frequency power of the communication band regulating any particular physiological system affects the physiological and overall behaviour as well as performance of the cyclists during physical exercise.

These mathematical results are in line with the prediction of the teleoanticipation model which suggested that, through a preventive process; the efferent signals from that “controller” can determine various peripheral processes and then the afferent signals from these peripheral systems can feedback information to change pace or power output (Ulmer, 1996). Moreover, the integrative central regulator model (Lambert et al., 2004; St Clair Gibson and Noakes., 2004) postulates that this controller paces the body during exercise through interactive communication between the central and the peripheral systems. In fact for a pacing to occur, the various physiological systems should be simultaneously controlled to produce an effective and non-catastrophic physical activity and behaviour. If there was no simultaneous control, the physiological systems would have been unmanageable which would subsequently lead to an undesirable catastrophic physiological system failure during the race or physical activity. Following certain predictions of physiological exercise models of fatigue and the mathematical analysis from this

research, there is in fact a control system which regulates the peripheral systems through an interactive biological communication process. Moreover, this control system is complex and has higher level of control to the peripheral systems as it has the ability to communicate simultaneously to these physiological systems using specific frequency bands frequencies and the power of which affects the physiological behaviour of the organ systems that subsequently influences performance during physical exercise.

8.6 General discussion of the physiological models based on the experimental results of study five

The last experimental case study investigated whether there were finite metabolic resources in the brain by performing exhausting physical and cognitive tasks. The decreasing cognitive performance in terms of poor accuracy of responses, and faster reaction times following a tiring exercise-involved cognitive trial showed that there was definitely a change in the cognitive behaviour in the central system as compared to the control cognitive trial. According to the subjective measures of fatigue (*i.e.* reduced activity, motivation and concentration as well as increased mental fatigue and general fatigue), it was certain that the cognitive load which was perceived by the participants post a tiring physical activity seemed to increase even though the cognitive task demand placed mentally on them was kept constant throughout the cognitive trials. This insinuates that for a perceived 'cognitive load' to increase, there should be a decrease in the level of arousal or metabolic energy in the brain. In this research, the high cognitive ratio and information processing (*i.e.* entropy) across the fronto-parietal network during the exercise-involved cognitive trial impaired cognitive performance by decreasing the arousal state according to the inverted-U relationship of performance with increasing arousal level (Yerkes and Dodson, 1908). In addition, the significantly

higher heart rate activities, physical fatigue and general fatigue post the exhausting physical exercise also implicate that the participants felt that they were applying greater physical effort while performing the cognitive tasks than during the control cognitive experiment.

According to the thermoregulatory model of fatigue (Nybo and Nielsen, 2001; in et al., 1999), during a physical activity, an increase in body temperature increases heart rate activities which were observed post the exercise-involved cognitive trial and this may represent an additional physical stress on the participants while performing the cognitive tasks. Also, the reduction in arousal post an exhausting physical exercise trial, could have caused the fatigue in the peripheral systems (via a decrease in neuromuscular propagation) as shown by the subjective measures (increase physical fatigue, general fatigue and reduce activity) and the fluctuations in heart rate activities as hypothesized by the neuromuscular fatigue model (Davis and Bailey, 1997).

Interestingly, the central fatigue model supports the idea that the brain regulates behaviour by regulating the arousal or the central activation. This is clear that the participants were more fatigued mentally and physically following the tiring exercise bout and this affects their cognitive behaviour and performance. Most participants felt very sleepy during the near end of the exercise involved cognitive experimental condition (also predicted by psychological/motivational model) which means that the performance of the participants in this cognitive experiment was also regulated at a subconscious level to prevent a conscious override of the bodily functions as shown by their poor cognitive performance (Davis and Bailey, 1997).

Therefore, the mathematical analysis of the physiological and brain activities, the subjective measures of fatigue, and the predictions of the physiological control models of exercise-induced fatigue, it may be deduced that not only the brain appeared to have finite metabolic resources but also the whole human body seemed to possess finite level of energy resources. Therefore, for sport performance to be a peak level, both the central systems and the peripheral systems should be equipped with necessary metabolic energy resources to reach optimum arousal and complete successfully a physical activity.

8.7 Limitations of this research

This thesis was able to analyse certain key principles of the existing physiological control models. However, more research should be devised to assess the veracity of the current mathematical findings under different sporting conditions as well as the hypothesis of other biological control models (such as the thermoregulatory and biomechanical models) in the aims to produce a solid and clear picture of the aetiology of exercise-induced fatigue. Furthermore, the mathematical methods that were used in this thesis were chosen mainly because they are currently applied to the field of biology and medicine and they are robust to noise (Higuchi, 1988; Rioul and Vetterli, 1991; Zbilut et al., 1995). Therefore, this could be improved by developing mathematical and statistical tools based on the temporal characteristics and frequency behaviour of the biological activities. The experimental case studies comprised both lab-based (experimental case studies 2, 3, 4 and 5) and one field data (experimental case study 1), this thesis assumed that the data collected in the laboratory conditions really give a succinct picture of the behaviour of the athletes in a real sporting environment.

8.7.1 Type and size of the sample population used in this research

The study population consisted mainly of young and healthy group of people with mean age 31.2 (±7.3) years and mean body mass index 23.9 (±2.1) as well as their physical activity varied from club level to elite level athletes. Based on these criteria, this thesis does not generalise the findings or the concepts of the control of fatigue to an older, or younger, or to a less physically active (sedentary) group of people. The population sample size used in the various experimental case studies varied between 8 to 12 participants and this was why strong inferential statistics were used to support the results data (Andrew et al., 2011; Gratton and Jones, 2004). In addition, by recording the physiological activities at high capture rates (between 10Hz to 400Hz), the physiological samples, under analysis, range from hundreds to thousands, and these were sufficient for this research to produce reliable results (Cronbach et al., 1972; VanVoorhis and Betsy, 2007; Marcoulides, 1993) but at the expense of great amount of time in extracting the physiological data, processing the data for analysis and in computation.

8.7.2 Accuracy of report

Even though the mathematical results obtained in this research are reliable and promising, the accuracy of this report could be improved by the following ways. Firstly, the accuracy of the experimental results could be improved by increasing the quantity of physiological data by using a higher capturing rate (Marcoulides, 1993), or secondly, by increasing the population sample covering a wider or narrower age group, or by repeating the experimental case studies to increase reliability. Last but not least, the quality of data can be improved by utilising appropriate pre-processing techniques such as data transformation to help in

conducting statistical or mathematical analysis easily on the biological data (van den Berg et al., 2006).

