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**The role of perception of effort in  
endurance performance testing and  
training**

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2016

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*GLOSSARY*

ACC	anterior cingulate cortex
ANOVA	analysis of variance
AR	active recovery
BL	blood lactate
CAD	cadence
CI	confident interval
CGM	the central governor model
CNS	central nervous system
CON	control group
EMG	electromyography
GET	gas exchange threshold
HIIT	high intensity interval training
HR	heart rate
LT	lactate threshold
MF	mental fatigue
MMP	maximum minute power output
MAP	maximal aerobic power
MVC	maximum ventilatory contraction
PLA	placebo group
PE	perception of effort
RPE	rating of perceived exertion
SSE	standard error estimation
TTE	time to exhaustion
$\dot{V}O_{2max}$	maximal oxygen consumption
W'	anaerobic work capacity

*Declaration*

The material contained within this thesis is original work conducted and written by the author. The following communications is a direct consequence of this work.

Conference communications

**Salam, H.,** Hopker, J, and Marcora, S. The effect of mental fatigue on critical power and the anaerobic work capacity (Abstract). 20<sup>th</sup> Annual European College of Sport Science Congress, Malmö- Sweden.

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# **CHAPTER 1**

## **GENERAL INTRODUCTION**

## **General introduction**

### **Physiological Determinants of Endurance Performance**

The determinants of endurance exercise performance have been studied for decades, from simple measures such as heart rate, to whole body measures including the maximal oxygen consumption ( $\dot{V}O_{2max}$ ), and more recently via microscopic molecular and genetic markers. The physiology underpinning endurance exercise performance was being investigated as early as 1910, when August Krogh developed a cycle ergometer to accurately determine oxygen consumption and energy expenditure. In the 1920s, A.V. Hill presented the concept of  $\dot{V}O_{2max}$ , and along with Henry Taylor, Per-Olof Åstrand and Bengt Saltin in the 1950s and 1960s, developed some of the seminal methodological approaches to measuring physiological parameters associated with endurance exercise performance. Indeed, Åstrand and Saltin's work from the 1960s was the first to present a clear relationship between  $\dot{V}O_{2max}$  and endurance exercise performance across a range of sports. However, it was also acknowledged that endurance exercise performance was not solely governed by an athlete's  $\dot{V}O_{2max}$  and that other submaximal physiological parameters were also important.

Thus in the 1970s and early 80s researchers also began to investigate the relationships between the lactate and/or ventilatory thresholds and endurance exercise performance as a way of determining the percentage of  $\dot{V}O_{2max}$  that could be sustainable for a given period of time. In this regard, Costill (1970) demonstrated a strong relationship between the curvilinear blood lactate response during an incremental exercise test, and marathon running performance. More recent research has established the concept of the lactate threshold within an incremental exercise test, and attempted to link it to endurance exercise performance by suggesting it

interacts with  $\dot{V}O_{2\max}$  to determine the exercise intensity that can be sustained for a given period of time during endurance exercise, also termed the “performance  $\dot{V}O_2$ ” (Holloszy and Coyle, 1984; Holloszy et al., 1977; Robergs et al., 2004).

The next factor that makes an important contribution to the physiology of endurance performance is exercise efficiency or economy. The efficiency or economy determines how much speed or power can be generated by an athlete for a given level of oxygen consumption. For example, research has demonstrated that the oxygen cost of running at a given speed can vary by up to ~40% (Costill et al., 1973; Farrell et al., 1979). Economy is determined by plotting oxygen consumption ( $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) against running velocity ( $\text{m}\cdot\text{min}^{-1}$ ), or by basically expressing the energy required per unit mass to cover a certain distance ( $\text{mL}\cdot\text{O}_2\cdot\text{kg}^{-1}\cdot\text{km}^{-1}$ ). As a direct measurement of power output is possible in cycling, it is possible to calculate mechanical efficiency, which is the relationship between work done and energy expended. This is similar to running economy, as when cycling at a given power output, the oxygen consumption and thus gross mechanical efficiency varies between individuals and has been shown to be a determinant of cycling performance (Hopker et al., 2012).

More recently, researchers have proposed a further ‘threshold’ related to endurance exercise performance, termed the critical power. Critical Power (CP) is defined as the estimation of the power output that can be sustained for a very long time without fatigue (Monod and Scherrer, 1965). Indeed, Smith et al. (1999) have demonstrated that the CP is correlated with 17 km cycling time trial performance in trained cyclists. In this context, if the cyclist were to ride at an intensity above their CP, then they would be on “borrowed time” as they progressively expend the finite work capacity that is available above the CP, known as  $W'$  (sometimes called the “anaerobic work capacity”). The  $W'$  is thus defined as the highest fixed work rate that can be performed above critical power prior to exhaustion (Poole et al., 1988).

It is important to acknowledge that endurance exercise performance is not solely determined by “neck-down” physiological factors. Indeed, there is clear evidence of a significant role played by the brain in determining endurance exercise performance.

### **Psychological Determinants of Endurance Exercise Performance**

The classification of practical psychological interventions that enhance endurance performance such as (Association and Dissociation, Goal Setting, Hypnosis, Imagery, Pre-Performance Statements, Psychological Skills Training Packages, and Self-Talk) as well as additional psychological factors (External Motivators, Mental Fatigue, Priming, and Experimenter Effects) that influence endurance performance could assist the performance of competitive endurance athletes (McCormick et al., 2015). Furthermore, identifying methods for enhancing endurance performance could persuade recreational subjects to make continued contributions to sport by increasing their self-efficacy (Desharnais et al., 1986) or perceived competence (Ryan et al., 1997). Although experimental investigations have been testing the effects of psychological factors on endurance performance for nearly five decades (Wilmore, 1968) and while, psychological theories have highlighted the effects of psychological factors on perception of effort (Rejeski, 1985; Tenenbaum, 2001), the psychobiological model of endurance performance is the only model that depends upon psychological theory that exclusively shows how psychological factors affect endurance performance. The psychobiological model depends upon motivational theory (Brehm and Self, 1989) and it proposes that potential motivation and perception of effort are the ultimate determinants of endurance performance (Smirmaul et al., 2013). Therefore, these interventions that influence perception of effort consistently affect endurance performance.



## **Perceived Exertion and Exercise Performance**

The Oxford Dictionary defines effort as “strenuous physical or mental exertion” and exertion as “physical or mental effort”, and has been proposed to be a powerful regulator of exercise intensity (Noble and Robertson, 1996). The concept of the perception of effort was first investigated within the field of exercise science by Gunnar Borg in the 1960’s. Borg (1962) proposed that the “perception of effort” be used as a subjective complement to the objective responses during physical tasks. He defined the perception of effort as “the feeling of how heavy, strenuous and laborious exercise is” (Borg, 1962). Within his text, Borg (1962) suggests that perception of effort is generated as a result of increased respiration or breathing rate, increased sweating, increased heart rate, and from muscle fatigue, the skin and the joints (Borg, 1998). Borg subsequently developed a scale to assess this concept known as Borg’s rating of perceived exertion (RPE) scale (Borg, 1998), which has since been widely accepted and used in the study of perception of effort during exercise.

Furthermore, the definition of perceived exertion has been expanded to include the notion of fatigue and discomfort. Noble and Robertson (1996), defined the perception of effort as “the subjective intensity of effort, strain, discomfort, and/or fatigue that is experienced during physical exercise.” However, it is well accepted that individuals can differentiate the perception of effort from other exercise-related sensations. As a consequence, Marcora (2009) proposed the perception of effort as “the conscious sensation of how hard, heavy, and strenuous a physical task is.” Indeed, Scherr et al. (2012) have demonstrated a strong link between the perception of effort and physiological parameters associated with endurance exercise performance (heart rate and blood lactate concentration) in 2,560 participants. Scherr et al. (2012) reported a strong relationship between the perception of effort (Borg RPE 6-20 scale), heart rate ( $r = 0.74$ ,  $P < 0.001$ ) and blood lactate concentration ( $r = 0.83$ ,  $P < 0.001$ ) during

submaximal exercise performance. Moreover, a fixed blood lactate level of 3 or 4 mmol/L was seen to correlate with RPE values of 10.8 and 13.6 respectively.

Moreover, recent research has attempted to demonstrate a link between the perception of effort and endurance exercise performance by experimentally manipulating the perception of effort via mental fatigue. An increased level of perceived exertion during both whole body (Marcora et al., 2009) and single-joint (Pageaux et al., 2013) exercise has been demonstrated when individuals are in a mentally fatigued state following a prolonged period of cognitively demanding tasks. These studies suggest that perception of effort can determine endurance exercise performance independently of alterations in cardiorespiratory, metabolic and neuromuscular parameters. Therefore, it is possible that perception of effort plays a major role in determining endurance performance (Marcora and Staiano, 2010). However, none of these studies have investigated whether the perception of effort can be used as a predictor of endurance performance as has previously been shown with traditional laboratory derived physiological parameters (e.g.  $\dot{V}O_{2\max}$ , lactate threshold, exercise efficiency; Joyner and Coyle, 2008). Therefore, the first study of this thesis aimed to establish whether the perception of effort could be used to predict endurance performance in comparison to more traditional physiological parameters such as  $\dot{V}O_{2\max}$ , lactate threshold, and exercise economy.

#### *Experimental manipulation of the perception of effort and exercise performance*

As outlined above, previous experimental studies (e.g. Marcora et al., 2009; Pageaux et al., 2013) have sought to manipulate perception of effort via the use of mentally fatiguing tasks as a method to validate its effect on endurance performance. However, this phenomenon was first demonstrated much earlier. In 1906, Mosso performed the first documented experiment assessing the relationship between mental fatigue and exercise performance by testing the

performance potential of his colleagues after long physiology lectures and viva examinations. The results of Mosso's study demonstrated that participants did comparatively poorly in a muscle fatigue test performed after the prolonged mental tasks involving sustained concentration. More recent studies have confirmed the findings of Mosso, by demonstrating the negative effect of prolonged mental exertion on self-paced endurance tasks (Marcora et al., 2009; Pageaux et al., 2013; Pageaux et al., 2014).

Thus, a purely cognitive process following mental exertion could mediate an alteration in the work vs. TTE relationship and perception of effort during exercise (de Moree et al., 2012). In this context, it is proposed that an individual will engage in an exercise task until the maximum amount of effort they are willing to exert (the potential motivation) is reached, or when success seems impossible (Marcora and Staiano, 2010). Once this point is reached, the individual will terminate their participation in the exercise task. This raises some concerns for exercise testing which involves time-to-exhaustion type testing. If exercise test participants arrive at the laboratory in a mentally fatigued state, it is possible that their performance will be reduced. In this regard, some concepts within exercise physiology, such as critical power, rely heavily on time-to-exhaustion testing. The mathematical calculation of critical power is subsequently derived from the plotting of total work done against time-to-exhaustion (Monod and Scherrer, 1965). The original work by Monod and Scherrer (1965) extracted two parameters CP, and a finite amount of work performable above CP (the "energy store"), more recently termed  $W'$ .

Experimental studies have demonstrated that CP is determined by oxidative function and that  $W'$  can be independently manipulated by altering muscle phosphocreatine stores, suggesting that it is dependent on finite anaerobic energy sources (Moritani et al., 1981; Poole et al., 1990). However, as CP and  $W'$  are derived from a series of constant work rate trials time-to-exhaustion exercise trials, their execution is largely determined by the perception of effort.

Thus, it is entirely feasible that the mathematically derived concepts of CP are modifiable by mental fatigue and increased perception of effort, as opposed to any physiological parameters previously “unequivocally” proposed to determine it (Jones et al., 2010). Thus, the second study of this thesis aimed to investigate whether manipulation of the perception of effort by inducing mental fatigue altered the CP and  $W'$ .

If mental fatigue increases the perception of effort during exercise, caffeine ingestion has been shown to have the opposite effect (Cole et al., 1996). Specifically, regardless of mode, intensity, or duration of exercise, caffeine ingestion has been shown to alter the perceptual response to exercise by either increasing the work output for a given PE (Cole et al., 1996; Plaskett and Cafarelli, 2001), or a reduced PE at a constant exercise intensity (Birnbaum and Herbst, 2004; Doherty et al., 2002, 2004). However, statistically significant effects of caffeine on PE have not been demonstrated in all research. Tarnopolsky et al. (1989) found that caffeine ingestion reduced PE compared to a placebo (by 16%) during exercise at 70%  $\dot{V}O_{2max}$ , but this was not identified to be a statically significant change. However, it is likely that the small sample size ( $n=6$ ), meant that Tarnopolsky et al. (1989) did not have enough statistical power within their methodology to detect this as a significant reduction in the PE.

Despite the body of evidence that consistently demonstrates the favourable effect of caffeine ingestion on the PE, this effect has not been exploited in the context of training. For example, if caffeine ingestion prior to training facilitated an increased work output for a given PE (Cole et al., 1996; Plaskett and Cafarelli, 2001) during training, it follows that a greater exercise intensity could be sustained within a given volume of training. In this regard, it has been suggested that high intensity training protocols (HIT), that elicit  $\dot{V}O_{2max}$ , or at least a very high percentage of  $\dot{V}O_{2max}$ , maximally stress the oxygen transport and utilisation systems and may therefore provide the most effective stimulus for endurance training (Laursen and

Jenkins, 2002; Midgley et al., 2006). For an optimal training stimulus, it is believed that individuals should spend at least several minutes per HIT session attaining an intensity greater than 90  $\dot{V}O_2\text{max}$  (Billat, 2001; Laursen and Jenkins, 2002; Midgley et al., 2006; Midgley et al., 2007). Thus, the ability to sustain a greater exercise intensity, or a HIT repetition for a longer duration as a result of caffeine ingestion would facilitate a greater percentage of  $\dot{V}O_2\text{max}$  to be sustained, or a longer time to be spent at  $\dot{V}O_2\text{max}$ .

Although the long-term ingestion of caffeine to facilitate a greater volume of HIT is an attractive idea, there is the suggestion that the effects of caffeine may be diminished over time, i.e. individuals become habituated to caffeine (Tarnopolsky et al., 1989; Van Soeren et al., 1993), or habitual caffeine users might be less susceptible to the effects of caffeine (Dodd et al., 1991). However, the effect of caffeine ingestion on the PE during HIT exercise has not been investigated within the context of a chronic period of exercise training and caffeine supplementation. Therefore, the aim of the final study in this PhD thesis was to test the hypothesis that caffeine acutely increases power output, heart rate (HR), and blood lactate during high intensity interval training (HIIT), and the second aim of the study was to test the hypothesis that chronic use of caffeine reduces its hypothesised acute ergogenic effects during HIIT (tolerance).

## **CHAPTER 2**

# **Literature review**

## **I. Literature review**

### **I. Endurance performance**

Endurance exercise performance can be described by the length of time that a given power can be sustained (i.e. capacity) or by the amount of time required to complete a given amount of work (i.e. power) (Coyle, 1995).

A number of researchers have attempted to model endurance performance by describing the multiple physiological systems that they believe contribute to performance. Although these are not necessarily specific to one endurance event, they are useful in explaining some of the key physiological factors associated with cycling performance. One such model is provided by Joyner and Coyle (2008) who propose that endurance performance can be defined by  $\dot{V}O_{2max}$ , the lactate threshold, performance  $\dot{V}O_2$ , and exercise efficiency/economy (See Figure 1). However, there are significant complexities associated with a model such as this. When you physiologically, psychologically or biomechanically attempt to unpick a component of the model, there are numerous additional components that could be included in each area. Despite the limitations that all proposers of such models acknowledge, these types of model are very useful for describing the physiological factors that contribute to an individual rider's success or limitations.

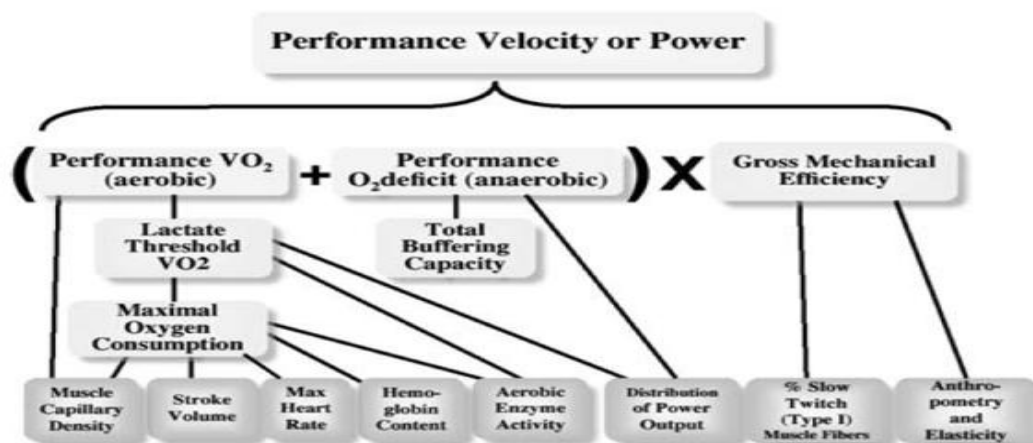


Figure 1: The Joyner and Coyle performance model (Joyner and Coyle, 2008)

Looking at the top line of the model (see figure 1) in its simplest form ( $[A + B] \times C =$  performance power), two individuals could have the same performance power or velocity without having the same physiological capabilities.  $A + B$  could be high and  $C$  low in one athlete, and vice versa in another, however the output (power or velocity) could be identical.

$\dot{V}O_{2max}$  represents the upper limit for this ‘maximal’ oxygen uptake and has been shown to be related to endurance performance (di Prampero, 2003; Helgerud et al., 2007; Joyner, and Coyle, 2008).  $\dot{V}O_{2max}$  is usually achieved during large muscle mass whole body exercise; where the heart generates a high cardiac output, there is high muscle blood flow, and large muscle oxygen extraction (Bassett and Howley, 2000; Dempsey, 1986; Kanstrup and Ekblom, 1984; Mitchell et al., 1958; Rowell, 1986; Saltin and Strange, 1992). However, this ‘physiological descriptor’ may not differentiate between abilities of endurance athletes. For example, values ranging from  $60 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$  to  $85 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$  have been reported in world-class endurance athletes, however even athletes competing at a much lower level might achieve numbers at the lower end of this range for this parameter (Joyner and Coyle, 2008).