CHAPTER NINE

Conclusion

9.1 Conclusions based on mathematical findings and existing biological theories

A current challenge in exercise science is the cause of fatigue that affects exercise performance under different sporting conditions. Despite various physiological control models have been developed, none of them has been able to explain with certainty this cause of exercise-induced fatigue (Noakes, 2000; Weir et al., 2006). For example, certain foundations of the physiological models (Davis and Bailey, 1997; Lambert et al., 2004; St Clair Gibson and Noakes., 2004) are the on-going developments of previously hypothesized control models (Baker et al., 1993; Newham et al., 1991; Ulmer, 1996) or clash with certain principles of the other existing theoretical models of exercise fatigue as described in the literature review (Bilodeau et al., 2001; Calbet et al., 2003; Edwards et al., 1995; Hill et al., 1923). According to the mathematical results obtained from the experimental case studies, this thesis partially supports certain principles or predictions of the current biological control theories. The following paragraphs draw conclusions from these mathematical modelling and analysis case studies and their overall implications in the light of the physiological control model theories. Then, the last section describes recommendations for future research in consolidating the current works of this thesis based on the mathematical findings and the existing physiological control theories, to help delay the onset of fatigue during exercise and improve sports performance.

From the mathematical findings of experimental case study one, the human organism regulates the amount of ATP by setting the metabolic processes (ATP production and depletion) of the anaerobic subsystems and the aerobic energy system at different rates which affected sprint performance. Moreover, the similarity of the time constants, for any anaerobic subsystem, revealed a common controlling mechanism, observed in all the elite athletes, which hinders their sprint performance. Hence, the observations from mathematical modelling and analysis in this particular case study are in agreement with one particular principle of the cardiovascular/anaerobic/catastrophic model which supports the idea that the depletion of a substrate (*i.e.* ATP) may affect the exercising muscles (Brooks et al., 2005; Hill et al., 1923; Shulman and Rothman, 2001). In the same line of thought, the mathematical results also approve one particular prediction of the energy supply/energy depletion model which supports the notion that exercise fatigue may be related to the incapacity of the metabolic energy pathways to generate sufficient energy to the active muscles (Noakes, 2000; Shulman and Rothman, 2001); And in this research, it was shown that the time at which the highest rate of ATP depletion from the PCr anaerobic system occurred, there was a simultaneous decrease in sustaining maximal speed. However, the CAC model and the energy supply/energy depletion model together with the existing physiological control theories (Davis and Bailey, 1997; Gonzalez-Alanso et al., 1999; Roberts et al., 1997; St Clair Gibson and Noakes., 2004; Ulmer, 1996; Weir et al., 2006) of exercise fatigue cannot explain the common controlling mechanism that was observed in regulating the metabolic processes in the physiological systems which influenced sport performance.

Findings from the experimental case study two show that pacing, during an endurance exercise time-trial, influenced the amount of adenosine triphosphate produced from the aerobic and the anaerobic systems but did not affect the total amount of energy produced from these two energy systems. These observations confirm one of the principles of the Energy Supply/Depletion model (Shulman and Rothman, 2001) which postulates that the insufficient amount of energy from a particular metabolic pathway causes exercise fatigue which subsequently impairs sport performance. Furthermore, pacing caused different degrees of homeostatic disturbance in the physiological systems as predicted by the hazard score index mathematical measure which was highest for variable pace, and lowest for self pace time-trial exercise. This degree of homeostatic disturbance influenced the physical behaviour of the cyclists which consequently affected their time-trial performance. In addition, the cyclists felt most fatigued (*i.e.* mean RPE for the whole trial was highest) during the variable pace trials (most anaerobic system dependent) and they felt least fatigued in even pace trial (most aerobic system dependent) whereby blood lactate concentration was highest for variable pace and least in even pace time trial. Undoubtedly, this relationship suggests that blood lactate concentration contributed to the perception of fatigue that the cyclists felt during the race, and hence supports one particular hypothesis of the CAC model (Brooks et al., 2005; Hill et al., 1923; Lucia et al., 2002) which posits exercise-induced fatigue occurs specifically when the rate of production of blood lactate concentration is higher than its rate of removal from the human body. Moreover, results from case study two favour the neuromuscular fatigue model (Davis and Bailey, 1997; Fowles et al., 2002; Nielsen and Clausen, 2000) as it was found that there was a reduction in cycling power output during the self pace time-trial despite an increase in the perception of effort as observed by the rating of

perceived exertion. However, during the variable pace time-trial, both the mean rating of perceived exertion and blood lactate were highest, and according to mathematical results, variable pace was classified as the most anaerobic energy system dependent as it involved high-intensity bouts most often as compared to self pace and even pace. Hence, they probably used most often the fast-twitch muscle fibers which would increase the accumulation of metabolites as shown by the high blood lactate concentration. Then, these observations show that at the expense of employing more elastic muscle (*e.g.* fast-twitch muscle fibers), there may involve an increase in unwanted by-products (as shown by an increase in blood lactate concentration) which conflicts with the efficient elastic energy system principle of the biomechanical model (Pennisi, 1997; Roberts et al., 1997). On the contrary, the task dependency model (Weir et al., 2006) hypothesis matches well with certain mathematical findings in case study two as in fact both the mechanisms (whether the physical activity was anaerobic system dependent or aerobic system dependent), and the perceptions (RPE) of fatigue varied with different types of pacing. Furthermore, there was the presence of the endspurts (accelerations) at the near end of the cycling time trial which showed, firstly, that physical performance was sub-optimal during the race, and secondly, there was the presence of a conscious neural effort which modified the pace representing one of the key principles of the psychological/motivational model (Davis and Bailey, 1997). Therefore, apart from some unbiased predictions of certain principles of the conceptual models in explaining the cause of exercise-induced fatigue, however, they cannot provide an explanation why the total energy expended (from both aerobic and anaerobic systems), for any pacing time trial, were practically constant.

The third experimental case study demonstrated the presence of system control mechanisms based on frequency changes of the spectrum of the physiological system activities during physical activity. This was one of the revolutionary predictions by Ulmer (1996) in the development of the teleoanticipation model stating that a physiological control is essential in optimising sport performance. Moreover, the complexity, and characteristics (*i.e.* in terms of predictability and stability) of the physiological system activities demonstrate the complex behaviour or function of the physiological activities as supported by the integrative central regulator model (Lambert et al., 2004; St Clair Gibson and Noakes., 2004), and the task dependency model (Weir et al., 2006). In the same line of thought, the task dependency model also postulates that the characteristics or properties of these physiological systems together with the type of physical exercise may contribute to the exercise-induced fatigue whereby it was demonstrated mathematically that the power output of each pacing trial was different in complexity (*i.e.* in terms of FD), and these different pacing time-trials influenced the predictability (*i.e.* in terms of determinism and recurrence rate) and stability (*i.e.* trapping time) of the physiological systems. However, none of the principles of these conceptual control models have been able to explain how and why certain physiological systems are more stringently protected than the others during physical activity and how the predictability of the behaviour of any physiological system affects sport performance.