Maximal oxygen consumption is dramatically influenced by the cardiovascular capabilities of an individual, specifically cardiac output, which has historically been considered to be the major factor contributing to successful endurance athletic performance (Bassett and Howley, 2000). Despite similarities in maximal heart rates between all ability categories of endurance athletes, as a cohort, elite athletes tend to have greater functional cardiac dimensions compared with lower-category athletes (i.e. they have higher stroke volumes) (Bassett and Howley, 2000). This elevates the ability to deliver key nutrients, to remove byproducts of metabolism and to thermoregulate. In terms of presenting oxygen to the muscle cell, the muscle capillary



network is an important determinant of the rate of oxygen delivery (Bassett and Howley, 1997, 2000; Coyle et al., 1984). Theoretically, greater capillary density will decrease the time required to deliver nutrients (oxygen, glucose, etc.) and reduce the time required to remove by products of metabolism (carbon dioxide, lactate, etc.). It has been suggested that enhanced capillary density might be a differentiating factor in the ability to sustain higher fractions of maximal aerobic capacity for a longer period (Coyle et al., 1984). This is likely to increase the sustainable power output or running speed of an endurance athlete owing to a greater aerobic contribution to the workload. Therefore, the energy required to complete an endurance competition is provided by the oxidative metabolism of carbohydrate and fatty acids. The cardio-respiratory system transports oxygen from the external environment to the internal environment of the muscle cells at a rate which is dictated by the metabolic demands of the working muscles. The oxygen uptake ( $\dot{V}O_2$ ) of an individual increases as the exercise intensity increases until a point is reached above which there is no further increase in oxygen uptake.

The oxygen-carrying capacity of the blood (haemoglobin concentration) is another well-researched parameter that has been shown (through manipulation) to influence  $\dot{V}O_{2\max}$  (Gledhill, 1985; Fabiato and Fabiato, 1978) and, anecdotally, to influence endurance performance (Howley et al., 1995; Noakes, 1988). The energy required to complete an endurance competition is provided by the oxidative metabolism of carbohydrate and fatty acids. The cardio-respiratory system transports oxygen from the external environment to the internal environment of the muscle cells at a rate which is dictated by the metabolic demands of the working muscles. The oxygen uptake ( $\dot{V}O_2$ ) of an individual increases as the exercise intensity increases until a point is reached above which there is no further increase in oxygen uptake (Williams et al., 1984). Ultimately, if the athlete can present more oxygen to the muscle cell, they are less likely to use anaerobic energy sources. The exception to this statement is

where the muscle cell is working at full aerobic potential and is, therefore, unable to consume any further oxygen.

As stated above, one major aspect associated with achieving a high  $\dot{V}O_{2\max}$  is the ability to recruit a large amount of muscle mass. The performance model of Joyner and Coyle indicates that aerobic enzyme activity is important in achieving a high  $\dot{V}O_{2\max}$ . However, if a greater muscle mass is recruited during exercise, more ‘aerobic enzymes’ are also engaged. The provision of energy when the maximal capacity of enzyme function is reached will therefore lead to an increased contribution from anaerobic sources to meet the workload demands. There is some anaerobic contribution before achieving  $\dot{V}O_{2\max}$ . This anaerobic contribution presents (and is most easily monitored) as circulating lactate in the bloodstream. Blood lactate could simply be seen as a stress marker that is indicative of carbohydrate use during exercise (Hopker and Jobson, 2013). Lactate is then implicated in the prediction of performance, because of accelerated carbohydrate use (at a rate which does not correspond to exercise duration) (Hopker and Jobson, 2013; Joyner and Coyle, 2008).

The lactate threshold therefore denotes a key exercise intensity above which the body is under significantly more stress, with accelerated use of carbohydrate stores. Therefore, pushing the lactate threshold rightwards and up to higher exercise intensity before these responses occur is a major consideration for training endurance athletes. The implications of this are that when high-intensity exercise performance is required during an endurance event and these requirements cannot be wholly fulfilled by aerobic mechanisms, production of the required exercise intensity requires supplementation from anaerobic energy sources. Even though lactic acid and lactate are often discussed in the context of fatigue and negative consequences on

athlete performance (Hopker and Jobson, 2013), their production does yield a small amount of important energy.

This small amount of energy is extremely important, because it could make a difference in accelerating away from other athletes, for example in a sprint finish. However, it is not capable of providing the predominant amount of energy for sustained endurance performance due to the accompanying metabolic acidosis. In this regard, endurance events lasting longer than 10 to 15 min are generally performed at a pace that does not evoke  $\dot{V}O_{2max}$ ; for example, 10 km performance occurs at approximately 90–100%  $\dot{V}O_{2max}$ , with a 42 km marathon being run at 75–85%  $\dot{V}O_{2max}$  (Bassett and Howley, 2000; Costill et al., 1973). In a time-trial situation, if the athlete starts too fast and utilizes their anaerobic energy provision at a rate that will exceed their overall capacity before the end of the race, they will have to reduce their work rate significantly below values that might be achievable with perfect ‘pace’ judgement to avoid premature fatigue due to metabolite accumulation and/or glycogen depletion. However, owing to the involvement of oxidative processes in lactate disposal, the oxygen cost of exercise does not necessarily drop, in which case it appears that the athlete is producing less power/velocity for a higher oxygen cost, in other words, they are less efficient. Thus, the lactate threshold is implicated in determining the sustainable/performance  $\dot{V}O_2$ , or exercise intensity that can be maintained during an endurance performance (Bassett and Howley, 2000). Indeed, previous studies have shown good relationships between lactate threshold and endurance performance (Karlsson and Jacobs, 1982; Wassermann et al., 1981), by demonstrating that lactate responds curvilinearly with an increasing exercise work load.

However, whichever the predominant energy system an athlete uses, the ability to convert the available energy into mechanical work is an important aspect related to endurance performance

(Joyner and Coyle, 2008). In this regard, efficiency or economy has been demonstrated to be an important determinant of endurance exercise performance (Hopker et al., 2013; McLaughlin et al., 2010). Indeed, the combination of running economy and  $\dot{V}O_{2\max}$ , defined as the velocity at  $\dot{V}O_{2\max}$  has been found to account for ~94% of the inter-individual variance in running performance over 16.1 km (McLaughlin et al., 2010). Efficiency has been investigated by many researchers from both physiological (Bailey et al., 2009; Bailey et al., 2010; Larson et al., 2010; Larson et al., 2011), and biomechanical perspectives (Hagberg et al., 1981; Marsh and Martin, 1997, and 2000). However, there is still fairly minimal understanding of how this component might change with training (Hopker et al., 2009; Hopker et al., 2010). There is also limited information on the mechanisms that might lead to changes in efficiency and the energy cost of movement (Höchtel et al., 2010). Some researchers have speculated that muscle structure (Coyle et al., 1992; Saltin and Gollnick, 1983) might be implicated. In this regard, a few investigations have reported that the percentage of type I muscle fibers might be a key factor, as they have a greater level contractile efficiency compared to type II muscle fibers (Coyle et al., 1992; Horowitz et al., 1994; Mogensen et al., 2006).

One such study was conducted by Horowitz et al. (1994), who demonstrated that cyclists with a higher percentage of type I fibers also had a significantly higher efficiency, and were able to maintain a 9% higher power output during a one-hour performance trial. However, this study only provides largely correlation based evidence, and intervention studies are required to evaluate the relationship between muscle fiber type, efficiency and cycling performance more thoroughly. Moreover, Coyle et al. (1992) reported a positive relationship between years of endurance training and percentage of type I fibers ( $r=0.75$ ). Again, due to the cross-sectional study design, it was not possible to determine whether the percentage of type I fibers were a response to years of training, or that those cyclists with more type I muscle fibers continued to train and race for longer.

Similar findings have been established during running based exercise. Classical studies from Conley and Crahenbuhl (1980) have demonstrated differences in running economy between elite and experienced athletes of comparable ability and similar  $\dot{V}O_{2\max}$ . Differences in running economy accounted for a significant proportion of the observed variation in 10km running performance between the groups. Therefore, within a group of individuals of similar  $\dot{V}O_{2\max}$ , exercise economy/efficiency is able to explain differences in endurance performance capability.

In addition, to explaining, the cardiovascular/anaerobic model predicts that endurance performance is determined by the capacity of the large size of the athlete's heart to pump unusually large volumes of blood and oxygen to the muscles. This procedure allows the muscles to get higher work rates before they exceed the available oxygen supply, increasing skeletal muscle anaerobiosis (Bassett and Howley, 1997; Noakes, 1988). This model remains the most popular for describing why fatigue develops during exercise; how the body adapts to training; how these adaptations improve performance and, as a result, how effective exercise training programmes should be planned (Noakes, 2000).

As demonstrated in Figure (1), a combination of physiological factors (performance  $\dot{V}O_2$ , performance  $O_2$  deficit, and gross mechanical efficiency) combine to determine the performance power or velocity that an athlete can sustain during an endurance event. By measuring the sustainable performance power or velocity of different durations, it is possible to obtain a picture of the combined influence of these physiological factors. The critical power concept (CP) can be used to provide an estimate of the capacity of these integrated physiological determinants of endurance performance. The concept of CP is based upon a mathematical model of the hyperbolic relationship between power output and tolerable exercise duration. Since the early work of Monod and Scherer (1965), it has been established that the hyperbolic relationship is reflective of physiological responses to endurance exercise

performance, where CP represents the highest sustainable rate of aerobic metabolism (Gaesser and Wilson, 1988), and  $W'$  is deemed to be a non-steady state and attributed to a total amount of anaerobic work that can be performed above the CP (Jones et al., 2010). Indeed, Poole et al. (1988) have demonstrated that steady state ventilation,  $\dot{V}O_2$  and blood acid base balance occurs at exercise intensity below the CP, however steady state does not occur within these parameters during exercise above the CP. At exercise above the CP, the blood lactate concentration and  $\dot{V}O_2$  continue to increase until the  $\dot{V}O_{2max}$  is reached, or the participant stops exercise due to fatigue. Thus, during exercise above the CP, an athlete is on “borrowed time”, as they progressively expend the “finite work capacity” that is available above CP, the  $W'$ . From this perspective, the CP could be defined in terms of the physiological responses to constant-power exercise performed above it (i.e. non-steady state), and below it (i.e. steady state).

Even though the CP and  $W'$  are reflective of physiological phenomena, their measurement is fundamentally based on performance i.e. the mechanical work done, or distance covered over time. The most common procedure for assessing the parameters of the power-duration relationship in the laboratory is by having participants perform at least three constant power exercise tests to exhaustion. The power outputs chosen are typically between 75% and 105% of the maximal power output achieved in a ramp test, and are intended to result in times-to-exhaustion of between 2 and 15 minutes (Hill, 1993). The resulting data can be analysed using both linear and nonlinear regression.

Monod and Scherrer (1965) presented the critical power using a nonlinear hyperbolic model, defined using the equation:

$$t = W'/P-CP$$

In this equation,  $t$  = the tolerable duration (time to fatigue),  $W'$  = a finite amount of energy reserve which is expressed in kJ,  $P$  = power output. CP is represented by the power asymptote (Figure 2, panel A). As outlined above, this model suggests that when CP is exceeded, it leads to exhaustion in a predictable duration defined by the finite amount of energy reserve i.e. the  $W'$ . Monod and Scherrer's hyperbolic model can be translated into a linear relationship which expresses the total work performed in relation to the tolerable duration of this work. This linear work-time relationship is expressed as:

$$P \cdot t = W' + (CP \cdot t)$$

In this equation  $P \cdot t$  = the total amount of work performed,  $t$  = time to exhaustion. CP is denoted by the slope of the relationship and  $W'$  is represented by the y-intercept (Figure 2, panel B).

Moritani et al. (1981) later added the linear power-inverse time model which is expressed as:

$$P = CP + W' + 1/t$$

Here, CP is represented by the y-intercept with  $W'$  being the slope of the relationship (Figure 2, panel C).

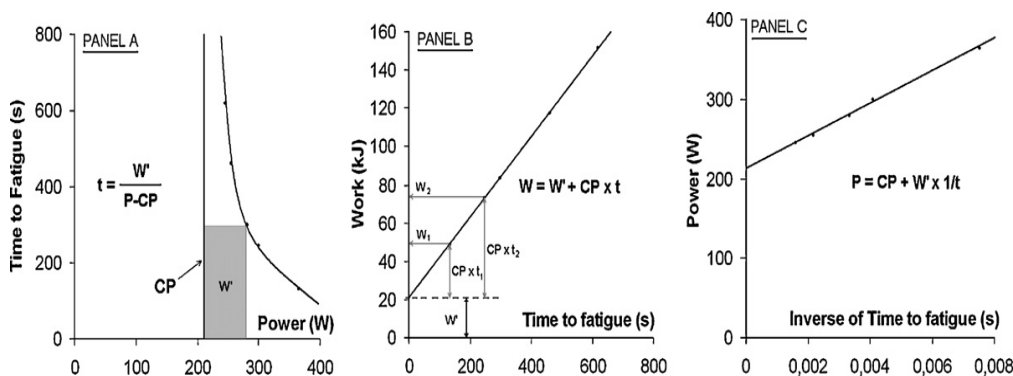


Figure 2: Schematic representation of critical power models. (hyperbolic time-power relationship– Panel A; linear work-time relationship – Panel B; linear power-1/t relationship – Panel C). From Deckerle et al. (2006).

However, a number of criticisms have been levelled at the CP model, as it implies CP to be sustainable for an indefinite period of time. In this regard, the models assume that CP is purely aerobic in nature and is rate, but not capacity limited. Conversely, they assume that  $W'$  is anaerobic in nature and is capacity, but not rate limited. Moreover, the models assume that at the onset of exercise when exercising at CP intensity the energy provision is solely supplied by aerobic metabolism (Morton, 2006). The models also assume that exhaustion occurs upon depletion of  $W'$ , and that exercise efficiency remains constant across all power and time domains. Finally, the models do not consider psychological aspects, which will affect constant work rate exercise, and eventually, require the athlete to terminate exercise performance.

In addition, the physiological assessments of endurance performance as mentioned previously such as ( $\dot{V}O_{2max}$ , Lactate threshold, and exercise economy) are the key variables that affect race pace. Once  $\dot{V}O_{2max}$  became practically obvious that elite runners had high values of  $\dot{V}O_{2max}$  it also became clear that for events lasting beyond 10 or 15 min, most or all of the competition was performed at an average pace that did not evoke  $\dot{V}O_{2max}$ , with much of the 42 km marathon run at roughly 75–85%  $\dot{V}O_{2max}$ , while 10 km was performed at 90–100%  $\dot{V}O_{2max}$  and 5 km at near to  $\dot{V}O_{2max}$  (Costill et al., 1973; Bassett and Howley, 2000). Next, as power output on a cycle ergometer or running speed increases in untrained participants, there is normally no sustained rise in blood lactate concentration until about 60% of  $\dot{V}O_{2max}$  is reached. In trained participants this value can be 75–90% of  $\dot{V}O_{2max}$ .

In this context, there are no inclusive longitudinal results on groups of endurance athletes followed over many years to determine the trainability of running economy or cycling



efficiency. Nevertheless, there are at least two examples reporting that running economy can be improved over several years of training in trained athletes (Conley et al., 1984; Jones, 2006). In fact, the recent world record holder for the women's marathon showed a significant 14% improvement in running economy over the period of five years of training (Jones, 2006). Moreover, cycling efficiency was observed to increase 8% over the period of seven years in a trained endurance cyclist (Coyle, 2005). In general, these case reports suggest that running economy and muscular efficiency might indeed improve with constant endurance training at a rate of about 1–3% per year (Green et al., 1984).

## **II. Perception of effort**

### **1. Definition and Evidence**

When performing any activities of daily living, individuals experience effort. Effort is defined as “strenuous physical or mental exertion” (Oxford Dictionary), and is known to intensify the harder a person tries (Preston and Wegner, 2009). Thus, effort can be perceived during both physical and mental tasks, and individuals can use these feelings to judge the task difficulty. Hence, both feeling of task difficulty and effort are strongly related. In turn, perception is defined as “the conscious experience of sensation” (Oxford Dictionary) and regarded as being the result of central processing (the brain’s interpretation) of sensory signals (Gardner and Martin, 2000). As perception results from central processing, perception varies between individuals, and is consequently highly subjective (Weiten, 2010). The perception of effort is therefore proposed to be a cognitive feeling of work associated with voluntary actions, and is crucial for the judgement of personal actions (Morsella et al., 2009; Preston and Wegner, 2009).

The perception of effort was first translated into an exercise context by the studies of Gunnar Borg (1962). Borg (1962), defined the perception of effort as “*the feeling of how heavy, strenuous and laborious exercise is*”, and attributed its’ origins to “*the sensation from the*

*organs of circulation and respiration, from the muscles, the skin, the joints and force.”* More recently the idea of fatigue and/or discomfort has been added to the definition of perception of effort as follows: *“the subjective intensity of effort, strain, discomfort, and/or fatigue that is experienced during physical exercise”* (Noble and Robertson, 1996; Utter et al., 2007). However, it has been reported by several studies that humans are able to disconnect their perceived exertion from other exercise-related feelings, such as force (Jones, 1995), pain (O'Connor and Cook, 1999), discomfort (Christian et al., 2014), or fatigue (i.e. effort can be observed with fatigue: de Morree and Marcora, 2010; de Morree et al., 2012). Indeed, using single limb exercise models, research studies have established that the perception of effort and perception of force are two perceptions closely related to another, but are not the same (Taylor, 2013).

Indeed, in neurophysiology, effort is usually related to the conscious perception of the central motor command (i.e. activity of premotor and motor areas of the brain associated with voluntary muscle contractions) sent to the working muscles (de Morree et al., 2012), while perception of force corresponds to the perception arising from the neuronal process of the corollary discharge of the central motor command, and the afferent feedback from the working muscles (Taylor, 2013). As a result, it is possible for experienced participants to discriminate between the perceptions of effort and force (Jones, 1995; Jones and Hunter, 1983). Specifically, effort indicates relates to the difficulty of driving the limb during exercise (de Morree, et al., 2014; de Morree et al., 2012), whereas force is related to the sensations of muscle tension experienced during muscle contraction (Jones and Hunter, 1983).

The International Association for the Study of Pain (IASP) has defined pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or

described in terms of such damage. This definition suggests that pain is an unpleasant subjective experience that has an emotional element. Resultantly, this distinguishes pain from effort, necessitating a different psychophysiological scale to be used from that which assesses the perception of effort. The neurophysiology of muscle pain is well established and involves Group III-IV nociceptors which send peripheral information to the central nervous system. These nociceptors are stimulated by intense skeletal muscle activity leading to increases in pain producing substances such as bradykinin, potassium, serotonin, histamine, hydrogen ions and prostaglandins (O'Connor and Cook, 1999). Increased levels of pain have been shown to reduce voluntary muscle contraction (Parfitt et al., 2006), requiring more muscles to be recruited to meet the required exercise demands and in turn indirectly resulting in an increased perception of effort.

O'Connor and Cook (1999) have established that individuals can dissociate between the perception of effort and muscle pain. Specifically, they used two different psychophysiological scales to investigate the perception of effort and muscle pain, and established that participants were able to maintain moderate intensity muscle pain for ~15 min, whilst still being able to separately rate their perception of effort. The ability of individuals to differentiate between the perceptions of effort and pain has also been investigated by other research groups, with participants being asked to rate their effort and pain separately during arm cycling (e.g. Gros Lambert et al., 2006), leg cycling (e.g. Astokorki and Mauger, 2016), and isolated (e.g. Pageaux et al., 2015) exercise, or by exercising at a fixed perception of effort while reporting their perception of pain (e.g. Astokorki and Mauger, 2016). Linked to the perception of pain, and included within Noble and Robertson's revised definition of the perception of effort (Noble and Robertson, 1996; Utter et al., 2007), is discomfort. In this regard, the same criticisms

levelled at the inclusion of pain in the definition of the perception of effort could also be levelled at the inclusion of discomfort. Indeed, a study conducted by Christian et al. (2014) demonstrated that individuals could independently rate their perception of discomfort when cycling in normoxia and hypoxia at a constant effort.