Some physiological models (Lambert et al., 2004; St Clair Gibson and Noakes., 2004) hypothesized that there may be a central regulator that paces the human organism during physical activity by interacting with the various biological systems. The mathematical results from the experimental case study four showed that the

system control mechanisms utilised specific frequency bands simultaneously for control communications with the peripheral systems, and the power of these specific frequency bands influenced (positively or negatively) the physical performance. In order to allocate frequencies to regulate the behaviour or function of the physiological systems, this needs a higher level of control that may happen in the central systems and external to these peripheral systems (Anthea et al., 1993). Moreover, the teleoanticipation model suggested that the efferent signals from that possible central controller determine biological processes from various peripheral systems, and then afferent signals from these physiological systems feedback information to change the pace of the physical activity accordingly. Thus, the interactive communication process between this central regulator and the peripheral systems illustrates the complex function of the human organism (Lambert et al., 2004; St Clair Gibson and Noakes., 2004; Ulmer, 1996). Therefore, the results from experimental case studies 3 and 4 showed that both the simultaneous control and the physiological systems' interactions demonstrate the complex and non-catastrophic behaviour of the functioning of the human organism subjected to a physical exercise which do not support the catastrophic physiological principles as predicted by certain peripheral fatigue models (Hill et al., 1923; Brooks et al., 2005).

The experimental case study 5 demonstrated through indirect measures that there were finite metabolic resources in the brain after performing exhausting physical and cognitive tasks which subsequently change the physical behaviour and cognitive performance of the participants. This change in behaviour was associated to the arousal state or neural activation theory (Steriade, 1996) of the human organism which posits that performance is impaired because of the

incapability to maintain same level of concentration (this same observation was found in this research). This poor cognitive behaviour in the exercise-involved cognitive trial experiment may be related to the prediction of the thermoregulatory model which states that the increase in physiological activities such as heart rate activities may cause an additional physical stress on the human body system (Nybo and Nielsen, 2001) that subsequently impairs performance. Moreover, the Neuromuscular model (Davis and Bailey, 1997) supports this observation by emphasizing that there may be a reduced central activation which consequently causes a decrease in the neuromuscular propagation that affects performance. The feeling of sleep (Gandevia, 1992) and initiation of yawning process (Hermanowicz, 2007; LeWinter, 2007) as observed by the participants post the exercise-involved cognitive trial confirms the presence of a withdrawal signal from continuing mental activity (or to prevent a conscious override on the bodily functions that may cause physiological damage) as predicted by both psychological/motivational model and the integrative central regulator model (Davis and Bailey, 1997; Lambert et al., 2004; St Clair Gibson and Noakes., 2004). Therefore, these physiological control model predictions and mathematical results from this case study do not support the catastrophic-failure principles of the central fatigue model.

From a general point of view, based on the mathematical results and the conceptual biological theories, the aetiology and the underlying mechanism for the exercise-induced fatigue seemed to be influenced by a reductionist approach (Lambert et al., 2004). Most of the hypotheses or predictions (that were assessed in this research) of these biological control models seem plausible because each physiological control model is perceiving the development of exercise fatigue on a

different but yet unbiased narrow perspective. For instance, some models perceived exercise fatigue to be developed peripherally, or centrally, or it is just a sensory perception rather than a physical phenomenon, or it is associated with external physical factors (types of physical activity and external environment). However, only when integrating altogether these possible causes of fatigue, then a clearer picture of the cause of exercise-induced fatigue can be observed. Therefore, the mathematical findings from this thesis as well as the literature review of the biological control models of fatigue extend the existing theory of the nature of fatigue by presenting evidence of a complex non-catastrophic integrative model of fatigue which is regulated by both the central and peripheral systems where changes in physical performance (such as in terms of work output) are both consciously and subconsciously regulated based on endogenous and exogenous feedbacks from the various physiological systems.

9.2 Future recommendations and applications of findings.

The recommendations are based on the mathematical findings of the various experimental case studies conducted in this research together with the existing biological control theories of exercise fatigue.

I. Exercise training for sprint running can be focused on devising 'training protocols' with sufficient recovery periods based on the rate of ATP production and depletion from the PCr utilization and oxygen-independent glycolysis metabolic processes to develop more robust anaerobic subsystems to the development of fatigue. Research could be also directed towards diet manipulation (Burke and Hawley, 2006) where nutritional intake of an athlete is related to exercise performance, and this type of strategy is to keep the athlete properly fuelled for peak performance for that physical activity.

II. More research should be conducted on investigating the 'ideal' pacing for various distances of a time-trial exercise, and also endeavoured to find 'an individualised pacing' based on individual performances. The type of pacing can be assessed in terms of complexity (using fractal dimension) complemented with the hazard score index to evaluate the homeostatic disturbance that each 'simulated' pacing could cause on the physiological systems (de Koning et al., 2011). In so doing, time and energy can be saved as well as costs of conducting physiological experiments are reduced by focusing on the ideal 'pacing protocol' for any particular distance. Interval training can be incorporated specifically for endurance athletes to enhance their aerobic capacity which will consequently delay the onset of fatigue as it was found that even pace was most aerobic energy system dependent whereby the cyclists felt least fatigue. During the interval training, the athletes need to train at a high intensity for a short period of time of about 10 minutes to improve their tolerance towards anaerobic metabolism.

III. Moreover, it was observed that a reduction in the neuromuscular propagation (Fowles et al., 2002; Nielsen and Clausen, 2000) can affect sport performance. Therefore, plyometric training can be used to delay the onset of fatigue by loading the muscle and then contracting it in rapid sequence. This type of exercise training not only can generate rapid and powerful movements but also enhance the nervous system. This is because when the muscle spindles are stimulated by the rapid stretch, there is a greater activation of the muscle and the neuromuscular pathways.

IV. In addition, biofeedback (Gruzelier et al., 2007; Vernon, 2009) may help in the training of the muscular strength. In this research, the physiological states or behaviours (as regulated by the system control mechanisms) influence the physical and cognitive performance but most of the body's physiological processes are not under volitional control. Nevertheless, learning to control certain aspects of physiology can be an important component for performing at peak levels (Peper and Schmid, 1984). Advocates of biofeedback training suggest that this method can help athletes to gain more confidence in their behavioural ability, improve focus, reduce stress and enhance power in sports (Costa et al., 1984; Croce 1986; Norris 1986). One example of biofeedback training (Zaichkowsky and Fuchs, 1988) is that the physiological information is recorded from bio-sensors, then processed and fed back to the individual in the form of an auditory and/or visual signal where the individual will learn to alter the physiological activity. This biofeedback can enhance performance (Landers, 1985) by optimising arousal (either an increase or reduction) according to the inverted-U model (Yerkes and Dodson, 1908); secondly, this biofeedback can improve performance by helping the individual to optimise autonomic control of a particular physiological process. Through a process of learned self-regulation, the athlete can become consciously aware of the processes such as muscle tension to manipulate them. Moreover, Parks (1997) states that biofeedback technique can provide a mechanism for accessing the higher states of consciousness, greater awareness and improved cognition.

V. In the same line of thought, this biofeedback can be extrapolated to train the cognitive strength or mental agility based on the brain activities, which is called the neurofeedback (Dempster and Vernon, 2009; Vernon, 2005; Parks, 1997).

By training the cognitive behaviours, this will eventually enable better mind management; improve mental performance, attention as well as athletic performance. One application can include a compact digital video EEG recording system which can be developed and used as a tool to allow the participants to know consciously their subconscious behaviours or physiological behaviours such as the level of cognitive fatigue or level of arousal so that the athletes can optimise sports performance.

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Appendix A

A.1 The relationship between pseudofrequency and scales

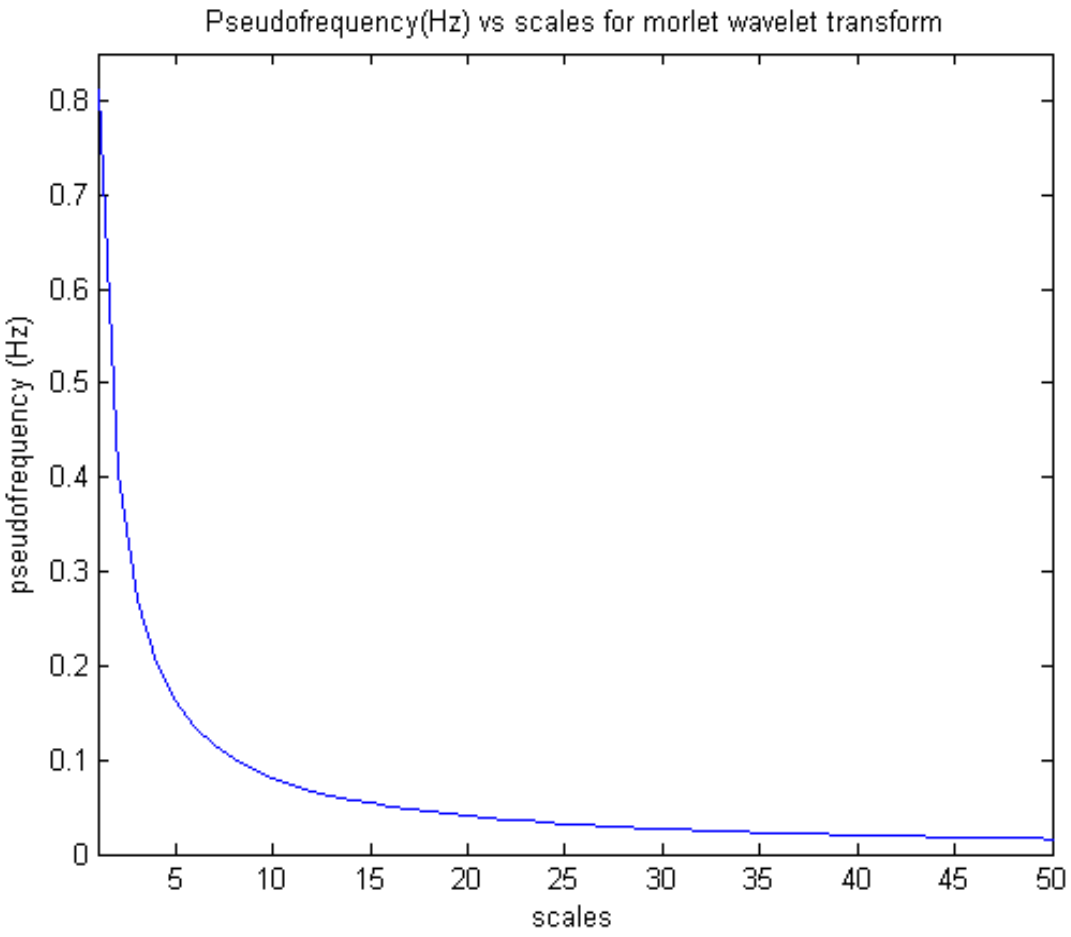

Figure A.1: The figure displays the relationship between pseudofrequency (Hz) and scales for the Morlet wavelet transform.

It is shown for the Morlet wavelet transform that there is an inverse relationship between pseudofrequency and scale. As the number of scales increases from 1 to 50, the pseudofrequency decreases in an inverse-relationship manner from 0.8 to 0 Hz.

A.2 The recurrence plot of a cosine function using different embedding dimensions

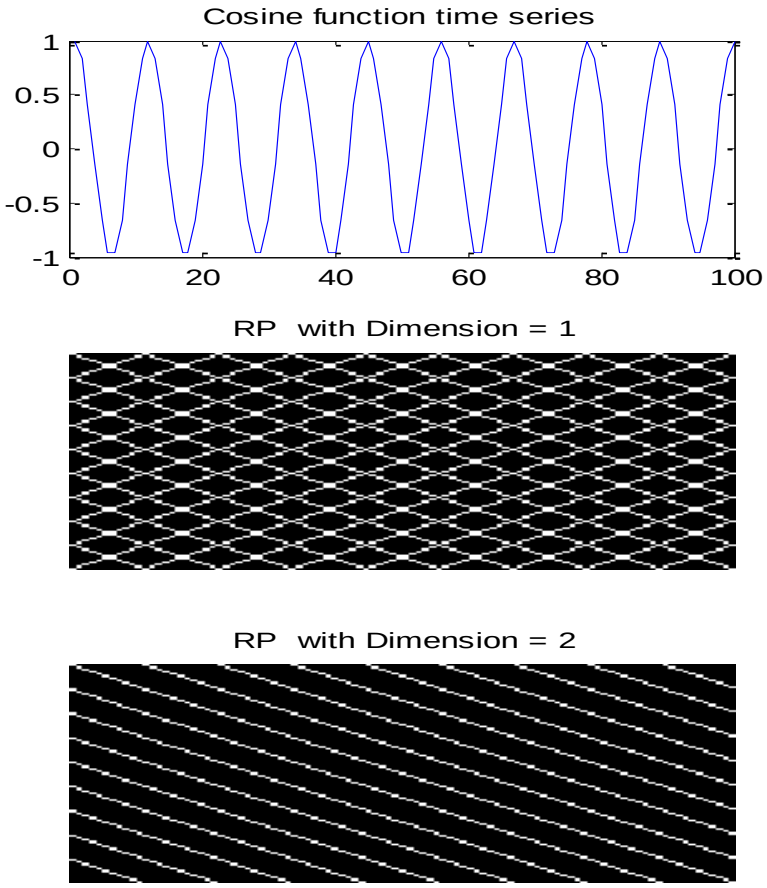

Figure A.2: This figure shows the effect of increasing the embedding dimension (from dimension 1(middle figure) to dimension 2 (bottom figure) using recurrence analysis to represent a cosine signal (top figure).