In summary, using a definition that does not include other exercise-related sensations could help researchers to obtain a better insight into the primary mechanisms generating perception of effort. It is logical to propose that the perception of effort can be rated individually from other exercise-related feelings, and that different exercise-related feelings have their own neurophysiological underpinnings (Marcora, 2009). Therefore, perceived exertion could be defined as the sensation of “how hard, heavy and strenuous exercise is” (Marcora, 2009; Marcora, 2010). This definition is consistent with the verbal descriptors initially proposed by Borg within his RPE scale (Borg, 1998), but does not include other exercise-related sensations (e.g. pain or discomfort). This will be discussed in more detail in the section below.

## **2. Measuring perception of effort**

Rating of perceived exertion (RPE) scales are commonly used by researchers to investigate the magnitude of the psycho-physiologically determined perception of effort. The most common scales are the Borg 6-20 scale (Borg, 1970) and the category-ratio (CR10) scale (Borg, 1982). Both scales are shown in Figure 3. Borg’s 6-20 RPE scale is a 15-point scale that was designed to reflect a linear increase in heart rate in response to an incremental exercise test (Borg, 1998). As such, the 6-20 scale is constructed in such a way that it reflects the heart rate, divided by 10. The shorter CR10 scale increases sharply in line with increasing the speed of activity (Borg, 1982). In addition, at the end of the scale a black dot is used to afford the potential that the

individual might rate their sensation to be higher than 10, meaning that it avoids a “ceiling” type effect, often experienced with these types of perceptual scales. The 6-20 and CR10 scales have both been shown to be valid and reliable methods to assess the perception of effort (Borg, 1998). However, it should be acknowledged that the scales are only valid and reliable if appropriate instructions and information are provided to the participant about how to rate their perception of effort. A familiarization session is often used for this purpose so that the participant can calibrate their rating of perceived effort to the effort required during the given task (Noble and Robertson, 1996; Eston et al., 2015). A familiarization session will therefore help to avoid any under- or over-estimation of the perception of effort, and also ensure that participants are able to differentiate effort from other exercise-related sensations.

Special consideration should be paid to the instructions provided to the subjects: (1) Written instructions including the definition of effort should be provided to the subjects before each laboratory testing session and the participant should have the opportunity to ask questions. (2) As effort differs from pain, force and discomfort, it has to be specified that participants must not include other sensations (e.g. discomfort) within their rating of effort. (3) As the effort experienced during isolated exercise and whole-body differs (e.g. whole-body exercise compared to lower cardiorespiratory responses to isolated exercise), exercise detailed descriptions on how to rate perceived exertion should be provided. The description “How hard is it for you to drive your arm or leg?” for isolated exercise and “How heavy is your breathing and how hard is it for you to drive your arms and legs?” for whole-body exercise have been reported to be sensitive to psychological (e.g. Pageaux et al., 2013; Marcora, et al., 2015) and physiological (e.g. de Morree and Marcora, 2013; de Morree et al., 2012) manipulations of perceived exertion. (4) Participants should be asked to first read the verbal expressions of the scale, and then to rate the corresponding number according to their sense of effort. (5) To

provide points of information on how to rate perceived exertion, exercise-anchoring or memory-anchoring should be performed. Exercise-anchoring is based on an exercise performed (e.g. “maximal exertion corresponds to the effort you experienced while you were performing a maximal voluntary contraction” for isolated exercise; “maximal exertion corresponds to the effort you experienced at exhaustion of the incremental test” for whole-body exercise) and memory-anchoring is based on participants’ memory (e.g. “maximal exertion corresponds to the highest effort you have ever experienced”). (6) If the sensation of effort experienced during the exercise is above the feeling linked to the anchoring performed for “maximal exertion”, the participant should be allowed to rate a value above “maximal exertion” to avoid a ceiling effect. (7) As perceived exertion is associated with the feeling experienced during exercise, perception of effort should be rated during the exercise. If impossible, the participants can be asked to provide their perceived exertion after completion of the exercise task, and it must be described to the participants that their RPE should refer to the feeling experienced during exercise, and not that experience post exercise (i.e. at rest).

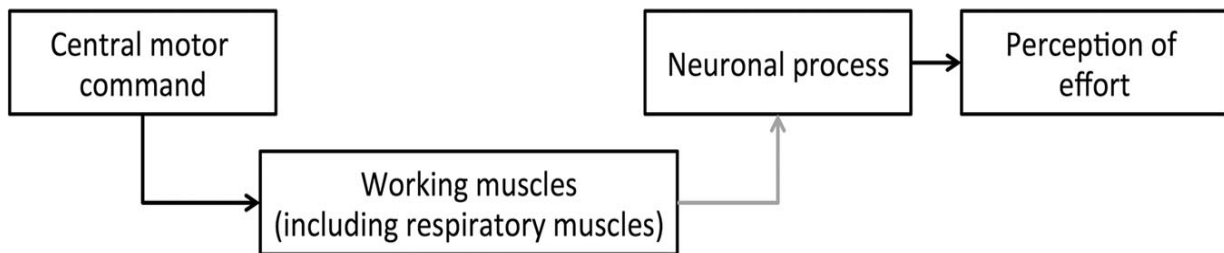
rating	description	rating	description
0	NOTHING AT ALL	6	NO EXERTION AT ALL
0.5	VERY, VERY LIGHT	7	EXTREMELY LIGHT
1	VERY LIGHT	8	
2	FAIRLY LIGHT	9	VERY LIGHT
3	MODERATE	10	
4	SOMEWHAT HARD	11	LIGHT
5	HARD	12	
6		13	SOMEWHAT HARD
7	VERY HARD	14	
8		15	HARD (HEAVY)
9		16	
10	VERY VERY HARD (MAXIMAL)	17	VERY HARD
		18	
		19	EXTREMELY HARD
		20	MAXIMAL EXERTION

Figure 3: CR10 and 6-20 Borg scales. (Borg, 1998)

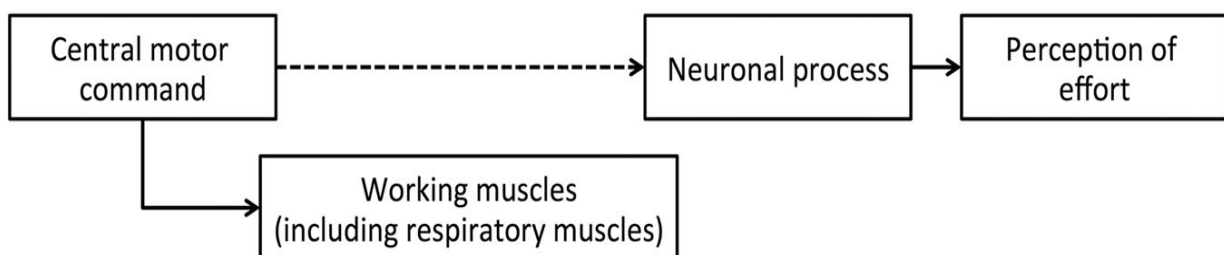
### **3. Neurophysiology of perceived exertion**

The neurophysiology of the perceived exertion is not well understood. To date, only a handful of studies have been conducted investigating the neurophysiology of perceived exertion. Human perception has been suggested to share three important phases: i) a stimulus ii) the central processing of this stimulus, leading to iii) the perception or conscious experience of the sensation (Gardner and Martin, 2000). Based upon these three common steps, two theoretical models have been developed to explain mechanisms associated with the perception of effort: the afferent feedback model (Figure 4a: Amann and Light, 2014; Amann et al., 2013; Noble and Robertson, 1996) and the corollary discharge model (Figure 4b: Marcora, 2009; McCloskey et al., 1974). More recently a combined model has also been proposed, which includes aspects from both afferent feedback and corollary discharge models (Figure 4c: Amann et al., 2010). Independent of the theoretical modelling of the perception of effort, several experiments have suggested that distinct brain areas are involved in the generation of the perception of effort, which include; the anterior cingulate cortex, insular cortex, pre-supplementary motor areas, supplementary motor areas, and possibly the thalamus (de Morree et al., 2012; Williamson et al., 2001; 2002).

**a - AFFERENT FEEDBACK MODEL**



**b - COROLLARY DISCHARGE MODEL**



**c - COMBINED MODEL**

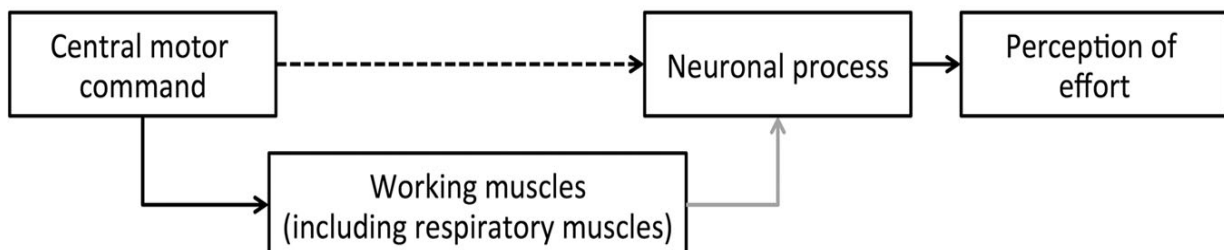


Figure 4: Afferent feedback (panel a), corollary discharge (panel b) and combined (panel c) models of perceived exertion. The grey line represents afferent feedback and the dotted line the corollary discharge associated with the central motor command (Pageaux, 2016).



### **Afferent feedback model**

Proponents of the afferent feedback model (e.g. Amann et al., 2013), propose a link between increased perception of effort and elevated blood lactate accumulation and muscle milieu metabolites concentration. As muscle afferents consist of type III-IV muscle fibres (i.e. free nerve terminations stimulated by contraction-induced chemical and mechanical stimuli; Rowell and O'Leary, 1990), these free nerve endings may be a possible candidate for being involved in the generation of the perception of effort. This afferent feedback model has anatomical support as group III-IV muscle afferents are recognised to have central projections to numerous spinal and supraspinal areas, including the sensory cortex (Craig, 2002). To examine whether muscle afferents have a direct influence on the generation of perceived exertion, researchers have attempted to disconnect afferent feedback from the central motor command via the use of epidural anaesthesia (Amann et al., 2010). However, as previously discussed, unfortunately drawing conclusions from the study of Amann et al. (2010) is problematic, as the sensations of discomfort and fatigue were included in the definition of the perception of effort provided to participants. Moreover, other epidural anaesthesia studies provide evidence against the afferent feedback model. For example, results of these studies demonstrate that despite a significant reduction in muscle afferent feedback from the working muscles, perception of effort was unchanged or higher with spinal blockade during cycling (Kjaer et al., 1999; Smith et al., 2003), and isometric contractions (Mitchell et al., 1989). Furthermore, the stimulation of group III-IV afferent stimulation following the injection of physiological concentrations of metabolites into skeletal muscle has been shown not to generate perception of effort at rest, i.e. without central motor command (Pollak et al., 2014).

### **Corollary discharge model**

The corollary discharge model suggests that perceived exertion is generated by a combination of the corollary discharge (the internal signals which arise from centrifugal motor command and that influence perception; McCloskey, 1981), and the central motor command (i.e. activity of motor and premotor areas of the brain associated with voluntary muscle contractions; de Morree et al., 2012). Corollary discharges are believed to have perceptual influences in two different ways; firstly, they are thought to alter the processing of incoming sensory information; secondly, they may be used as the only stimulus to produce some specific sensations i.e. perceived exertion (McCloskey, 1981).

Research supporting this model has demonstrated that changes in central motor command required to perform a task are associated with similar changes in the perception of effort (McCloskey et al., 1983). Moreover, in the presence of muscle fatigue, perceived exertion has been shown to increase, despite the absence of any exercise-induced metabolites in the muscle (i.e. those known to stimulate group III-IV muscle afferents), however, the corollary discharge model does not exclude an indirect role of afferent feedback on perception of effort during its role in motor control (i.e. direct adjustment of the central motor command). The corollary discharge model only reported that afferent feedback is not the sensory signal generating the feeling of effort (i.e. stimulation of muscle afferents does not generate the feeling of effort, but other sensations such as discomfort, muscle pain or muscle tension). Moreover, inhibition of motoneurons at a spinal or supraspinal level induced by afferent feedback can potentially result in an increase in central motor command to ensure the same submaximal force production. This inhibition induces an increase in central motor command (Marcora, 2009; Pageaux, 2016). However, the corollary discharge model has a few complexities it needs to be pointed out that

conscious procedures can generate positive signals to compete with corollary signals. In other words, athletes can resist this negative effect through conscious motivation (e.g., self-talk; see Blanchfield et al., 2014). It is important to note that sensory information and awareness can also have a negative effect on voluntary control; for example, if the exerciser engages with or listens to unpleasant music or negative self-talk while running. Those negative effects are theorized to decrease the motivational state and increase the perception of effort. In addition, brain responses to high levels of anxiety are also responsible for increasing perception of effort and decreasing voluntary control with a negative effect on performance (Parry et al., 2011). Consequently, it is assumed that a greater amount of corollary discharge might be sent to sensory regions of the brain, reducing efferent signals, and muscle activity.

Instead, during fatigued conditions, research has shown that increased perception of effort is related to increased motor related cortical potentials, which are recognised as an indirect index of central motor command (de Morree et al., 2012). Furthermore, the results of a study undertaken by de Morree et al. (2014) reported that perceived exertion and the motor related cortical potentials are both decreased following caffeine ingestion. The study provides neurophysiological evidence that alerts in activity of premotor and motor areas of the cortex during endurance exercise can occur even without major changes in muscle activation and force output. These cortical changes happen in the same trend as changes in perception of effort caused by caffeine and time-on-task. This cross-situational covariation between MRCP amplitude and RPE during muscle contraction provides correlative evidence in favor of the theory that perception of effort arises from neurocognitive processing of corollary discharges from premotor and motor areas of the cortex. The fact that muscle activation during endurance exercise was not influenced by caffeine and time-on-task suggests that corollary discharges which are limited within the brain (central corollary discharges) contribute to perception of effort. Caffeine appears to reduce perception of effort through a reduction in the activity of

premotor and motor areas of the cortex that are necessary to produce a certain force, and time-on-task seems to have the opposite effect. (de Morree et al., 2014).

In addition, perception of physical effort is moderately unaffected by the suppression of sensory afferences, representing that this function relies mainly on the processing of the central motor command. Neural signals in the supplementary motor area associate with the rating of effort, suggesting that the motor signal involved in perception of effort could originate from this area. Therefore, Zenon et al. (2015) have tested by disrupting neural activity in the supplementary motor area in the primary motor cortex, or in a control site by means of continuous theta-burst transcranial magnetic stimulation, instead measuring perception of effort during grip forces of different intensities. They found that disruption of supplementary motor area activity, but not of the primary motor cortex, led to a consistent decrease in the perception of effort, whatever the measure used to assess it. Consequently, Zenon et al. (2015) have modeled perception of effort in a structural equation model and found that only supplementary motor area activity disruption led to a significant alteration of perception of effort. These findings present that perception of effort relies on the processing of a signal originating from motor-related neural circuits upstream of the supplementary motor area and that the supplementary motor area is a key node of this network (Zenon et al., 2015).

### **C. Combined model.**

Both models mentioned above have their limitations, thus, the afferent feedback model is a complicated framework, on the other hand, the corollary discharge model is simple in terms of generation of perception of effort, therefore it is necessary that scientists should take further action and investigate the combined model. However, it is well known that corollary discharges do not only generate specific sensations, but also modify the processing of incoming sensory

information. The combined model suggests that perceived exertion results from the integration of both the corollary discharge and afferent feedback linked with the central motor command. Indeed studies which have investigated the role of afferent feedback on fatigue during exercise performance have suggested that there is the potential for increased central motor command to maintain the submaximal force production following a spinal blockade via epidural anaesthesia (Kjaer et al., 1999; Amann et al., 2010). Therefore, the inhibition of motor neurons at a spinal or supraspinal level is likely to increase central motor command, leading to an increase in the perception of effort.

However, there is a paucity of evidence supporting this model, and where evidence is available it appears to be contradictory. Kjaer et al. (1999) found no reduction in the perception of effort during cycling with reduced afferent feedback. Conversely, Amann and colleagues (2010) demonstrated that in spite of reduced afferent feedback from the locomotor muscles, RPE was lower during submaximal exercise following a spinal blockade compared to a placebo condition performed with an intact somatosensory feedback system.

Bergstrom et al. (2015) conducted an experiment on eight runners and their critical heart rate was determined from a series of four exhaustive bouts. The study results indicated that muscle afferents may have afforded feedback from metabolic and mechanical stimuli that contributed to the perceptual responses. In addition, only RPE consistently indicated exhaustion and the study outcomes supported its use to monitor exercise performed at a constant HR.

In spite of several studies suggesting the validity of the combined model (e.g. Amann et al., 2010; Bergstrom et al., 2015), no study has specifically aimed to test it.

### **III. Endurance performance and the perception of effort**

Earlier on in this chapter it was established that endurance performance can be determined by well-established physiological parameters including,  $\dot{V}O_2\text{max}$ , lactate threshold, and exercise economy. However, there is much less known about psychological determinants of endurance performance, and the link between physiological and psychological factors. There are two main models that have been proposed to explain the link between physiological and psychological factors affecting endurance exercise performance.

#### **The Central Governor Model**

The central governor model (CGM, Figure 5) first proposed by Noakes et al. (2001) describes the role of the brain as a regulator of endurance exercise performance. This CGM model develops an earlier theory of 'teleoanticipation', where endurance performance is regulated by peripheral and central manipulations of the perception of effort (Ulmer, 1996). The teleoanticipation theory reported that in order to avoid a critical homeostatic disturbance, a feedback control system should exist, that serves to regulate endurance exercise performance with a known end point. Thus, the model suggests that the brain continuously monitors feedback from the body to subconsciously modify self-paced exercise (Tucker, 2008). In this regard, the CGM proposes that the central nervous system controls exercise in order to avoid any catastrophic physiological failure (e.g. heart ischemia) and to sustain body homeostasis (Noakes, 2011). The CGM further suggests that maximal fatigue is never reached, and a physiological reserve exists that cannot be used by individuals. As suggested by the inhibitory afferent feedback model (Amann et al., 2011) outlined above, the CGM suggests that afferent feedback from the working muscles (and other organs such as the lungs and heart) serves to downregulate muscle recruitment when exercising individuals are at, or close to their

physiological reserve; hence fatigue occurs. This de-recruitment of skeletal muscle fibres is proposed by CGM advocates to avoid individuals reaching a critical level of fatigue leading to disastrous physiological failure (Noakes, 2011).