The main difference that occurred while increasing the dimension of representing a time series signal onto a recurrence plot is that the pattern becomes less complex (from a diagonal grid like pattern for dimension 1 to a stripe pattern for dimension 2). In so doing, one has the tendency to underestimate the true behaviour of this signal.

Appendix B

B.1 The energy components of the external mechanical work

The rate of external mechanical work comprises of three components (Laurent and Locatelli, 2002; Di Prampero et al., 1993; Lloyd, 1967; Peronnet and Thibault, 1989; Ward-Smith, 1985; Ward-Smith and Mobey, 1995).

These three energy components are described as follows:

(i) The first energy component is the rate of change of kinetic energy of the sprinter in the horizontal direction which is given by $v(t) \cdot \frac{dv}{dt}$ where $v(t)$ represents the instantaneous velocity in the horizontal direction, and $\frac{dv}{dt}$ is the rate of change of horizontal velocity;

(ii) The second energy component is the rate of change of potential energy of the sprinter in the vertical direction relative to his crouching state centre of mass height at the beginning of the race is described by $g \cdot \frac{dh}{dt}$ where g is the acceleration of free fall (9.81 ms^{-2}) and $\frac{dh}{dt}$ is the rate of change of vertical height;

(iii) And the third energy component is the rate of work against aerodynamic drag, and it is given by $D(t) \cdot \frac{v(t)}{m}$ where $D(t)$ is the drag force (*N*) at time t and $v(t)$ is the instantaneous velocity (ms^{-1}) while variable m is the mass (kg) of the athlete.

Therefore, the overall equation for the rate of change of external mechanical work is summarised as shown in equations Equ.B.1 (1) and Equ.B.1 (2)

$$\frac{dW}{dt} = v(t)\frac{dv}{dt} + g\frac{dh}{dt} + \frac{D(t)v(t)}{m} \quad , \qquad \text{…Equ.B.1 (1)}$$

where $D(t) = \frac{p\, C_d\, F_a (v(t)-V_w)^2}{2}$ …Equ.B.1 (2)

In equation Equ.B.1 (2), the notation F_a represents the frontal projected area (m^2) of each sprinter, and it is determined by $0.2025 \cdot \mathrm{height}^{0.725}\mathrm{mass}^{0.425}$ (Laurent and Locatelli, 2002); the notation C_d is the drag coefficient and it is 0.9; the variable V_w represents the wind speed (ms^{-1}) and the symbol p is the air density (kgm^{-3}).

B.2 The rate of change of aerobic energy

The rate of chemical energy derived from aerobic metabolism is calculated from the following equation:

$$\frac{dCae}{dt} = R(1 - e^{-\lambda t}) \qquad \text{… Equ.B.2 (1)}$$

In Equ.B.2 (1), the variable R is the maximum sustainable aerobic power per unit mass, and the variable λ represents the rate of aerobic energy release (van Ingen Schenau et al., 1995).

B.3 Flowchart diagram summarising the computation of the parameters

In the flowchart diagram (Figure B.1), the linear variable (P) represents power of the anaerobic subsystems. The error (ε) is kept to a minimum by calculating the residual error between the calculated anaerobic power, and the sum of the estimated anaerobic subsystem powers. The nonlinear parameters (β_1, β_2 and β_3) represent the rate constants of the anaerobic subsystem power distributions (the working principle of this flowchart diagram is described in Chapter three).

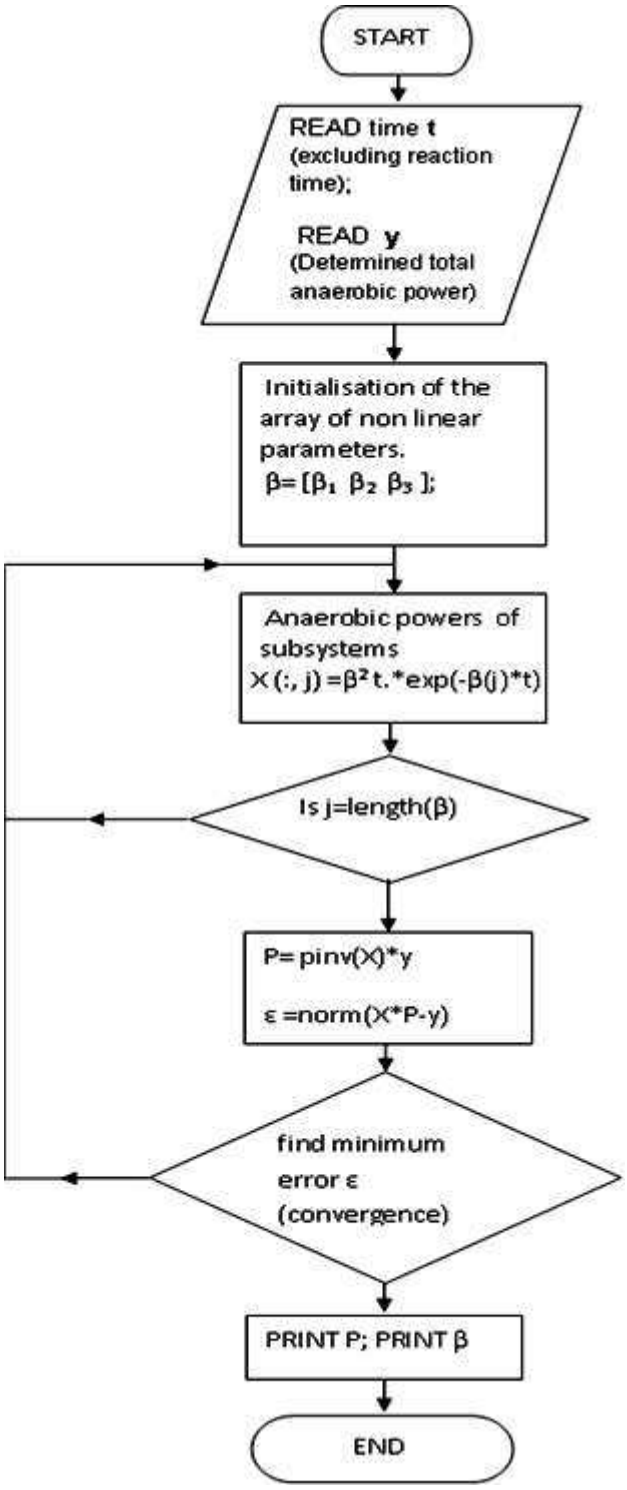

Figure B.1: Flowchart of the computational program to find the linear variables (anaerobic subsystem powers), and the nonlinear variables (the rate constants of the anaerobic subsystem powers).

B.4 Gamma distribution model to represent the three anaerobic subsystems' powers

According to Wlodarczyka and Kierdaszuk (2006), Gamma distribution model is used extensively in biochemistry and it is a flexible model for physical systems that are exponentially distributed as well as it represents a good fit for the sum of independent exponential random variables. It is expressed and parameterized in terms of a shape (α) parameter, and a rate parameter (β).

$$g(t;\ \alpha,\beta)\ = \frac{\beta^{\alpha}}{\Gamma(\alpha)}\ t^{\alpha-1}e^{-\beta t}\ \ , \qquad \text{... Equ. B.4(1)}$$

Where $\Gamma(\alpha) = (\ \alpha - 1)!$ ... Equ. B.4(2)

For this model, the shape α is taken as 2 so that a first-order in time,t, is produced to represent the behaviour of each anaerobic subsystem power distribution curve (Hogg and Craig, 1978). And therefore, the rate of change of chemical energy is expressed as the sum of three first-order gamma distribution function, and is shown in the following equations (B.4(3) and B.4(4)). By taking the shape α to be 2, the equation of gamma distribution function to represent the behaviour for each anaerobic subsystem power simplifies to:

$$g(t;\beta)\ = \frac{\beta^{2}}{\Gamma(2)}\ t^{(2-1)}e^{-\beta t}\ \ , \qquad \text{... Equ. B.4(3)}$$

And $\Gamma(2) = (\ 2 - 1)! = 1! = 1$ ... Equ. B.4(4)

By taking the results of Equ. B.4 (4), the Equ. B.4 (3) simplifies further as shown in Equ. B.4 (5) which consists of the factor time t and the rate parameter β.

$$g(t;\beta) = \beta^2 \cdot t \cdot e^{-\beta t} \quad , \qquad \text{... Equ. B.4(5)}$$

Now the rate of change of chemical energy from the anaerobic subsystems is the sum of the rate of change of energy from the three anaerobic subsystems that were the ATP-endogenous, PCr utilisation anaerobic process, and the oxygen-independent glycolysis anaerobic subsystem.

$$\frac{dCan}{dt} = g_1(t;\beta_1) + g_2(t;\beta_2) + g_3(t;\beta_3) \qquad \text{... Equ. B.4(6)}$$

$$\frac{dCan}{dt} = {\beta_1}^2 \cdot t \cdot e^{-\beta_1 t} + {\beta_2}^2 \cdot t \cdot e^{-\beta_2 t} + {\beta_3}^2 \cdot t \cdot e^{-\beta_3 t} \qquad \text{... Equ. B.4(7)}$$

Since the rate of change of energy of the anaerobic subsystem is actually the power (*P*) of the anaerobic subsystem,

$$P_n = g_n(t;\beta_n) \qquad \text{... Equ. B.4(8)}$$

In Equ. B.4 (8), the nonlinear parameter $\beta_n = \frac{1}{\tau_n}$ represents the inverse of time constants of each individual distribution for the three subsystems respectively. Then, Equ. B.4 (7) simplifies to the following equation:

$$\frac{dCan}{dt} = P_1(t) + P_2(t) + P_3(t) \qquad \text{... Equ. B.4(9)}$$

In Equ. B.4 (9), the notation P_n represents the anaerobic subsystem powers at time *t* and it is measured in *watts per kilogram (*Wkg^{-1}). This is simplified in mathematical notation into the following equation:

$$\frac{dCan}{dt} = \sum_{n=1}^{3} P_n(t) \, , \qquad \text{... Equ. B.4(10)}$$

The linear parameters P_1 denotes the rate of energy released from endogenous ATP at time t, P_2 denotes rate of energy released from Phosphocreatine (PCr) utilisation at time t and P_3 denotes rate of energy released from oxygen-independent glycolysis utilisation at time t.

B.5 Anaerobic subsystem power and the corresponding rate parameter for each sprinter.

The mean and standard deviation of the anaerobic subsystem powers P_1, P_2 and P_3 were 6.6±1.78 Wkg^{-1}, 40.5±2.97 Wkg^{-1} and 9.98±1.04 Wkg^{-1} respectively. The rate parameters of the anaerobic subsystems β_1, β_2, and β_3 were 0.94±0.05s^{-1}, 0.31± 0.015 $s^{-1}$and 0.11±0.004s^{-1}.

Ranking of sprinters	Anaerobic subsystem power (Wkg^{-1})			Nonlinear variables (s^{-1})		
	P_1	P_2	P_3	β_1	β_2	β_3
1	7.1	42.8	10.8	0.9239	0.3315	0.1130
2	7.4	38.9	9.4	0.9341	0.2962	0.1131
3	7.9	44.1	10.8	0.9286	0.3352	0.1115
4	9.5	37.3	10.2	0.9956	0.2990	0.1075
5	3.6	39.8	10.8	0.8486	0.3045	0.1194
6	6.0	38.8	10.1	0.9652	0.3152	0.1112
7	5.7	37.5	10.1	0.9570	0.2958	0.1151
8	5.6	44.8	7.7	0.9863	0.3038	0.1109

Table B.1: The results for the anaerobic subsystem power per unit mass, and the nonlinear parameters for all the elite sprinters are shown.

Appendix C

C.1 Statistical *F*-ratio

The F-ratio is a test statistic to find whether the difference between two or more independent variables is statistically significant or stable by computing the ratio of the variance between groups, and the variance within groups (Lomax, 2007; Sawilowsky, 2002). The computation of the statistics F-ratio is summarised in the following equations (Lomax, 2007):

$$F = \frac{between - group\ variability}{within - group\ variability}$$

In the above formula, the between-group variability is given by:

$$\frac{\sum_i \ n_i \ (\boldsymbol{y_i} - \boldsymbol{y})^2}{k - 1}$$

And the within-group variability is given by:

$$\frac{\sum_{ij} \ n_i \ (y_{ij} - \boldsymbol{y_i})^2}{N - k}$$

The variables $\boldsymbol{y_i}$, n_i, $\boldsymbol{y}$, k, y_{ij}, N represent the sample mean in the i^{th} group, the number of observations in the i^{th} group, the overall mean of the data, the number of groups, the j^{th} observation in the i^{th} out of k groups and the overall sample size respectively.

Moreover, the number of degrees of freedom for the between-group variability is represented by the notation df1 and the number of degrees of freedom for the within-group variance is represented by the notation df2. Therefore, these two degrees of freedom are represented by the following formula where k and N represents the number of participants (sample size) and k is the number of groups.

$$\text{df1} = k - 1 \ldots \text{(i)}$$

$$\text{df2} = N - k \ldots \text{(ii)}$$

C.2 Product moment correlation coefficient

The Pearson's correlation coefficient between two variables is defined as the covariance of the two variables (X and Y) divided by the product of their respective standard deviations (σ_X ,σ_Y). This is represented by the following mathematical equations.

$$\rho_{X,Y} = \frac{cov\,(X,Y)}{\sigma_X \sigma_Y} ,$$

where cov is covariance, notation σ_X represents the standard deviation for variable X and notation σ_Y represents the standard deviation for variable Y.

$$\rho_{X,Y} = \frac{\mathrm{E}\,[(X-\mu_X)(Y-\mu_Y)]}{\sigma_X \sigma_Y}$$

Where symbol E represents Expectation, μ_X represents the mean of the variable X and $\mu_Y$represents the mean of variable Y.