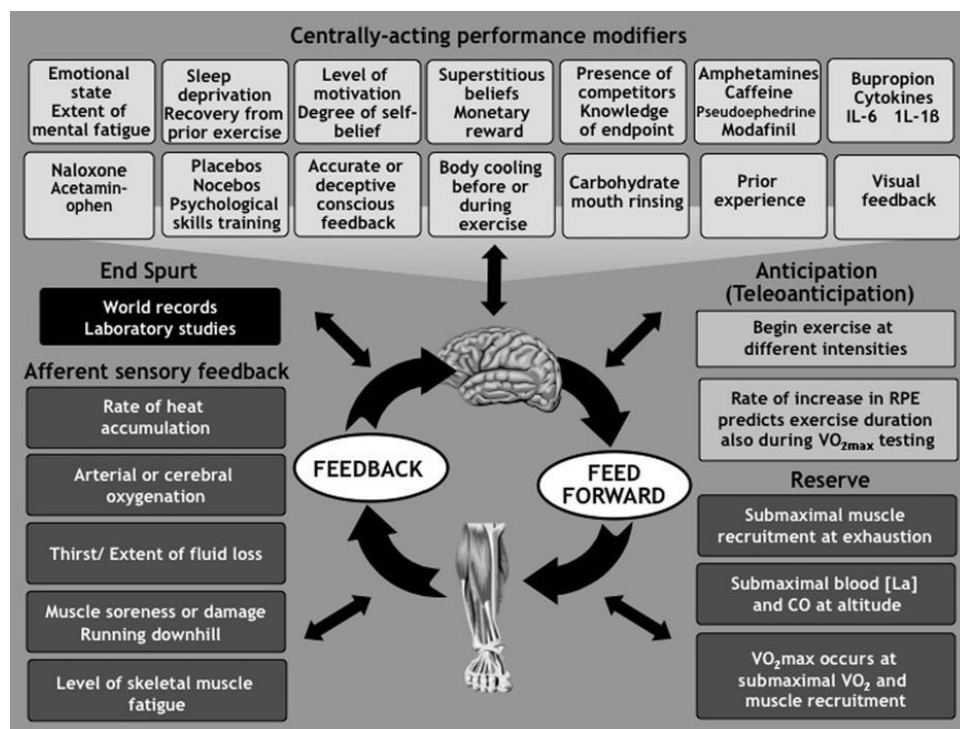


Figure 5: Best representation of the central governor model (Taken from Noakes, 2012)

During time trial performance, the CGM suggests that the work rate of self-paced exercise is adjusted via sensory afferent feedback, which is understood consciously as the perception of fatigue and/or the perception of effort (Noakes et al., 2004; Noakes, 2012; St Clair Gibson et al., 2006). The work rate associated with self-paced exercise is therefore continually changed through this sensory afferent feedback, which forms as a feed forward regulator of skeletal muscle fibre recruitment (Noakes, 2012). In this regard the CGM integrates both conscious and unconscious information (related to the perception of effort) as the final element that regulates performance and fatigue (Noakes, 2012). Nevertheless, the CGM has been criticised (Marcora, 2008; Inzlicht and Marcora, 2016), as it suggests that the central governor is a subconscious system that could be overridden by consciousness as otherwise there would be a threat to

homeostasis (Noakes et al., 2005). However, recent work suggests that there is evidence of a conscious override of the subconscious system, with motivation being a key determinant of physical exertion (McCormick et al., 2015).

### **The Anticipatory-RPE regulation model**

This model occurs during self-paced exercise, the exercise work rate is regulated by the brain based on the integration of numerous signals from many physiological systems. It has been suggested that the brain regulates the degree of muscle activation and therefore exercise intensity specially to prevent harmful physiological disturbances. It is currently proposed that the rating of perceived exertion (RPE) is generated as a result of several afferent signals during exercise and serves as a mediator of any following alterations in skeletal muscle activation levels and exercise intensity. A conceptual model for how the RPE mediates feed-forward and anticipatory regulation of exercise performance is suggested, and this model is applied to previously explained research investigations of exercise in several conditions, including hypoxia, heat and reduced energy substrate availability. Finally, the purpose of this model concerning the current novel investigation is that change performance and pacing strategies are described utilising an RPE clamp design, central nervous system drugs and the provision of inaccurate distance or duration feedback to exercising athletes.

This model combines anticipatory/feed-forward as well as a feedback mechanism, using an expectation of exercise length to set an original work rate and to generate what has been described as a subconscious “template” for the level of increase in the RPE. During exercise, afferent feedback from several physiological systems is responsible for the conscious RPE’s generation, which is constantly matched with the subconscious template by means of adjustments in power output. This subjective rating is biologically associated, allowing pacing



strategy to be adjusted to prevent catastrophic alterations in the monitored physiological variables (homeostats) (Tucker, 2009).

### **The afferent feedback model**

The role of group III/IV muscle afferents on the peripheral fatigue's development is demonstrated through their contribution to the hemodynamic, cardiovascular, and ventilatory adjustments happening during exercise. Increases in these parameters rising with the onset of exercise are, next to central command (Waldrop et al., 1996), mostly determined by neural feedback from the working muscle and assure increased blood flow and O<sub>2</sub> delivery to the working muscle (Asmussen et al., 1943; Coote et al., 1971; Hollander et al., 1975; Kao, 1963; McCloskey and Mitchell, 1972; Tibes, 1977). Both of these variables show key components in the rate of peripheral fatigue's development during exercise (Barclay, 1986; Fulco et al., 1996; Katayama et al., 2007). This is especially the case with decreases in blood flow/O<sub>2</sub> delivery that intensify this rate, while increases in blood flow/O<sub>2</sub> delivery attenuate this rate.

The study done by Amann et al. (2011) designed to examine the role of group III/IV locomotor muscle afferents in the development of peripheral locomotor muscle fatigue, intrathecal fentanyl has been used to attenuate sensory feedback from the lower limb during prolonged-load leg cycling (Amann et al., 2011). Peripheral fatigue was quantified through the pre-exercise to post-exercise decrease in quadriceps twitch force evoked through supramaximal femoral nerve stimulation. Previous studies utilizing an identical pharmacological approach to temporarily block group III/IV muscle afferents have reported that exercise, in the absence of sensory feedback, is considered to be substantial hypoventilation and an attenuated exercise pressor response, including decreased peripheral (leg blood flow) and central (i.e. cardiac output) and hemodynamic responses during exercise (Amann et al., 2010; Amann et al., 2011;

Gagnon et al., 2012). Thus, when prolonged-load leg cycling is performed in the absence of locomotor muscle afferent feedback, leg blood flow and O<sub>2</sub> delivery are clearly attenuated compared to the same exercise performed with an entire neural feedback mechanism. Given the significant role of blood flow/O<sub>2</sub> delivery in the development of fatigue (Amann and Calbet, 2008), the rate of peripheral fatigue's accumulation is up to 60% faster during exercise with impaired vs intact group III/IV muscle afferent feedback (Amann et al., 2011, Sidhu et al., 2014a, Sidhu et al., 2014b). Occupied together, by facilitating ventilatory and circulatory responses, group III/IV muscle afferent feedback ensures sufficient muscle blood flow/O<sub>2</sub> delivery during exercise and thus prevents premature fatigue at the level of the locomotor muscle. This neural feedback process plays a significant role in optimizing fatigue resistance during physical activities in healthy humans.

The arterial baroreflex has been suggested to attenuate the central effects of group III/IV muscle afferents on the exercise pressor response through their interaction in the central tractus solitarii (Kim et al., 2005; Sheriff et al., 1990; Waldrop and Mitchell, 1985). In other words, the group muscle III/IV-mediated pressor responses to exercise have been reported to be larger in the absence of the arterial baroreflex. Based on previous experimental studies in endurance exercising, athletes showed that attenuated feedback from group III/IV locomotor muscle afferents caused a reduction in muscle blood flow/O<sub>2</sub> delivery and an accelerated level of peripheral fatigue (Amann et al., 2010, Amann et al., 2014), it can be speculated that arterial baroreflex buffering of group III/IV-mediated muscle reflexes exacerbates the peripheral fatigue's development during endurance exercise. Nevertheless, Waldrop and Mitchell have presented that the arterial baroreflex modulates the pressor response without altering muscle blood flow during induced muscular contraction in anesthetized cats (Waldrop and Mitchell, 1985). Even though unknown in humans, if baroreceptor buffering of muscle afferents does not

limit blood flow to the working muscle, it could be argued that it does not affect the peripheral fatigue's development.

It is important to report that the role of group III/IV muscle afferents in the development of peripheral fatigue may be different in patients with heart failure (Amann et al., 2014) which are characterized by muscle reflex abnormalities (Garry, 2011; Piepoli et al., 2008) with inflated afferent feedback as the probable underlying mechanism (Middlekauff and Sinoway, 2007; Notarius et al., 2001; Piepoli and Coats, 2007). While feedback from these neurons still facilitates central hemodynamics in this population (Amann et al., 2014), it has been reported to account for the extreme hyperventilatory response (Olson et al., 2014) and inflated sympathoexcitation (Amann et al., 2014; Notarius et al., 2001) during physical activity in these patients. To our knowledge, there has been no recent investigation available on the effects of other diseases characterized by abnormal neural feedback from group III/IV muscle afferents (e.g., hypertension) on the development of peripheral and central fatigue during exercise.

### **The psychobiological model**

The psychobiological model has been observed to provide a valid explanation of effects of both physiological (Marcora et al., 2008) and psychological (Marcora et al., 2009; Pageaux et al., 2013) manipulations on endurance exercise performance during time to exhaustion. Recently, the psychobiological model has been used to explain changes in endurance exercise performance following manipulations which changed both physiological (muscle fatigue; de Morree and Marcora, 2013) and psychological (mental fatigue; Pageaux et al., 2014) parameters. The basic principles of the psychobiological model were based on goal-directed

behaviour, rather than just the output of a biological appliance that converts chemical energy into mechanical energy.

The theoretical consequence of this approach is that endurance performance should be fully describable by psychology, the scientific investigation of the human mind and behaviour (The British Psychological Society, 2010). In addition, the psychobiological model proposes that consciousness is an emergent property of the brain (Searle, 1998). Thus, the primary function of consciousness is to constrain and direct skeletal muscle output in order to produce adaptive behaviour (Morsella et al., 2015; Morsella et al., 2009; Poehlman, et al., 2012). Moreover, to highlight the framework of both existing theories which the central governor model, which attributes negligible importance to the role of psychological aspects as an exercise endurance performance modulator, or from recent formulations of the central governor model that integrated ad hoc psychological explanations as the model developed (Noakes, 2000). However, the important role of the psychological factor in modulating performance has been established since the 60s (Ikai and Steinhaus, 1961). On the other hand, the psychobiological model has only been discovered recently, with a new framework model having been proposed by Marcora and colleagues (Marcora, 2007; Marcora et al., 2009). Therefore, the Psychobiological model gives more attention to motivational and perceptual factors, and their respective effect on the conscious procedure of decision-making and behavioral regulation. Indeed, this model for identifying exhaustion depends on the psychological exercise (in) tolerance, whereas the central governor model identifying these phenomena depends upon physiological inability (i.e., physiological limit) or subconscious and anticipatory procedure (i.e., not subject to willingness) (Smirmaul et al., 2013).

The emphasis on conscious processes of action control is one of the main differences between the psychobiological model and the central governor model developed by Noakes and

colleagues (Noakes, 1997, 2012; Noakes et al., 2005). This theoretical model postulates that recruitment of the locomotor muscles is regulated by the central governor intelligent system subconsciously in the brain, to ensure that endurance exercise performance terminates before damage to the muscles fibres is induced (Noakes, 1997, 2012) and it thus avoids “conscious override” that might damage the athletes in high motivation conditions (Noakes et al., 2005). Since 2006, Marcora has argued that the central governor model is imperfect (Inzlicht and Marcora, 2016; Marcora, 2007, 2008, 2009), and that an applicable theoretical explanation of how the brain regulates endurance exercise can be provided without a central governor (Marcora, 2007, 2008, 2009 and 2010) as follows.

Indeed, the psychobiological model (Marcora, 2010) is an effort-based decision-making model that depends upon motivational intensity theory (Brehm and Self., 1989), and assumes that the conscious regulation of pace is determined mainly by five different motivational / cognitive factors:

- 1) Perception of effort
- 2) Potential motivation
- 3) Knowledge of the distance/time to cover
- 4) Knowledge of the distance/time remaining
- 5) Previous experience/memory of perception of effort during exercise of varying intensity and duration

Potential motivation indicates the maximum effort a person is prepared to use to achieve a goal, and can be easily affected by external factors (e.g. higher motivation during activities

with participants than during laboratory testing). Knowledge of the distance/time to cover, knowledge of the distance/time remaining, and previous experience/memory of perception of effort during exercise of varying intensity and duration are self-explanatory and can clarify why knowledge of the end-point is important for endurance performance (Marcora, 2008), or why individuals begin races of different distances/durations at different exercise paces (Joseph et al., 2008). Perception of effort (factor 1) is the key factor of this model. Indeed, according to this model, the sensible regulation of pace is mainly determined by the effort perceived by the participants.

Thus, when perceived exertion is increased by mental fatigue (Pageaux et al., 2014) or muscle fatigue (de Morree and Marcora, 2013), or reduced (same perceived exertion for a higher power output) by pharmacological manipulation (Watson et al., 2005), the individual will consciously change their pace to account for the positive/negative influence of the experimental manipulation on perceived exertion. In turn, this will lead to an enhanced (if decreased perceived exertion) or reduced (if increased perceived exertion) self-paced endurance exercise performance. During time to exhaustion tests, the psychobiological model suggests that exhaustion occurs not solely due to muscle fatigue, rather, by the conscious decision to disengage from the endurance exercise task. In highly motivated individuals, this effort-based decision is taken when they perceive their perception of effort as maximal and extension of the endurance task seems impossible.

Contrary to the other models previously outlined, the psychobiological model of endurance exercise performance hypothesises that the sensory signal processed by the brain to produce perceived exertion is not sent via afferent feedback from skeletal muscles or other interoceptors (Marcora, 2009). Instead, perceived exertion arises from the general processing of the corollary discharge, which is linked with the central motor command (de Morree et al., 2012; Marcora, 2009), therefore explaining the change of perceived exertion and performance, when the

central motor command is increased to compensate for muscle fatigue (de Morree and Marcora, 2013),

However, it is important to note that the classical explanation for the possibility of increased central drive to the limb during endurance performance, which is that fatigue develops during endurance exercise of moderate to high intensity. This is when the capacity of the cardiorespiratory system to provide oxygen to the exercising muscles falls behind their demand, thus inducing “anaerobic” metabolism. Therefore, the oxygen supply to contracting skeletal muscles as well as other physiological demands, such as glycogen depletion, affect the onset of fatigue during exercise and they cannot be ignored as they will set the limits of muscle fatigue (Noakes, 2000). On the other hand, central processing of the corollary discharge is changed by mental fatigue (Pageaux et al., 2013; Pageaux et al., 2014). Previous studies that have changed perception of effort via various experimental manipulations (physiological or psychological) have demonstrated an alteration in endurance exercise performance (e.g. Watson et al., 2005). As a consequence, in order to test the validity of this model, further research should aim to manipulate perceived exertion and/or motivation to investigate whether endurance performance could be changed in the absence of an alteration in perceived exertion and/or motivation.

In addition, there are several psychological and physiological factors that affect the rating of motivation, such as recreation, social recognition, enjoyment, appearance, personal development, affiliation, ill-health avoidance, competition, fitness and health pressures. However, it is harder for participants to rate their motivation accurately in laboratory tests by using only a questionnaire, due the lack of several factors which influence motivation (Brehm and Self., 1989; Trail and James, 2001). Figure (6) provides an illustration of how the model can explain the maximal intensity and maximal duration of exercise that can be maintained in the absence of pacing. The solid lines characterise the time course of perception of effort

during endurance exercise at various exercise intensities, with the dotted lines representing the participant potential motivation. When potential motivation is high, exhaustion occurs at later point compared to low potential motivation modified by exercise performance (green vs orange line). When potential motivation is the same, an alteration in time course of perception of effort due to higher exercise intensity, induces a quicker disengagement from the exercise task (red vs green line).

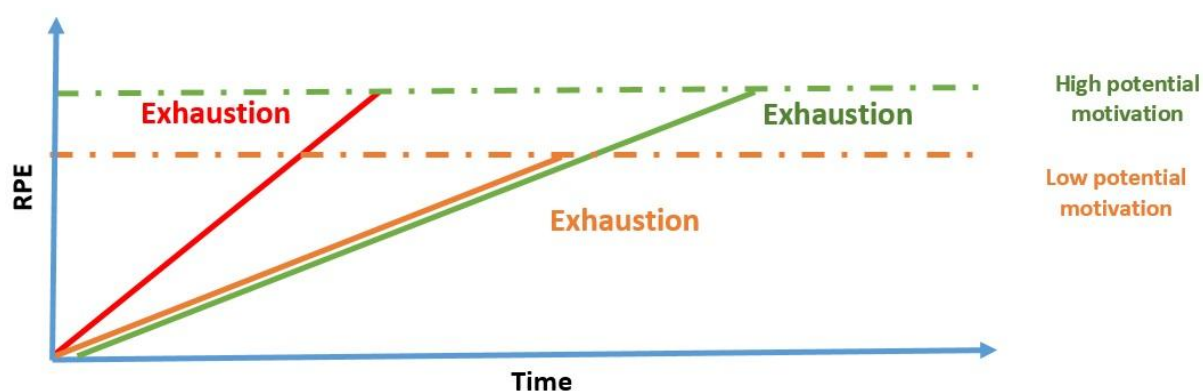


Figure 6: Exhaustion during time to exhaustion tests explained by the psychobiological model.

#### **IV. Manipulation of the perception of effort and resultant effects on endurance performance**

##### **1. Mental fatigue**

Extended periods of mental exertion are known to induce mental fatigue, a psychobiological state considered by particular feelings of “tiredness” and “lack of energy” (Boksem and Tops, 2008). Indeed, cognitive performance has been shown to be negatively affected by mental fatigue and induce impairments in attention, cognitive control, and action monitoring (e.g. Boksem and Tops, 2008; Van der Linden et al., 2003). On the other hand, there is a



paucity of research investigating the effects of mental fatigue on physical exercise performance. In 1906, Mosso stated that his colleagues did poorly in a muscle fatigue test performed after delivering viva examinations and long physiology lectures. Moreover, Bray et al. (2008, 2012) reported that performing a challenging cognitive task prior to, or between, isometric contractions significantly reduced endurance exercise and MVC repeatability of isolated upper limb muscles.

Marcora et al. (2009) were the first to demonstrate the effect of prolonged mental exertion on endurance performance during whole-body exercise. The study induced mental fatigue in a group of fit and healthy participants by requiring them to perform a 90 min bout of a cognitively demanding task, and found that high intensity cycling time to exhaustion performance reduced significantly (mental fatigue:  $10.6 \pm 5.2$  mins vs. control:  $12.5 \pm 5.6$  mins). However, the physiological mechanisms highlighting the negative effect of mental exertion on subsequent endurance exercise performance are not currently well known. Marcora et al. (2009) did not find any influence of mental fatigue on the respiratory, cardiovascular, and metabolic responses to high-intensity cycling performance. In Marcora's study, participant motivation prior to the period of the high intensity cycling test was also not affected by mental fatigue.

However, motivation is difficult to accurately measure by a questionnaire, due the lack of psychological and physiological factors of motivation especially in laboratory cases, which are more difficult to be unaffected (Brehm and Self., 1989). The only factor that could explain the participant's decision to terminate the time to exhaustion test early in the mental fatigue condition was the higher perceived exertion. Thus, according to the psychobiological model of endurance performance, mentally fatigued participants disengaged earlier from the task as the

perceived exertion was maximal, and continuation of the endurance exercise task did not appear to be possible.

There are several studies (Brownsberger et al., 2013; Pageaux et al.2015; Pageaux et al.2014) which have investigated the effect of mental fatigue on self-paced endurance exercise performance. These authors (de Moree et al., 2012; Marcora and Staiano, 2010; Pageaux et al., 2014) demonstrated that mentally fatigued participants generated a lower power output at a given rating of perceived exertion. Indeed, a reduction in endurance exercise performance following mental exertion has been previously linked to response inhibition (Pageaux et al., 2014). Response inhibition is a cognitive procedure that is mediated to an inhibition of unwanted motor or emotional responses, and has been associated with decision-making in human volition (Haggard, 2009), and perceived exertion during exercise (de Moree et al., 2012).

In this regard, Pageaux et al. (2014) have found that following 30 min of mental exertion involving response inhibition, subsequent self-paced endurance exercise performance is reduced, due to a higher perception of effort. In addition, Marcora et al. (2009) demonstrated that following the development of mental fatigue, subjects disengaged from a time to exhaustion task earlier than in a control condition non-fatigued. Marcora et al. (2009) suggested that this was because of a higher perception of effort during the time to exhaustion task, meaning they reached their maximal level of perceived effort and disengaged from the task earlier than in the control condition.

These findings tend to support the psychobiological model of endurance exercise performance in which perceived exertion has a major role in limiting endurance performance (Marcora and Staiano, 2010). Indeed, a recent study by Pageaux et al. (2014) provides evidence that self-paced running performance is reduced by prior mental exertion inducing a response inhibition.

Response inhibition following a mentally taxing task (stroop task), was linked with a reduction in the average running speed selected by the participant through the time trial. Nevertheless, the pacing strategy was not influenced by prior mental exertion involving response inhibition. Interestingly Pageaux's study suggests that performing a mentally taxing task can reduce subsequent endurance exercise performance without participants feeling mental fatigue at rest (Pageaux et al., 2014).

In contrast to the above-mentioned studies which have shown that prolonged periods of mental exertion might increase the perception of effort, several studies have demonstrated that it is also possible to reduce the perception of effort during exercise. There are several stimuli that have been used as stimulators of CNS and exercise endurance tasks, such as taurine, carbohydrates, glucoronolactone and vitamins of the B-group (Clauson et al., 2003), in addition, the experimental study has been conducted on ten runners who performed  $\dot{V}O_2\text{max}$  and time to exhaustion tests. Thus, repeated measures analysis shows that, significant differences were found in  $\dot{V}O_2\text{max}$  ( $P < 0.001$ ) and time to exhaustion ( $P < 0.000$ ) values in all testing sessions (Rahnama et al., 2010). As a result, these beverages have stimulant effects on the central nervous system (CNS) and their consumption is accompanied by an expectation of improving the user's performance physically and mentally (Oteri et al., 2007).

In addition, Swart et al. (2009) presented that amphetamines (methylphenidate) ingested prior to  $\dot{V}O_2\text{max}$  and time to exhaustion, enabled the participants to cycle for approximately 32% longer before power output fell to 70% of the starting value, compared to the placebo condition output at a fixed RPE. Participants receiving methylphenidate had significantly higher power outputs, heart rates, oxygen consumptions, ventilatory volumes and blood lactate concentrations although electromyographic activity remained unaffected. Therefore, the ingestion of a centrally acting stimulant hence allowed participants to exercise for longer at

higher metabolic stress and cardiorespiratory system indicating the presence of a muscular reserve in the natural state. Moreover, this suggests that endurance performance is not only “terminated” by mechanical failure of the exercising muscles (“peripheral fatigue”), but also performance during prolonged endurance exercise under normal conditions is highly regulated by the central nervous system to assure that whole-body homeostasis is protected and an emergency reserve is always present (Swart et al., 2009). However, amphetamines mentioned in the list of drugs banned by WADA is determined by the World Anti-Doping Agency, which was established in 1999 to deal with the increasing problem of doping in the sports world.

## **2. Caffeine**

Caffeine is one of the most commonly used performance enhancing substances by athletes, with 3 out of 4 elite athletes reported to ingest caffeine prior to competitions (Del Coso et al., 2012). In addition, caffeine is also metabolized by the liver, creating an enzymatic action, resulting in three main metabolites, which are paraxanthine, theophylline and theobromine. These then create a pharmacological effect on the CNS via specific signalling pathways (Goldstein et al., 2010). Although many scientists have come across implications of the effect of caffeine on the CNS’s influences to exercise, researchers have also found conflicting evidence that suggests that researchers cannot conclude whether the effects of CNS are muscular or neural (Goldstein et al., 2010; Green et al., 2007; Spriet,1995). Several scientists have investigated these three metabolites from plasma in urine after caffeine ingestion (Tang-Liu, 1983). Tang-Liu (1983) investigated how these metabolites occur after caffeine ingestion. Tang-Liu (1983) established that ring oxidation of these three metabolisms occurs then causes further metabolism to monomethyluric acids, dimethyluric acids, and monomethyixanthines (Tang-Liu, 1983). O’Donnell (2012) reported that paraxanthine is the major metabolite used in caffeine ingestion. As a result, using caffeine as an antagonist, it can be suggested that this

barrier between the receptors can delay the effects of tiredness, resulting in increased performance.

**a. Mechanisms**

While the pharmacological effects of caffeine ingestion are well known (Burke, 2008; Del Coso et al., 2008 and 2009; Doherty et al., 2004), its ergogenic effect on endurance performance activities is debated within the scientific literature. Moreover, there are several mechanisms surrounding caffeine and the ergogenic aid. Knowing the importance of these mechanisms is necessary to understand the effect caffeine has on the body. One of the descriptions of caffeine is that it stimulates adrenaline secretion resulting in the mobilisation of free fatty acids (FFA). This enhanced delivery of fuel in the active muscles results in a 'Randle' effect, increasing fat oxidation and sparing limited and critical muscle glycogen stores (Graham, 2001). This glycogen plays a minimal role in substrate and exercise duration and does not affect glycogen availability (Green et al., 2007).

The ergogenic effect of caffeine has been the focus of an extensive amount of research over the last two decades leading to its widespread use among athletes during competition (Spriet et al., 1992). The improved endurance performance following caffeine consumption has been suggested to arise from its effect of reducing the muscle's reliance on muscle glycogen stores, increasing serum free fatty acid availability (Flinn et al., 1990), and increasing reliance on fat metabolism, as demonstrated from respiratory exchange ratios (Spriet et al., 1992). Instead, studies have suggested that caffeine may exert its effect neurally, increasing calcium release from the sarcoplasmic reticulum and altering muscle contractile properties. It is likely that the concentration of caffeine required to produce calcium release that would have a significant ergogenic impact on muscle function could lead to negative events that would compromise endurance performance (Kalmar and Cafarelli, 1999).

A further main mechanism that might explain the ergogenic effect of caffeine are acts such as an adenosine receptor antagonist (Fredholm et al., 1999). This means that caffeine binds to the same receptor as brain adenosine, but without reducing neural activity. As a consequence, the activation of the pituitary and adrenal glands increase noradrenaline and dopamine release in the brain, leading to increased attention, reduced perception of effort, and ultimately increased time to exhaustion (Bell and McLellan, 2002). A reduction in perception of effort is normally experienced during endurance exercise after caffeine ingestion. Doherty and Smith (2005) have found that caffeine consumption reduces ratings of perceived exertion (RPE) by an average of 0.47 standard deviation, compared with a placebo and that 29% of the difference in the ergogenicity of caffeine was induced by changes in RPE (Doherty and Smith, 2005).

There is data that the highlighting physiological mechanism for reduced RPE is caffeine's antagonism of adenosine receptors (Davis et al., 2003; Fredholm et al., 1999), which also seems to account for hypoalgesia of naturally inducing muscle pain during dynamic exercise (Motl et al., 2003; O'Connor et al., 2004). To date, however, the implications of attenuated perception of effort and muscle pain during exercise for improved performance remain largely not investigated (Doherty and Smith, 2005). A decreased perceived exertion might, then, lead to enhancement in performance by allowing subjects to work at a higher intensity. A potential mechanism is by caffeine blocking brain adenosine receptors, mainly A<sub>1</sub> and A<sub>2A</sub> sub-types, competitively blocking their action (Holtzman et al., 1991; Ribeiro and Sebastião, 2010) and influencing an increase in noradrenalin, glutamate, and the release of dopamine (Ferré et al., 1997; Smits et al., 1987). The ability of caffeine can reduce cerebral blood flow (Cameron et al., 1990). Caffeine causes a reduction in myocardial blood flow, by preventing A<sub>1</sub>, A<sub>2A</sub> and A<sub>2B</sub> adenosine receptors in blood containers and limiting adenosine-mediated vasodilatation (Namdar et al., 2009).

A<sub>1</sub> receptors are available over most parts of the brain areas (Cappelletti et al., 2015). Therefore, the mechanism of caffeine's action, when there is consistent caffeine consumption during endurance exercise testing, regardless of intensity, mode, or the length of exercise, is an alteration in the athlete's perceptual response. When ingested prior to exercise, caffeine has been suggested to delay the onset of exhaustion (Costill et al., 1978), due to a stimulation of FFA mobilisation (Bellet et al., 1965), leading to a glycogen-sparing effect (Hickson et al., 1977). In vitro studies have suggested that caffeine may inhibit phosphorylase a (Kasvinsky et al., 1978) and stimulate intramuscular triglycéride breakdown directly (Crass, 1973) or indirectly (Randle, 1963). Therefore, due to central nervous system stimulation and inhibition of phosphodiesterase activity, caffeine ingestion results in an increased FFA mobilization 1-2 h post ingestion (Bellet et al., 1968; Wilcox, 1982). An elevated plasma FFA level has been shown to have a glycogen-sparing effect during exercise in both Rats and human (Hickson et al., 1977).

#### **b. Doses and timing**

From many researchers, it has been established that a dosage of 3-6mg/kg is an effected method of increasing performance (Doherty, 1998; Goldstein et al., 2010; Lieberman, 2002; Rogers, 2013). However, Bruce et al. (2000) reported that dosages of 6-9mg/kg were similarly effective when improving performance in a 2000m rowing exercise.

Even though Bruce et al (2000) found 6-9mg/kg had a positive effect, Van Soeren and Graham (1998) established conflicting evidence, that a larger dosage of 9mg/kg had more negative effects. Van Soeren and Graham (1998) reported that when participants ingested 9mg/kg, they became overdosed and mentally confused, the participants could not concentrate, were giddy, talkative, could not perform simple tasks such as telling the time exactly and some even felt

intoxicated. In contrast, the consumption of lower doses of 1 mg/kg of caffeine did not improve performance (Jenkins et al., 2008). Similar outcomes have been found in specific team-sports activities: the consumption of 6 mg/kg of caffeine increased the ability of repeated sprint performance (Glaister et al., 2008 ; Stuart et al.,2005), while the consumption of 1 mg/kg of caffeine did not improve repeated sprint performance (Glaister et al., 2008; Stuart et al., 2005). Several studies have suggested that caffeine has an ergogenic effect when given one hour prior to endurance performance, lasting between 30-120 minutes (Costill et al., 1978; Tarnopolsky and Cupido, 2000). In addition, extensive investigations have been performed based on the variation of caffeine dosages. In previous studies this has ranged from 1-15 mg of caffeine/kg of body mass (Bell and McLellan, 2002). Pasman et al. (1995) found that 5, 9 or 13 mg/kg of caffeine produced significant but quite similar ergogenic benefits when performing endurance cycling. As mentioned above, the caffeine dose has an impact on the ergogenic effect experienced. Talanian and Spriet (2016) found that low and moderate doses of caffeine (low = 1.5 mg.kg<sup>-1</sup>, 100 mg vs. moderate = 2.9.9 mg.kg<sup>-1</sup>, 200 mg) consumed late in a 40 min TT enhanced performance over a placebo condition. However, the moderate dose improved TT performance to a greater extent than the lower dose. Much higher doses have also been used within the research literature. Bortolotti et al. (2014) investigated the use of 6 mg.kg<sup>-1</sup> body weight ingested 1 hour prior to a 20-km cycling time trial. However, interestingly on this occasion caffeine ingestion did not show any performance improvements over a placebo condition. The authors failed to find an effect of caffeine on physiological or psychological parameters measured during the time trial, and suggested that differences in protocols between studies might have provided a confounding effect.



## **Overview and conclusions of the literature review**

Therefore, in several studies (as outlined above), alterations in the perception of effort independently from physiological parameters have been shown to reduce self-paced, time-to-exhaustion and time trial based exercise performance. These findings have important implications for physiological testing, where parameters are determined using self-paced, time-to-exhaustion and time trial protocols. Then, the present thesis explored the neurophysiology models of perception of effort and their role during endurance performance regulation and how perception of effort can be generated. This was followed by the effect of mental fatigue, as well as caffeine, on perception of effort and how this reflects on endurance performance. Therefore, the next section will identify the aims and hypotheses across all experimental studies.

## **V. Aims and outline of the thesis**

The aim of the present thesis was to investigate the relationship between the perception of effort and endurance exercise performance. In four experimental studies the thesis will explore the following aims and hypotheses:

*Chapter 3 - Study one:*

***Title: RPE as a predictor of endurance performance***

**Aim:** To compare the predictive ability of the rating of perceived exertion and traditional physiological parameters (e.g. VO<sub>2</sub>max, LT, economy) on exercise performance.

**Hypothesis:** Perception of effort would be a stronger predictor of 30 minute cycling performance than VO<sub>2</sub>max, LT, and cycling economy

*Chapter 4 - Study two:*

***Title: The effect of mental fatigue on critical power and the anaerobic work capacity***

Aim: To establish whether mental fatigue induced increases in the perception of effort will change the physiological performance parameters, critical power and W'.

Hypothesis: Mental fatigue will reduce time to exhaustion performance resulting in an altered CP and W'.

*Chapter 5 - Study three:*

***Title: The reliability of rating of perceived exertion via caffeine ingestion during high-intensity interval training***

Aim: To examine the reliability of self-paced exercise at a fixed rating of perceived exertion during high intensity interval exercise, following the ingestion of 3 mg.kg<sup>-1</sup> caffeine.

Hypothesis: Power output, cadence and heart rate are high levels of reliability during self-paced exercise at fixed RPE intensities via caffeine ingestion.

*Chapter 6 - Study four:*

***Title: Does chronic use of caffeine reduce its acute ergogenic effects during high intensity interval training?***

Aims:

1. To examine whether acute caffeine ingestion prior to training increases the work that can be completed during an RPE-clamped self-paced high-intensity exercise.

2. To establish whether chronic caffeine ingestion during a period of exercise training will alter the acute effects of caffeine on RPE-clamped self-paced high-intensity exercise.

Hypotheses:

1. Acute caffeine ingestion will increase the work rate during RPE-clamped self-paced high intensity exercise.
2. Chronic caffeine ingestion will reduce the acute effects of caffeine on work rate during RPE-clamped self-paced high intensity exercise.

**CHAPTER 3**

**RPE AS A PREDICTOR OF ENDURANCE**

**PERFORMANCE**

## **RPE AS A PREDICTOR OF ENDURANCE EXERCISE PERFORMANCE**

### **I. Abstract**

Introduction: The purpose of this study was to examine the physiological and perceptual determinants of endurance exercise performance in a heterogeneous group of recreationally active individuals.

Methods: Fourteen participants (8 males and 6 females) were recruited to take part in the study. Each participant completed an incremental exercise test to exhaustion to identify  $\dot{V}O_{2\max}$  and maximal minute power, a submaximal test to assess cycling efficiency and lactate threshold, a fixed power output equivalent to 75% MMP, the slope of the increase in perception of effort over time, termed 'perceptual efficiency, and a 30 min time trial to assess cycling performance. Pearson bivariate 2-tailed correlation tests were used to determine correlations between variables, and single linear regression tests were performed to determine the standard error of the estimate (SEE), and a 95% confidence interval (CI).

Results: The single best predictor of laboratory cycling time trial performance was  $\dot{V}O_{2\max}$  ( $r = 0.94$ ,  $p < 0.01$ ). The standard error of the estimate was 14.0 % and the maximal minute cycling power output was also strongly correlated with time trial performance ( $r = 0.90$ ,  $p < 0.01$ ;  $SEE = 18.5\%$ ). Finally, the RPE slope was inversely correlated with laboratory based cycling performance ( $r = -0.66$ ,  $p < 0.05$ ,  $SEE = 31.4\%$ ). Blood lactate was at 2  $\text{mM.L}^{-1}$  (W) ( $r = 0.66$ ,  $p < 0.05$ ,  $SEE = 31.49\%$ ). However, blood lactate at 4  $\text{mM.L}^{-1}$  (W) ( $r = 0.48$ ,  $p > 0.05$ ,  $SEE = 36.84\%$ ), and also cycling economy at 180/W ( $r = 0.20$ ,  $p > 0.05$ ,  $SEE = 41.09\%$ ) were not correlate with 30 min cycling time trial performance.

Discussion: The results showed that  $\dot{V}O_{2max}$  was the strongest predictor of laboratory time trial performance. Even though other measures of participant mass, maximal minute power output, RPE slope, and cycling power at a blood lactate of 2mmol/L were significantly related to cycling time trial performance. A linear regression analysis identified that the single best predictor of cycling performance was  $\dot{V}O_{2max}$ .