Appendix D

D.1 The frequency band power for volume of oxygen consumption (VO_2)

The mean normalized wavelet power for each frequency band for the volume of oxygen consumption physiological activity for all cyclists for each pacing time trial is depicted in Figure D.1. Moreover, the changes in HF band, LF band and ULF band for this particular physiological activity are compared as shown below.

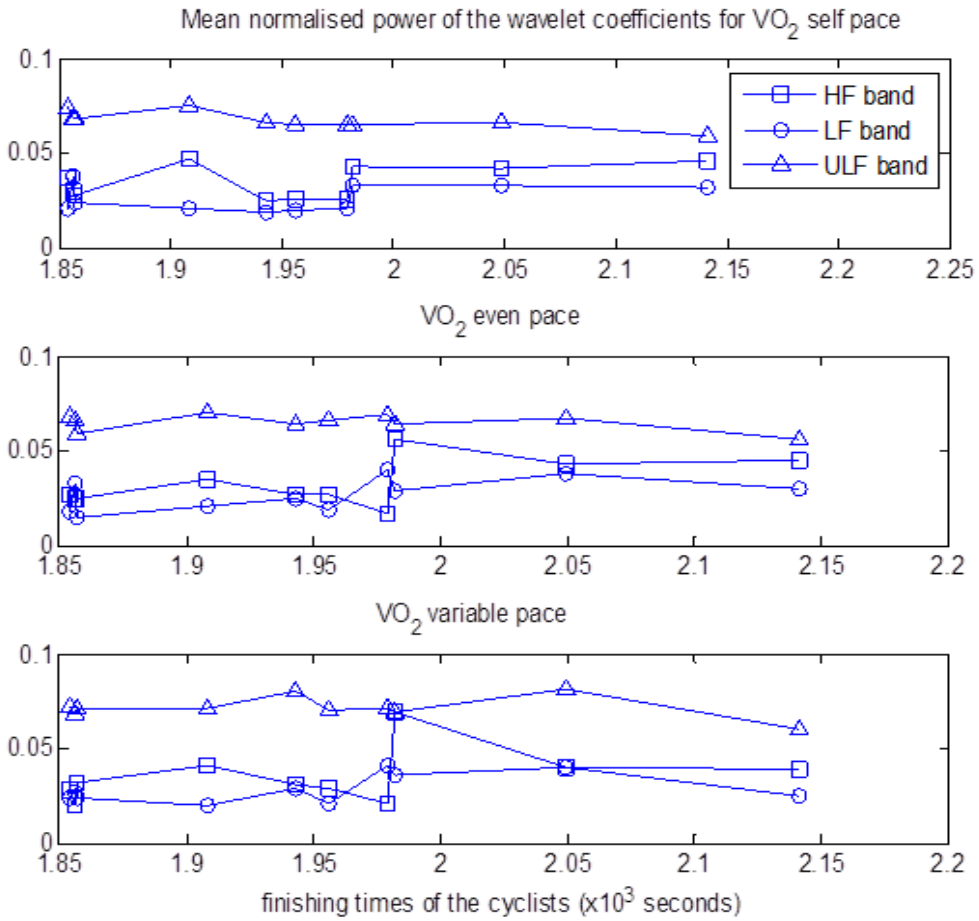

Figure D.1: The x-axis represents the finishing times of the cyclists and the y-axis represents the normalised wavelet power so that the changes in the three different frequency bands can be compared.

D.2 The frequency band power for heart rate (HR)

The mean normalized wavelet power for each frequency band for heart rate physiological activity for all cyclists for each pacing time trial is depicted in Figure D.2. Moreover, the changes in HF band, LF band and ULF band for this particular physiological activity are compared as shown below. There was a significant difference between HF band and LF band ($p < 0.01$), and there was a small positive correlation between HF band with increasing performance times of the cyclists (correlation $r = 0.3$ and statistical $p = 0.03$).

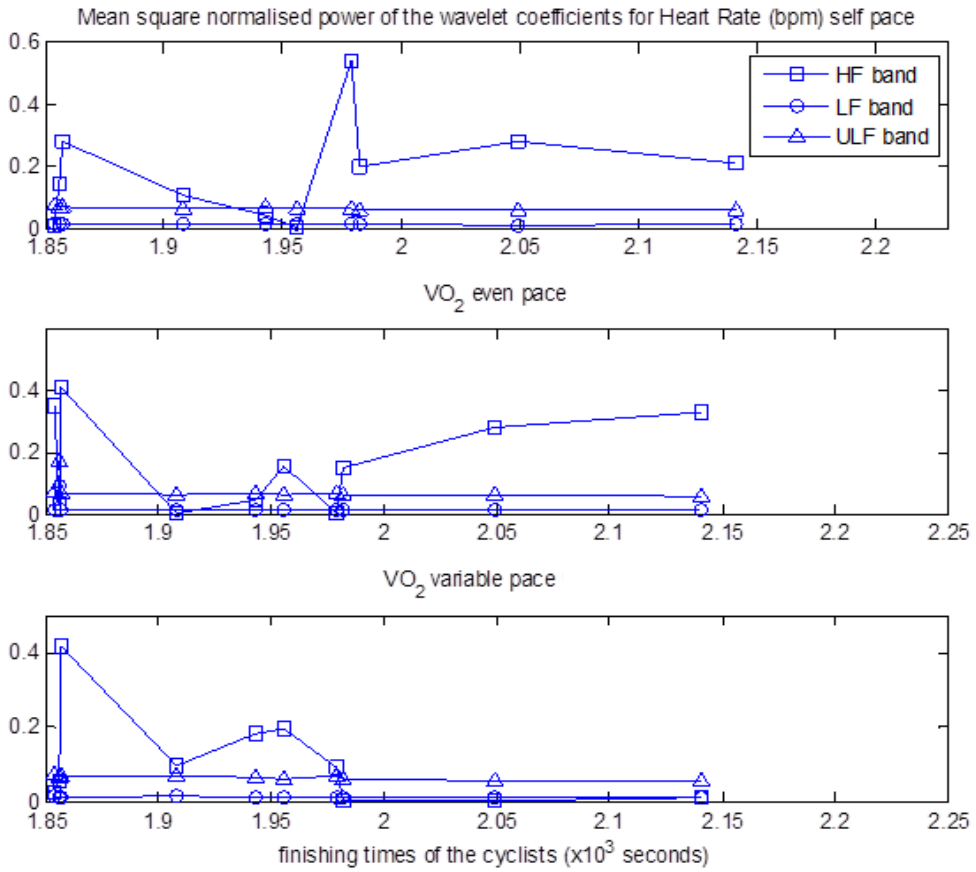

Figure D.2: The x-axis represents the finishing times of the cyclists, and the y-axis represents the normalised wavelet power of the heart rate activities so that the changes in the three different frequency bands can be compared.

Appendix E

E.1 Descriptive statistics for the percentage accuracy of responses

The descriptive statistics for the accuracy of the responses of the participants while responding to the visual cues for both the control and exercise experimental condition is shown in Table E.1. In addition, the average accuracy of the responses for every 5 minute block cognitive task trial for a period of 30 minutes is shown.

Time-on-task (minutes)	Condition	Mean	Standard deviation
5	Control	96.667	1.3027
	Exercise	93.667	1.4975
10	Control	94.000	1.2000
	Exercise	92.000	1.2660
15	Control	92.000	1.6396
	Exercise	90.000	1.8396
20	Control	90.000	1.8528
	Exercise	88.000	1.3501
25	Control	88.000	0.8528
	Exercise	86.500	1.5667
30	Control	87.000	0.6396
	Exercise	84.750	1.2154

Table E.1: Descriptive statistics for the percentage of accuracy of responses (n = 12).

E.2 Descriptive statistics for reaction time (ms) of the responses

The descriptive statistics for the reaction time of the participants while responding to the visual cues for both the control and exercise experimental condition is shown in Table E.2. In addition, the average reaction time of the responses for every 5 minute block cognitive task trial for a period of 30 minutes is shown.

Time-on-task (minutes)	Condition	Mean	Standard deviation
5	Control	550.0000	20.0280
	Exercise	320.0000	10.0302
10	Control	530.0000	8.52803
	Exercise	355.0000	6.39602
15	Control	490.0000	12.06045
	Exercise	390.0000	6.39602
20	Control	455.0000	6.39602
	Exercise	425.0000	8.52803
25	Control	450.0000	8.52803
	Exercise	425.0000	9.53463
30	Control	470.0000	20.07920
	Exercise	405.0000	15.39602

Table E.2: Descriptive statistics for the reaction time (ms) of responses (n = 12).

E.3 Descriptive statistics for the cognitive ratios

The descriptive statistics for the average cognitive ratios of the participants for both the control and exercise-involved cognitive trials are shown in Table E.3. In addition, the table displays the values of the cognitive ratio for every 5 minute interval of the cognitive trial for both experimental conditions.

Time-on-task (minutes)	condition	Mean	S.D
5	1.00	2.7167	.57181
	2.00	3.0250	.46734
	Total	2.8708	.53445
10	1.00	2.7083	.29064
	2.00	2.8667	.39158
	Total	2.7875	.34680
15	1.00	1.0958	.44643
	2.00	1.0208	.32576
	Total	1.0583	.38410
20	1.00	1.2583	.48140
	2.00	1.3667	.40973
	Total	1.3125	.44066
25	1.00	1.1583	.35982
	2.00	1.2000	.01706
	Total	1.1792	.25002
30	1.00	1.0333	.21881
	2.00	1.0083	.26443
	Total	1.0208	.23770

Table E.3: Descriptive statistics for the cognitive ratio for both experimental conditions (n = 12).

Appendix F

Recurrence Quantification Analysis of the System Control Mechanisms underlying Physiological Data

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In exercise physiology, the study of the complex rhythms arising from the peripheral and central systems of the human body is crucial to optimise athletic performance. According to a novel theoretical model of fatigue[1, 2], there is a central governor in the brain that regulates the physical activity to ensure that this exercise activity is completed without homeostasis failure through interactive communication between the physiological and central systems in a deterministic way. Therefore, there is an increasing need to investigate on the characteristics of these system control mechanisms that regulate our homeostasis and control our behaviour and activity. In order to determine the characteristics of these complex system control mechanisms, recurrence analysis is used to locate any rhythms or patterns in these physiological data[3, 4]. In this study, various pacing strategies that are self pace, even pace and variable pace were used for a 20-km cycling time trial to observe how these pacing strategies influence the heart rate (HR) activities (BPM) and the volume of oxygen consumption (VO_2 / $L \cdot min^{-1}$) of these cyclists.

It is observed that for VO_2, there is no significant difference between the RQA measures that are recurrence rate (RR), determinism (DET) and trapping time (TT) for all ten cyclists performing the different pacing strategies. The mean RR is 9%, DET is 29.6% and TT is 2.5 s and there is no significant difference between the aforesaid RQA measures for heart rate activities for all pacing strategies and the mean value RR is 10%, DET is 89% and TT is 8.2 s. The difference in the trapping times and the determinism values between VO_2 and HR suggests that each physical system has different characteristic behaviour. It is observed that the heart rate activities of these cyclists stay three times longer in a particular physiological state than that of the respiratory system. Interestingly, for the heart rate with mean DET of 89% implicates that these activities can be predicted much easier than the random process of the events observed from the respiratory system even though the probability of recurrence in both cases are low. Moreover, the trapping times for the heart rate activities are significantly different for each pacing strategy and there is the tendency of the imposed pacing strategy to force that physical system to imitate its behaviour as shown by the duration of the mean trapping time to remain in a state is highest in even pace (10.9s) and least in variable pace (6.7s).

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Continuous Wavelet Analysis of Physiological Data for various Pacing Strategies of a 20-km Cycling Time Trial

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Recently, in exercise physiology, a novel model to regulate the central neural effort and fatigue has been proposed. This model theorizes that physical activity is controlled by a central regulator in the brain, and the human body works as a complex integral system, unlike the Cardiovascular/Anaerobic/Catastrophe model of Sir A.V. Hill of exercise physiology. In this study, physiological data were collected from club level cyclists for different pacing strategies that were self pace, even paced, and variable paced for a 20km cycling time trial in order to assess the underlying system control mechanisms that show how the brain paces the human body during exercise. Continuous Wavelet Transform (CWT) was used to analyse the non-stationary and nonlinear physiological signals that were heart rate (HR/bpm) and volume of oxygen consumption (VO2/ $Lmin^{-1}$). Normalised mean wavelet powers were used to compare the powers at different frequency bands of the continuous wavelet spectrum. These frequency bands were classified as High Frequency (HF), Low Frequency (LF) and Ultra Low Frequency (ULF) bands. There was a significant difference in the ULF band for the volume of oxygen consumption ($p<0.01$) that decreased with increasing performance times of cyclists for all pacing strategies. As for the heart rate activities, both ULF and LF band powers were practically constant for all cyclists, and there was a significant difference in the HF band power compared to the other frequency bands. It was shown that the brain paces the human body by acting as an external drive to that particular peripheral system and it uses specific frequency bands to control and communicate with a particular peripheral system in the aims to reach the end of that physical task without homeostasis failure.