## **II. Introduction**

Cycling time trials are important elements of Grand Tour cycling events, such as the Tour de France (Lucia et al., 2004). Typically time trials within road cycling events cover distances between 10 and 44 km over periods of 10 min to 60 min. Subsequently, success in these individual tests of human endurance is largely determined by the cyclist's level of aerobic fitness (Åstrand, 2003). Elite athletes have been seen to possess aerobic capacity 50-100% greater than those normally seen in healthy young individuals. Indeed, a strong relationship has been found between  $\dot{V}O_{2max}$  and cross-country skiing race performance levels (Ingjer, 1991). However, most endurance events are performed at an exercise intensity that does not evoke  $\dot{V}O_{2max}$ , with 40km time trial performance being conducted in the region of 70-80% maximal aerobic power (Smith et al., 2001). Thus, as suggested by Lucia et al. (2004), submaximal factors such as the lactate threshold, fractional utilisation of  $\dot{V}O_{2max}$  and cycling economy are more important than  $\dot{V}O_{2max}$ . Indeed, power output at lactate threshold has been shown to interact with both  $\dot{V}O_{2max}$  and economy; it is closely related to maximal aerobic power (Sunde et al., 2010). In turn, maximal aerobic power has been shown to be a strong predictor of 16km cycling time trial performance (Balmer et al., 2000). In addition, Joyner and Coyle (2008) formalised the proposed determinants of endurance performance into a schematic

model. The model suggests endurance performance power or velocity is governed by three key parameters: performance  $\dot{V}O_2$ ,  $O_2$  deficit, and gross mechanical efficiency. More recently, Storen et al. (2013) have adapted a model of performance first proposed by di Prampero (2003) to investigate determinates of endurance performance. Storen et al. (2013) demonstrated that a combination of  $\dot{V}O_{2max}$ , cycling economy and performance  $\dot{V}O_2$  could account for ~84% of the variation in 15km time trial performance in well-trained cyclists, with anaerobic capacity only accounting for an extra ~2%. However, it is important to consider that cycling performance is not only determined by physiological parameters. Recently Scherr et al. (2012) demonstrated a strong link between Borg's rating of perceived exertion (RPE) and physiological parameters associated with endurance exercise performance (heart rate and blood lactate concentration) in 2,560 participants. Scherr et al. (2012) demonstrated a strong relationship between RPE (Borg scale 6-20), heart rate ( $r = 0.74$ ,  $P < 0.001$ ) and blood lactate concentration ( $r = 0.83$ ,  $P < 0.001$ ) during submaximal exercise performance. Moreover, a fixed blood lactate level of 3 or 4  $mM.L^{-1}$  was seen to correlate with RPE values of 10.8 and 13.6 respectively. Scherr's study suggests that perception of effort could be an alternative method to consider endurance performance determinants compared to more "traditional" physiological parameters. Indeed, Marcora and Staiano (2010) have demonstrated that the perception of effort can be seen to determine endurance exercise performance independently of alterations in cardiorespiratory, metabolic and neuromuscular parameters. Therefore, it is possible that perception of effort could play a major role in determining endurance performance. However, Marcora and Staiano's study was conducted using a time to exhaustion test, and therefore does not permit the self-regulation of speed/power output experienced during endurance exercise performance (i.e. pacing strategy). Therefore, the degree to which perception of effort can account for variation in a self-paced time trial endurance performance, and how this compares to "traditional" physiological parameters is unknown. As a consequence, the hypothesis of this

study was that the perception of effort would be a stronger predictor of a 30 minute cycling performance than  $\dot{V}O_2\text{max}$ , LT, and cycling economy.

### **III. Methods**

Fourteen participants, 8 males (age  $30 \pm 6$  years, weight  $77 \pm 14$  kg, height  $178 \pm 8.0$  cm) and 6 females (age  $27 \pm 4.8$  years, weight  $61.8 \pm 53$  kg, height  $167.5 \pm 7.6$  cm), were recruited to take part in the study. All participants cycled as part of their habitual weekly physical activity regime. The experimental protocol and procedures were approved by the Ethics and Research Committee of the University of Kent. All participants provided written informed consent and completed a health questionnaire prior to participation.

#### Experimental Design:

All participants completed four test visits, the first being an incremental exercise test to volitional exhaustion to identify  $\dot{V}O_2\text{max}$  and maximal minute power, the second to assess cycling economy and lactate threshold, the third to identify the rate of increase in the perception of effort at a fixed work rate, and the final visit to assess cycling time trial performance.

During visit 1, the participants completed an incremental exercise test to exhaustion on a cycle ergometer (Schoberer Rad Messtechnik, Jülich, Germany). Following a 5-minute warm-up at 50 W, the participants performed the incremental cycling test until volitional exhaustion using a 25 W/min increment rate. Pulmonary gas exchange was measured using a breath-by-breath



analyser (MetaLyzer, Cortex Biophysik, Leipzig, Germany) throughout the test. Their heart rate was continuously measured throughout the test (S810i, Polar, Kempele, Finland), and the perception of effort, recorded as a rating of perceived exertion (Borg 6-20 scale; Borg, 1998), was taken at the end of each minute.  $\dot{V}O_{2max}$  was calculated as the highest  $\dot{V}O_2$  attained during a 60 s period in the test. Maximal minute power output (MMP) was calculated as the highest 60 second power output from the test using the formulae:

$$W_{max} = WE + (25W/t \times tE)$$

Where  $W_{max}$  = maximal power output (W); WE = power output of last stage completed; 25 W = work rate increment; t = work rate duration (s); and tE = duration of final stage (s).

During visit 2, the participants completed a submaximal exercise test to assess their lactate threshold and cycling economy. Initially, the participants warmed up on a cycle ergometer (Schoberer Rad Messtechnik, Jülich, Germany) for 5 minutes at 50 W. The exercise test commenced at 80 W and consisted of between 6-8 subsequent stages, each 6 minutes in duration. The work rate was increased by 25 W at the end of each 6 minute stage. Expired air was collected during the last minute of each stage using Douglas bags (Hans Rudolph, USA). After the test, the collected air was analysed for the fraction of expired oxygen and carbon dioxide using a Servomex 5200 gas analyser (Servomex, Crowborough, East Sussex). Blood samples to assess for the level of lactate accumulation were taken at minutes 3 and 6 of each stage. The test terminated once the participant's measured blood lactate exceeded 4 mM.L<sup>-1</sup>. Cycling economy was subsequently calculated as the oxygen cost of cycling for a given power output in W per litre of oxygen consumed.

During visit 3, the participants cycled continuously for 15 minutes at a power output equivalent to 75%MMP (established during visit 1) on a cycle ergometer (Schoberer Rad Messtechnik,

Jülich, Germany). The test required participants to complete 15 minutes of cycling at 75%MMP, or until their cadence fell below the required 80 rev. min<sup>-1</sup> for more than 5 seconds. The Borg 6-20 RPE scale (Borg, 1998) was used to assess perceived exertion at each minute throughout the test. The slope of the increase in perception of effort over time was subsequently calculated to provide a single value for RPE (Angius et al., 2016), and used to provide an indication of ‘perceptual efficiency’ i.e. the relationship between the required work rate (the physical stimulus), and the change in RPE (the perceptual response). The participants then rested for 15 minutes, after which they undertook a familiarisation of the performance time trial to be conducted during visit 4.

Finally during visit 4, the participants performed a cycling performance time trial. After a 10 minute warm-up at 100 W, the participants completed a time trial in which they were asked to complete as much work as possible within a period of 30 minutes. During the performance time trial, the participants cycled on an SRM ergometer (Schoberer Rad Messtechnik, Jülich, Germany). Expired gases analysed by an online breath-by-breath system (MetaLyzer, Cortex Biophysik, Leipzig, Germany), and RPE were monitored every 5 minutes during the test. Power output, cadence and heart rate were continuously monitored throughout. Blood samples were taken pre, every 5 minutes during and at the end of the test to assess and test blood lactate concentration.

#### Statistical Analysis:

Data were initially assessed for normality of distribution using a Shapiro-Wilk test. Pearson bivariate 2-tailed correlation tests were used to determine correlations between physiological variables, and RPE slope with cycling time trial performance. Moreover, a single regression

analysis was used to determine the standard error of the estimate (SEE), and a 95% confidence interval (CI), and F values. Multiple regressions were not used because of the low number of subjects ( $n = 14$ ). Data are presented as mean  $\pm$  standard deviation unless otherwise stated. Statistical analyses were performed using the software program SPSS, version 20.0 (Statistical Package for Social Science, Chicago, Illinois, USA). Statistical significance was accepted at an alpha value of  $P < 0.05$ .

#### IV. Results

All participants completed each of the 4 laboratory visits. The mean  $\pm$  SD of data for each of the laboratory tests is presented in Table 1 below.

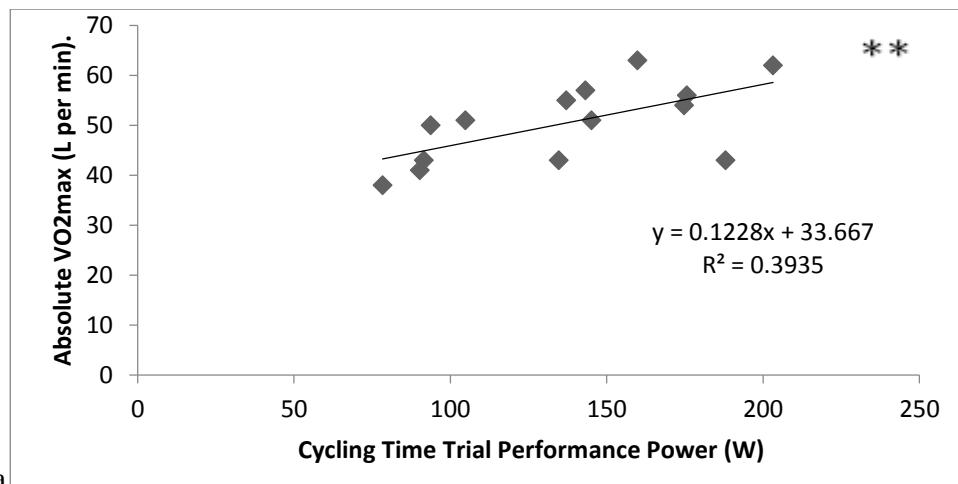
Table 1: Results from laboratory tests ( $n = 14$ ).

	Mean	$\pm$	SD
<b>Incremental Exercise Test</b>			
$\dot{V}O_{2\max}$ (L.min <sup>-1</sup> )	3.54	$\pm$	0.87
$\dot{V}O_{2\max}$ (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	50.5	$\pm$	7.89
MMP (W)	292.07	$\pm$	81.7
<b>Lactate Threshold and Economy Test</b>			
Power at 2 mM.L <sup>-1</sup> (W)	91.35	$\pm$	33.44
Power at 4 mM.L <sup>-1</sup> (W)	152.92	$\pm$	47.88
Economy 80W (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	53.69	$\pm$	8.96
Economy 105W (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	61.57	$\pm$	10.93
Economy 130W (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	64.06	$\pm$	11.06
Economy 155W (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	68.36	$\pm$	13.33
Economy 180W (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	67.21	$\pm$	9.62
<b>75% MMP Test</b>			
RPE Slope (A.U.)	0.72	$\pm$	0.26
<b>30 min Time Trial Test</b>			
Mean Power (W)	137.13	$\pm$	40.32
Mean $\dot{V}O_2$ (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	37.59	$\pm$	7.38
% $\dot{V}O_{2\max}$	74.97	$\pm$	11.6
Work done (kJ)	246.28	$\pm$	72.67

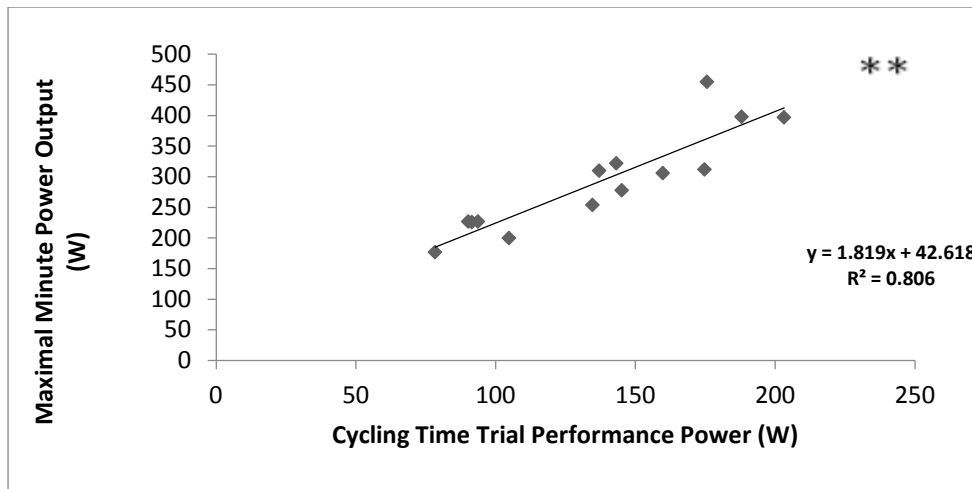
*$\dot{V}O_2\text{max}$  and MMP vs. Cycling Time Trial Performance:*

The single variable that correlated best with cycling time trial performance was absolute  $\dot{V}O_2\text{max}$  ( $r = 0.94$ ,  $p < 0.01$ ; SEE = 14.0%; 95% CI = 33.59 to 52.86 figure 7 a) Maximal minute power output from the incremental exercise test was also significantly correlated with cycling time trial performance ( $r = 0.90$ ,  $p < 0.01$ ; SEE =18.5%; 95% CI = 0.31to o.58 figure 7 b) However,  $\dot{V}O_2\text{max}$  per kilogram of body mass was not found to correlate with cycling time trial performance ( $r = 0.62$ ; SEE=32.6%  $P < 0.05$ ; 95% CI = 0.70 to 5.70 figure 7c)

a)



b)



c)

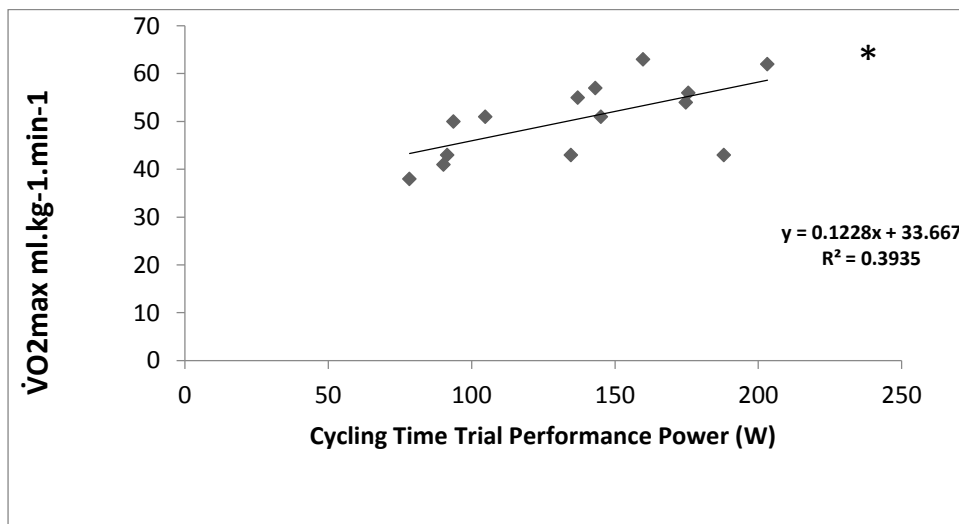


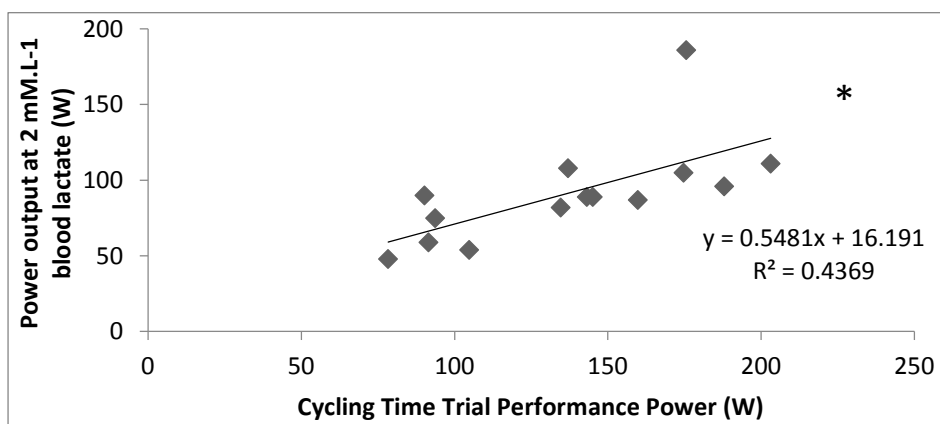
Figure 7: Relationship between time trial performance and maximal physiological parameters.

a)  $\dot{V}O_2\text{max}$  in litres per minute ( $P < 0.01^{**}$ ). b) Maximal Minute Power Output in watts ( $P < 0.01$ ). c)  $\dot{V}O_2\text{max}$  per kilogram body mass ( $P < 0.05^*$ ).

*Lactate threshold vs. Cycling Time Trial Performance*

Cycling power output at 2 mM.L<sup>-1</sup> blood lactate was correlated with cycling time trial performance ( $r = 0.66$ ; SEE = 31.4%  $p < 0.05$ ; 95% CI = 0.23 to 1.37 figure 8 a). However, cycling power output at 4 mM.L<sup>-1</sup> blood lactate was not correlated to time trial performance ( $r = 0.48$ ; SEE = 36.8 %,  $p > 0.05$ ; 95% CI = -0.61 to 0.87 figure 8 b)

a)



b)

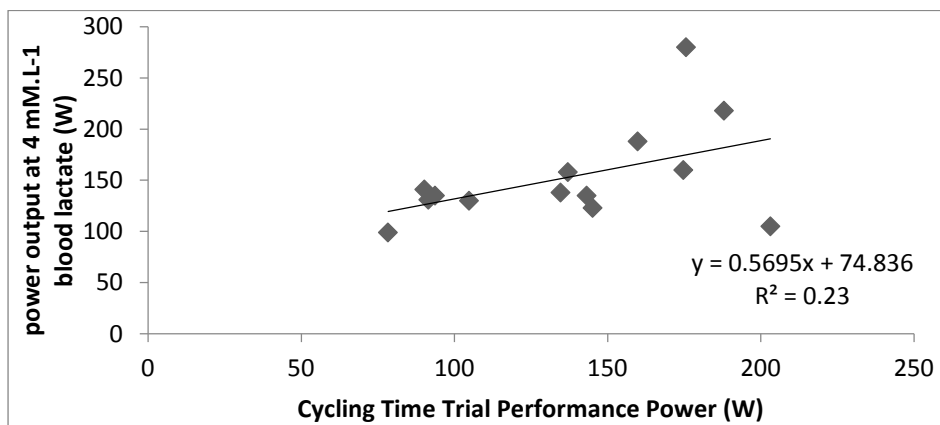
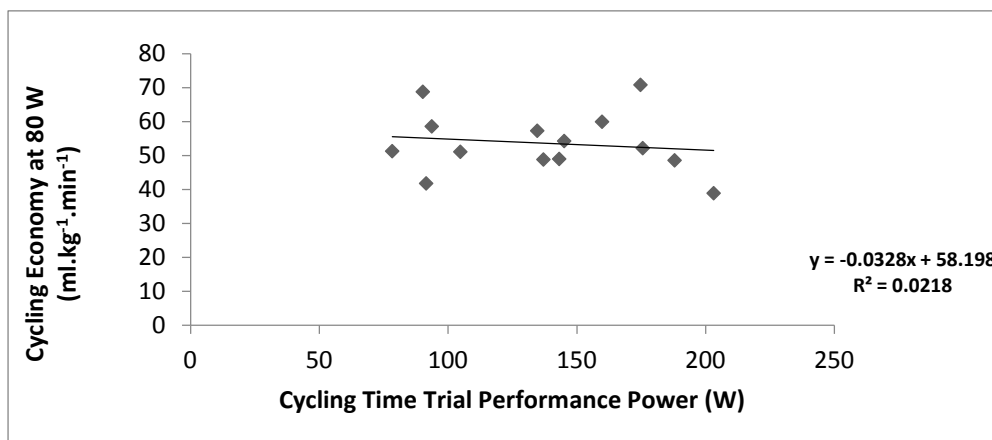


Figure 8: Relationship between cycling time trial performance and: a) power output at 2 mM.L<sup>-1</sup> blood lactate (W;  $P < 0.05^*$ ); b) Power output at 4 mM.L<sup>-1</sup> blood lactate (W;  $P > 0.05$ )

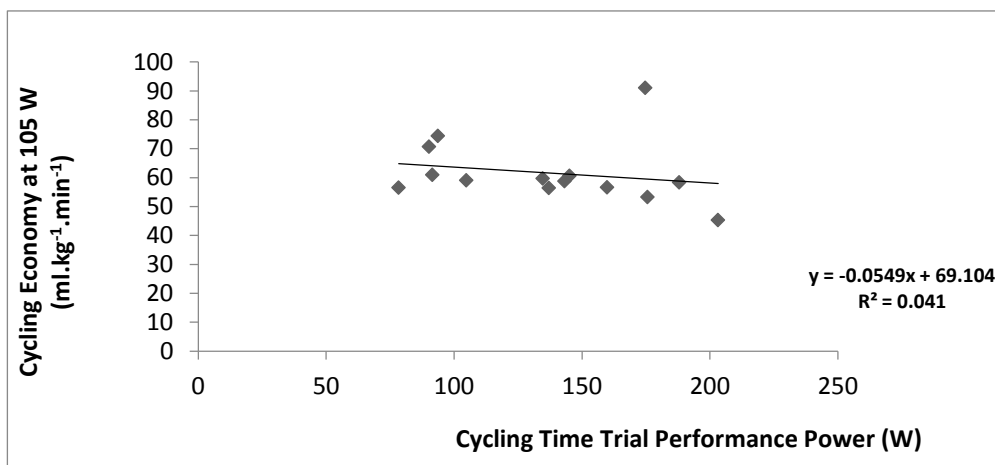
*Cycling economy vs. Cycling Time Trial Performance*

There were no significant correlations found between cycling economy at 80 W ( $r = 0.20$ ; SEE = 41.9%; 95% CI= figure 9 a ), 105W ( $r = 0.20$ ; SEE = 41.9%; 95% CI= figure 9 b ), 130W ( $r = 0.20$ ; SEE = 41.9%; 95% CI= figure 9 c ), 155W ( $r = 0.20$ ; SEE = 41.9%; 95% CI= figure 9 d ), 180W( $r = 0.20$ ; SEE = 41.9%; 95% CI= figure 9 e), and time trial performance (all  $P > 0.05$ ).

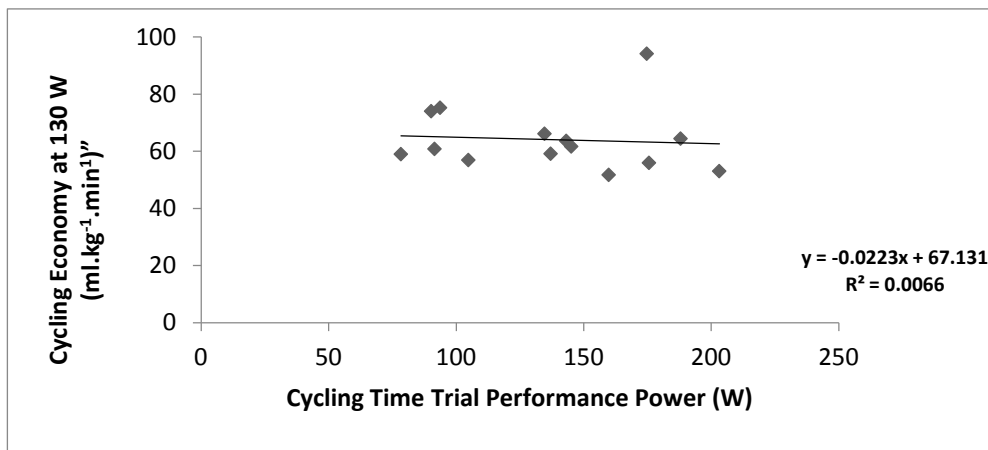
a)



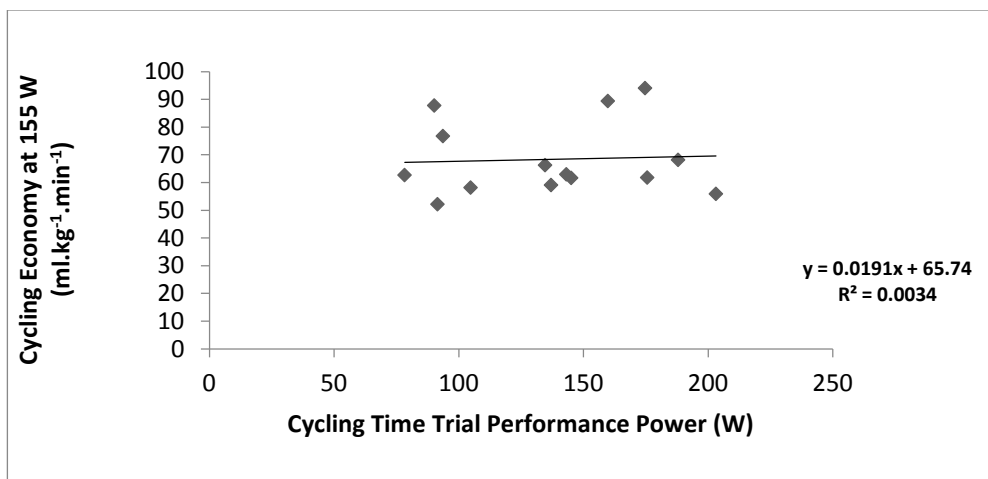
b)



c)



d)



e)

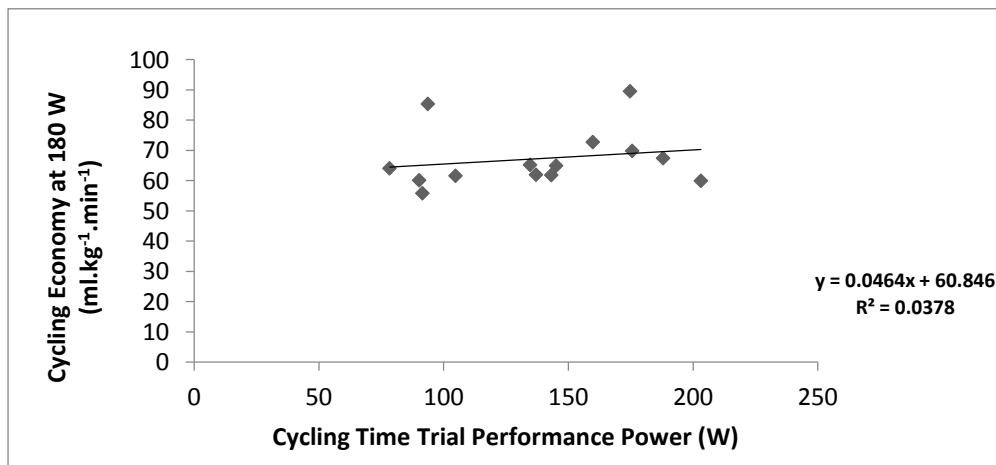




Figure 9: Relationship between cycling time trial performance and cycling economy at: a). 80W; b). 105 W; c). 130 W; d). 155 W; e). 180 W. All  $P > 0.05$ .

#### *RPE vs. Cycling Time Trial Performance*

RPE was inversely correlated with cycling time trial performance ( $r = -0.66^*$ ,  $P < 0.05$ ; SEE = 31.4%; 95% CI = -172.3 to -29.35 figure 10).

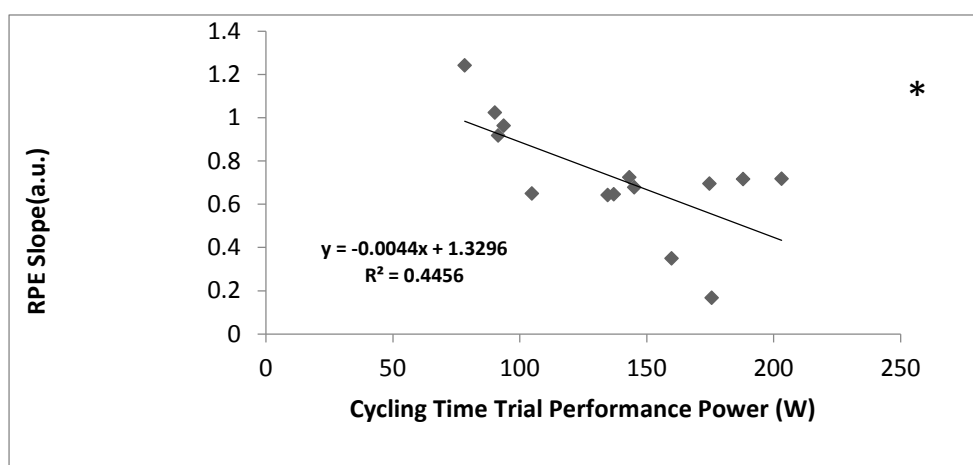


Figure 10: Relationship between cycling time trial performance and RPE slope ( $P < 0.05^*$ ).

## **V. Discussion**

The primary aim of the current study was to investigate the relationship between cycling time trial performance and physiological ( $\dot{V}O_{2\max}$ , LT, cycling economy) and psychobiological (RPE) parameters. Results from the current study demonstrated that absolute  $\dot{V}O_{2\max}$  ( $L \cdot \min^{-1}$ ), maximal minute power, body mass, power output at  $2 \text{ mM} \cdot L^{-1}$  blood lactate, and RPE were correlated with 30 min cycling time trial performance. However, the single variable correlating best with cycling time trial performance was absolute  $\dot{V}O_{2\max}$  ( $L \cdot \min^{-1}$ ) ( $r = 0.94$ ,  $P < 0.01$ ).

In support of this finding, previous research has suggested that endurance performance can be predicted by  $\dot{V}O_{2max}$ , as both  $\dot{V}O_{2max}$  and exercise performance are determined by the integrative capacity of the heart to produce a high cardiac output, whole body haemoglobin, muscle oxygen extraction and high muscle blood flow, and the ability of the lungs to oxygenate the blood (Bassett and Howley, 2000; Dempsey, 1986; Kanstrup and Ekblom, 1984; Mitchell et al., 1958; Rowell, 1986; Saltin and Strange, 1992). However,  $\dot{V}O_{2max}$  has been shown to be a poor predictor of performance within elite athletes, due to large differences in performance, but relatively similar  $\dot{V}O_{2max}$  values (Storen et al., 2013). This is contrary to the findings of the current study, although it is highly likely that this is the result of the relatively “untrained” study population. This study recruited moderately trained participants with a wide range of aerobic fitness, and thus performance potential. It is therefore unsurprising that a strong correlation was established between  $\dot{V}O_{2max}$  and time trial performance.

Whilst it is acknowledged that endurance performance is predominantly determined by physiological parameters (Joyner and Coyle, 2008), there is an ever-increasing acknowledgement that the perceptual response to exercise also forms an important part of the jigsaw. Indeed, an interesting and novel finding from the current study was that the perception of effort was a significant correlate of laboratory based cycling time-trial performance ( $r = -0.66$ ;  $P < 0.05$ ). Using a novel test design, the increase in a participant’s RPE was observed as a linear function time during a bout of high intensity cycling at a fixed power output of 75% MMP. The slope of the increase in RPE over the 15 min period of exercise was used as an indicator of what we have termed “perceptual efficiency”. Thus, at the same relative exercise intensity, individuals who demonstrate a steeper slope of increase in RPE are assumed to have a lower “perceptual efficiency” than individuals with a shallower slope over the same time period. The results of the current study demonstrate an inverse relationship between the RPE

slope and a 30 min cycling time trial performance. Thus, individuals with a steeper slope of increase in RPE during a fixed bout of exercise at 75% MMP demonstrated a poorer time trial performance than those who had a shallower increase over the same time period. Indeed, several models report that the RPE is of critical importance in determining endurance exercise performance (Amann and Dempsey, 2016; Noakes et al., 2004; Marcora and Staiano, 2010; Tucker and Noakes, 2009).

The afferent feedback model suggests that following the onset of exercise, thermal, mechanical and chemical stimuli alter intramuscular receptor activity, which affects the firing rate of small-diameter group III/IV afferent fibres (Amann and Dempsey, 2016). Thus, it is likely that this neural feedback was increased during the constant power cycling exercise at 75% MMP, as peripheral muscle fatigue developed due to the accumulation of muscle metabolic by-products. This heightened afferent feedback is thought to cause reflex inhibition of alpha motor neurons at both the muscle and supraspinal levels (Gandevia, 2001), and be a major factor involved in the development of central fatigue (Amann and Dempsey, 2008). The afferent feedback model of endurance exercise performance suggests that this feedback from peripheral muscle fatigue inhibits central motor drive, limiting endurance exercise performance, and resulting in an increased perception of effort (Amann et al., 2013; Wright, 2008).

Scherr et al. (2012) established a strong relationship between RPE and the blood lactate response in a group of 2,560 individuals ( $r = 0.83$ ;  $p < 0.001$ ). Specifically, the lactate threshold and the individual anaerobic threshold were strongly correlated with RPE values of 10.8 and 13.6. Moreover, fixed lactate thresholds of 3 and 4  $\text{mM.L}^{-1}$  were correlated with RPEs of 12.8 and 14.1 respectively. Thus, in line with the afferent feedback model, the increased level of sensory feedback from the accumulation of metabolic by-products could have resulted in the generation of the greater RPEs in Scherr's study. Indeed, afferent feedback from lactate

production has been suggested to be a key factor in the generation of the perception of effort during cycling time trial performance (Wright, 2008).

An alternative hypothesis is provided by Marcora and colleagues (2008, 2009 and 2010) whose psychobiological model suggests that the perception of effort may regulate endurance performance as a consequence of the interaction between potential motivation and effort-based decision-making. According to the psychobiological model of endurance performance, the self-regulation of power/speed output during endurance exercise performance (pacing) is induced mainly by five different motivational/cognitive aspects: (1) perception of effort; (2) potential motivation; (3) knowledge of the distance/time to cover; (4) previous experience/memory of perceived exertion during exercise of varying intensity and duration, and (5) knowledge of the distance/time remaining. However, the psychobiological model is somewhat limited, in that it states that the perception of effort is the sole determinant of endurance performance. Results from the current study suggest that even through “perceptual efficiency” is a significant correlate for endurance exercise performance, it does not have as much predictive power as the  $\dot{V}O_{2max}$ .

Several previous studies have documented significant relationships between physiological parameters (e.g.  $\dot{V}O_{2max}$ , maximal minute power output, lactate threshold, power output at lactate threshold, efficiency), and endurance cycling time-trial performance (Balmer et al., 2000; Hawley and Noakes, 1992; Hoogeveen and Hoogsteen, 1999). The findings of the current study are largely supportive of the previously published literature. Indeed, maximal minute power output has previously been documented to strongly correlate with 30 min cycling time trial performance ( $r = 0.90$ ;  $P < 0.05$ ) (Hawley and Noakes, 1992). Moreover, in accordance with the findings of previous studies (Kuipers et al., 1985; Noakes, 1988), the current study found that LT at  $2 \text{ mM.L}^{-1}$  was correlated with time trial performance ( $r = 0.66$ ;  $p$

< 0.05). This is not an unexpected finding, as power output at LT at 2 mM.L<sup>-1</sup> has been suggested to be influenced by  $\dot{V}O_{2max}$  and cycling economy (Conley and Krahenbuhl, 1980). Indeed, work by Coyle et al. (1988) demonstrated that during a prolonged period of cycling at 88%  $\dot{V}O_{2max}$ , participants with a lactate threshold that occurred at a higher percentage of their  $\dot{V}O_{2max}$  were able to cycle for longer than those who had a LT occurring at a lower %  $\dot{V}O_{2max}$ .

Participants in the present study were all recreationally active males and females representing a rather heterogeneous performance group. The large range of  $\dot{V}O_{2max}$  difference values recorded from the participants may serve as a limitation to the interpretation of the study findings. As  $\dot{V}O_{2max}$  sets the upper limit for endurance performance, and aerobic metabolism is the key energy system for the time trial used within this research study, it is no surprise that with a heterogeneous cohort in terms of both aerobic capacity and time trial performance, the two are strongly correlated. It is worth considering that if a more homogenous group of trained individuals were recruited, the dominance of  $\dot{V}O_{2max}$  as a performance predictor is likely to be reduced, increasing the contribution of submaximal parameters (Storen et al., 2013).

## **Conclusion**

Endurance exercise performance is determined by several physiological, mechanical and psychological/perceptual variables. Ultimately, the ability of an individual to tolerate or sustain a high power output during endurance exercise performance is limited by their capability to resist fatigue. This study demonstrates that absolute  $\dot{V}O_{2max}$  (L.min<sup>-1</sup>) was the single best predictor of 30 min cycling time trial performance in a group of moderately trained individuals. Other significant predictors were maximal minute power, body mass, power output at 2 mM.L<sup>-1</sup>

<sup>1</sup> blood lactate, and RPE. In comparison to the traditional physiological parameters, the perception of effort has received limited research attention, but as demonstrated by this study, it is an important predictor of endurance performance. Consequently, future studies should investigate the role of the perception of effort and its role in determining endurance exercise performance.

**CHAPTER 4**

**THE EFFECT OF MENTAL FATIGUE ON  
CRITICAL POWER AND THE ANAEROBIC  
WORK CAPACITY**

## **THE EFFECT OF MENTAL FATIGUE ON CRITICAL POWER AND THE ANAEROBIC WORK CAPACITY**

### **I. Abstract**

Introduction: Time-to-exhaustion (TTE) tests used in the determination of critical power and  $W'$  are strongly influenced by perception of effort (PE). This study aimed to investigate whether manipulation of the PE altered the CP and  $W'$ .

Method: Eleven trained cyclists completed a series of TTE trials to establish CP and  $W'$  under two conditions, one following a cognitive task (incongruent Stroop) involving mental fatigue (MF), or a control (CON) task (reading a magazine). Both cognitive tasks lasted 30 min followed by a TTE test. Ratings of PE and heart rate (HR) were measured during each TTE. Blood lactate was taken pre and post each TTE trial. Ratings of perceived mental and physical fatigue were taken pre- and post-cognitive task, and following each TTE trial.

Results: Perception of MF significantly increased as a result of the cognitive task compared to the baseline and the CON task ( $P < 0.05$ ), without a change in perceived physical fatigue ( $P > 0.05$ ). PE was significantly higher during TTE in the MF condition ( $P < 0.05$ ). Pre-post blood lactate accumulation was significantly lower after each TTE in the MF condition ( $P < 0.05$ ). HR was not significantly different between conditions ( $P > 0.05$ ). Neither cognitive task induced any change in CP (MF  $253 \pm 51$  vs. CON  $247 \pm 58$ W;  $P > 0.05$ ), although  $W'$  was significantly reduced in the MF condition (MF  $2.3 \pm 4.5$  vs. CON  $2.9 \pm 6.3$ kJ;  $P < 0.01$ ).

Discussion: The results of this study demonstrate that MF has no effect on CP, however, it reduces the  $W'$  in trained cyclists. Lower lactate accumulation during TTE trials following MF,



suggests that cyclists were not able to fully expend  $W'$ , even though they exercised to volitional exhaustion.

## II. Introduction

The critical power (CP) concept is based upon a mathematical model of the hyperbolic relationship between work done and tolerable exercise duration. Since the early work of Monod and Scherer (1965), it has been accepted that the hyperbolic relationship is reflective of physiological responses to endurance exercise performance, where CP represents the highest sustainable rate of aerobic metabolism (Gaesser and Wilson, 1988; Hill, 1993). Indeed, Poole et al. (1988) demonstrated that if exercise is performed at the CP, following the initial “fundamental” increase in  $\dot{V}O_2$  a delayed steady state is achieved within a few minutes. Moreover, the increase in  $\dot{V}O_2$  is replicated by the blood lactate response, which following an initial rise at the start of exercise, stabilizes after some minutes. When exercise was performed at 5% above the CP,  $\dot{V}O_2$  continued to increase until  $\dot{V}O_{2\max}$  was reached, and blood lactate continued to rise until the participants were no longer able to exercise. Thus within the hyperbolic work-time relationship, the asymptote is defined as the CP. The curvature constant of the model is known as the  $W'$  and represents the amount of work that can be performed above the CP, regardless of the chosen work rate above CP (Monod and Scherrer, 1965; Moritani et al., 1981; Poole et al., 1988).  $W'$  has been attributed to the depletion of intramuscular PCr and glycogen, with accumulation of fatigue-related muscle metabolites including  $H^+$  and  $P_i$  (Fitts, 1994). Indeed, using  $^{31}P$  MRS, Jones et al. (2008) demonstrated that a knee extension exercise performed 10% below the CP resulted in steady state responses in intramuscular PCr, pH and  $P_i$  being achieved between 1 and 3 minutes after the start of

exercise. However, when exercise was performed 10% above the CP, intramuscular PCr and pH reduced over time until exhaustion was reached. Jones et al. (2008) therefore concluded that the critical power represents a critical threshold for muscle metabolic control, above which exercise tolerance is limited by intramuscular factors, including PCr and concomitant increases in inorganic phosphate and hydrogen ions (Fitts, 1994). Specifically, an acidic pH has been associated with impaired muscle function by inhibiting phosphofructokinase activity and disruption of  $\text{Ca}^{2+}$  release from the sarcoplasmic reticulum, as well as its binding to troponin (Fitts, 1994).

The work-time relationship does not directly measure the physiological parameters that are purported to contribute to its determination. Instead, exercise performance, often measured as the time-to-exhaustion (TTE), is plotted against the externally measured work done (per unit time) to determine the CP and  $W'$ . However, TTE tests are strongly influenced by perception of effort (PE), defined as “the conscious sensation of how hard, heavy and strenuous exercise is” (Marcora, 2009). The psychobiological model of endurance performance has been shown to provide a valid explanation of the effects of both psychological (Marcora et al., 2009; Pageaux et al., 2013) and physiological (Marcora et al., 2008) manipulations on endurance performance during time to exhaustion tests. The psychobiological model (Marcora, 2009) is an effort-based decision-making model based on motivational intensity theory (Brehm and Self, 1989), and postulates that the point of exhaustion during a TTE test is not caused by muscle fatigue, rather the conscious decision to disengage from the endurance task. Thus, in highly motivated individuals, this effort-based decision is taken when their PE is maximal and continuation of the endurance task seems impossible. Recent studies have demonstrated a negative effect of prolonged mental exertion on self-paced endurance tasks (Marcora et al., 2009; Pageaux et al., 2014). Marcora et al. (2009) conducted the first experimental study on the effect of prolonged

mental exertion on exercise endurance performance. The investigators induced mental fatigue in a group of healthy, but untrained participants using a prolonged and demanding cognitive task and found a significant reduction in subsequent high intensity cycling time-to-exhaustion performance. Interestingly, the reduction of TTE performance was independent of alterations in cardio, respiratory, or metabolic responses to exercise, instead being explained by a higher PE experienced by mentally fatigued participants.

Therefore, it is possible to speculate that a higher PE may limit TTE and subsequently alter the CP and  $W'$  independently of changes within the aforementioned underlying muscle physiology. Indeed, Nakamura et al. (2005) have previously demonstrated strong correlations between PE and critical power determined from repeated TTE testing. As a consequence, we hypothesised that mental fatigue from a prolonged and demanding cognitive task would reduce subsequent TTE performance, and therefore alter the CP and  $W'$ .

### **III. Methods**

#### *Participants and Ethical Approval*

Eleven well-trained male cyclists (age  $38\pm 6$  years; body mass  $76.5\pm 9.6$  kg;  $\dot{V}O_{2\text{ peak}}$   $60.5\pm 4.1$  ml.kg<sup>-1</sup>.min<sup>-1</sup>) were recruited to take part in the study. Following institutional ethical approval in line with the Declaration of Helsinki, participants provided written informed consent to participate. All participants were given written instructions describing the procedures related to the study but were naive of its aims and hypotheses. Participants were told that the study was investigating the effect of two different cognitive tasks (computerized task vs reading

magazines) on endurance cycling performance. At the end of the last visit, the participants were debriefed as to the true nature of the study.

### *Study Design*

Each participant visited the exercise testing laboratory on five separate occasions with a minimum of 2 days between visits. On the first visit, the participants completed an incremental exercise test to identify  $\dot{V}O_{2\text{ peak}}$  (see  $\dot{V}O_{2\text{ peak}}$  determination below) and were familiarized with the time to exhaustion tests to be used on subsequent visits. On arrival at the laboratory, the participants were asked provide a capillary blood sample for the determination of resting blood lactate and glucose concentration (Biosen C-Line; EKF Industrie, Elektronik GmbH, Barleben, Germany). At this time, a heart rate monitor (S810i, Polar, Kempele, Finland) was fitted to the participant to record heart rate (HR) continuously throughout the visit. During the experimental visits (see Fig. 11), participants completed two randomly assigned and counterbalanced TTE tests. The TTE tests were performed following either a 30 min cognitively demanding computerized task designed to elicit mental fatigue, or a control task involving 30 min of reading a magazine (see experimental conditions below). After the first TTE, the participants had a 30 min period of passive recovery, before undertaking a further 30 min of the same experimental manipulation prior to completing the second TTE test. TTE tests were conducted at power outputs equivalent to 40, 60, 80% of the difference between the gaseous exchange threshold and  $\dot{V}O_{2\text{ peak}}$ , as well as at the work rate equivalent to  $\dot{V}O_{2\text{ peak}}$  (see TTE testing below). Power outputs (40% $\Delta$  60% $\Delta$ , 80% $\Delta$  or 100%  $\dot{V}O_{2\text{ peak}}$ ) were randomized and divided into two visits per condition (CON & MF). Participants were asked to rate their perceived level of mental and physical fatigue prior to, and following each experimental manipulation, and

after each TTE test. They were also asked to rate their level of motivation immediately prior to each TTE test (see psychological measurements). A blood lactate sample was also taken prior to and following each TTE test.

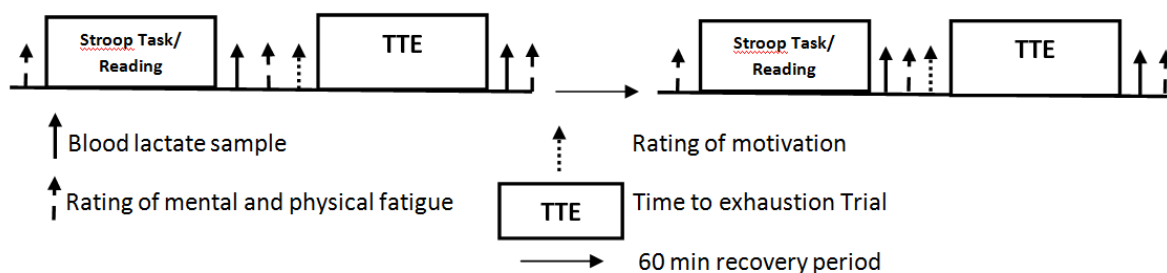


FIGURE 11: An overview of the study protocol.

### $\dot{V}O_{2\text{ peak}}$ determination

Upon arrival at the laboratory, the participant's height and weight (Seca, Hamburg, Germany) were measured. Following a 5 min warm-up at 100 W, the participants completed an incremental exercise test until volitional exhaustion using a 25 W per minute ramp rate. Expired gases were assessed on a breath-by-breath basis using an online gas analyser (Cortex Metalyzer, Cortex Biophysik GmbH, Leipzig, Germany), throughout the test. The participants cycled on an electromagnetically braked cycle ergometer (Schoberer Rad Messtechnik, Jülich, Germany), with power output being recorded continuously throughout the test. Prior to the test, the ergometer was adjusted for each participant, and settings were recorded to allow reproduction at each subsequent visit. The participants were also given standard instructions for the overall rating of PE using the 15-point Borg scale (Borg, 1998). Each participant was then subsequently asked to rate his PE at each minute during the test. HR was monitored continuously throughout the test using a chest strap and watch (S810i, Polar, Kempele, Finland). Throughout the visit laboratory conditions remained stable, and participants were

cooled using electric fans. Following the test,  $\dot{V}O_{2\text{ peak}}$  was identified as the highest  $\dot{V}O_2$  attained during a 60 s period in the test. For setting work rates for the subsequent TTE trials, it was necessary to determine the gaseous exchange threshold (GET) using a cluster of measurements, including: the first disproportionate increase in  $\text{CO}_2$  production ( $\dot{V}CO_2$ ) from visual inspection of individual plots of ( $\dot{V}CO_2$  vs.  $\dot{V}O_2$ ), an increase in expired  $VE/\dot{V}O_2$  with no increase in  $VE/\dot{V}CO_2$ , and an increase in end-tidal  $O_2$  tension with no change in end-tidal  $\text{CO}_2$  tension. Subsequently 40% $\Delta$  (the work rate at GET plus 40% of the difference between the work rate at the GET and the work rate at  $\dot{V}O_{2\text{ peak}}$ ), 60% $\Delta$ , 80% $\Delta$ , and the work rate at  $\dot{V}O_{2\text{ peak}}$  were calculated.

### *TTE Testing*

Participants were positioned on an electromagnetic cycle ergometer (Schoberer Rad Messtechnik, Jülich, Germany) and instrumented for the physiological measurements before starting the TTE test, 15 min after the end of the experimental manipulation. The constant-power cycling TTE test consisted of a 3-min warm-up at 40% of peak power output followed by a rectangular workload corresponding to 40% $\Delta$ , 60% $\Delta$ , 80% $\Delta$  or 100%  $\dot{V}O_{2\text{ peak}}$ . Pedal cadence was freely chosen between 60 and 100 RPM and was recorded continuously throughout each test. Time to exhaustion was measured from the start of the rectangular workload until the pedal cadence was less than 60 RPM for more than 5 s, despite standardized verbal encouragement (Andreacci et al., 2002). Heart rate was recorded continuously throughout all TTE tests, and PE was taken every minute using the Borg 6-20 scale (Borg, 1998). To account for the different exercise durations, HR & PE data were subsequently plotted against TTE with the slope of the relationship being calculated. During all trials participants cycled in an air-conditioned laboratory and were cooled using an electric fan.

*Experimental Conditions*

*Mental Fatigue*

The mental fatigue condition (MF) comprised of 30 min of engagement with an altered incongruent version of the Stroop word-colour task. Participants were seated comfortably in an isolated quiet room while performing the task on a computer. Four different words (yellow, green, blue, red) were consecutively showed on the screen until the participant confirmed an answer, and were followed by a 1,500 ms interval. Participants were instructed to press one of four coloured buttons on the computer keyboard (yellow, green, blue, red) with the correct response being the button conforming to the ink colour (either, yellow, green, blue, red) of the word presented on the screen. For example, if the word 'red' appeared in green ink, the participant should press the green button. If the ink colour was blue, the subject should follow pressing the button linked to the real colour of the word, not the ink meaning (e.g. if the word blue appears in red, the button blue has to be pressed). The computer selected the colours presented in the word randomly (100 % incongruent). To ensure that the task was understood properly, the participants performed 5 min of the task as familiarisation before starting the actual task. Participants were told to answer as accurately and quickly as possible following the presentation of each word. Feedback was presented on the screen after each selection to inform the participants of incorrect or correct answers, accuracy and speed response. E-Prime software (Psychology Software Tools, Pittsburgh, PA, USA) was used to develop the task, and analyse the reaction time and accuracy scores. The HR response was measured throughout the task.

### *Control*

During the control condition (CON), the participants sat quietly and read a fitness magazine for a period of 30 min. During the control condition, the HR was continuously recorded.

### *Psychological Measurements*

The participants were asked to rate “how do you feel right now?” in terms of both mental and physical fatigue before and after each experimental condition, and after each TTE test. The two items were answered on a 10-point scale (1 = not at all, and 10 = extremely). The participants were also asked to rate their motivation status immediately prior to the TTE test. The ten-point scale ranged from 1 = not motivated, to 10 = extremely motivated.

### *Calculation of Critical Power and W'*

Linear regression was used to provide estimates of CP and W' from the four TTE trials using both the work-time [ $P = W' + (CP \cdot t)$ ] models.

### *Statistical analysis*

Assumptions of statistical tests such as normal distribution and sphericity of data were checked as appropriate. A two-way repeated-measures ANOVA (2 condition x 4 work rates) was used to assess differences in recorded TTE, blood lactate, RPE and heart rate slope responses. Differences in perceived mental and physical fatigue, and motivation were also assessed using a two-way repeated-measures ANOVA but with the additional component of measurement



time point (2 condition x 4 work rate x 3 time point). Greenhouse–Geisser correction to the degrees of freedom was applied when violations to sphericity were present. CP and W' data between conditions were analysed by paired t-tests. Statistical analyses were performed using the software program SPSS, version 21.0 (Statistical Package for Social Science, Chicago, Illinois, USA). Statistical significance was accepted at an alpha value of  $P < 0.05$ . All data are presented as means  $\pm$  standard deviation (SD) unless stated otherwise.

#### **IV. Results**

##### *Manipulation Check*

A 30 min incongruent Stroop task was used to cause MF in participants. There was no condition x intensity x time point, or condition x intensity interaction found for perception of mental fatigue ( $P > 0.05$ ). However, a significant interaction effect was found for condition x time point ( $P < 0.01$ ; Fig. 12a). A significant main effect of condition was found; the participants' rating of perceived mental fatigue increased after the MF task compared to the CON task ( $P < 0.01$ ). There was no significant condition x intensity x time point, or condition x intensity ( $P > 0.05$ ). However, a significant interaction effect of the condition x time point ( $P = 0.04$ ) was found with greater levels of perceived physical fatigue being evident after the TTE trial in both conditions (Fig 12b).

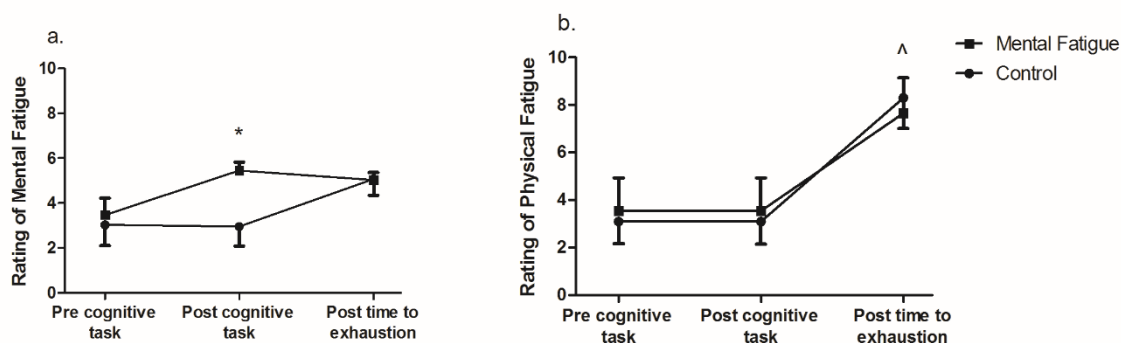


Figure 12: Effect of mental fatigue and control interventions on perception of mental fatigue over all tests (40%Δ 60%Δ, 80%Δ and 100%). \* = significant effect of condition ( $P < 0.05$ ); ^ = significant effect of time point ( $P < 0.05$ ).

There was condition x intensity interaction effect ( $P = 0.79$ ) for perceived level of motivation prior to the TTE tests. Perceived level of motivation not significantly different following MF or CON conditions immediately prior to the TTE trials ( $P = 0.30$ ).

#### *Effects of experimental condition on TTE*

Participant maximal power output from the incremental ramp test was  $394 \pm 47$  W, with the resultant TTE power outputs being  $289 \pm 45$  W,  $326 \pm 47$  W,  $360 \pm 46$  W and  $394 \pm 47$  W 40%Δ 60%Δ, 80%Δ and 100%  $\dot{V}O_{2\text{peak}}$  respectively. There was no interaction effect between condition and intensity ( $P = 0.31$ ), although TTE was significantly reduced following MF at each exercise intensity compared to CON ( $P < 0.01$ ; table 2).

Table 2: Time to exhaustion (s) at 40%Δ, 60%Δ, 80%Δ and 100% for mental fatigue and control conditions. \* = significant shorter than control condition (P <0.05)

TTE (s)	40%Δ	60%Δ	80%Δ	100% $\dot{V}O_{2peak}$
<b>Control condition</b>	720 ± 180	422 ± 88	275 ± 58	190 ± 38
<b>Mental fatigue</b>	648 ± 171*	341 ± 84*	231 ± 65*	156 ± 38*

*Effects of experimental condition on CP and W'*

Critical power was not significantly different between MF and CON conditions (MF 253 ± 51W vs. CON 247 ± 58W; P>0.05; see Figure 13a). However, there was a significant reduction in estimated W' following MF (MF 22.8 ± 4.5 kJ vs. CON 29.3 ± 6.3 kJ; P<0.01; see Figure 13b).

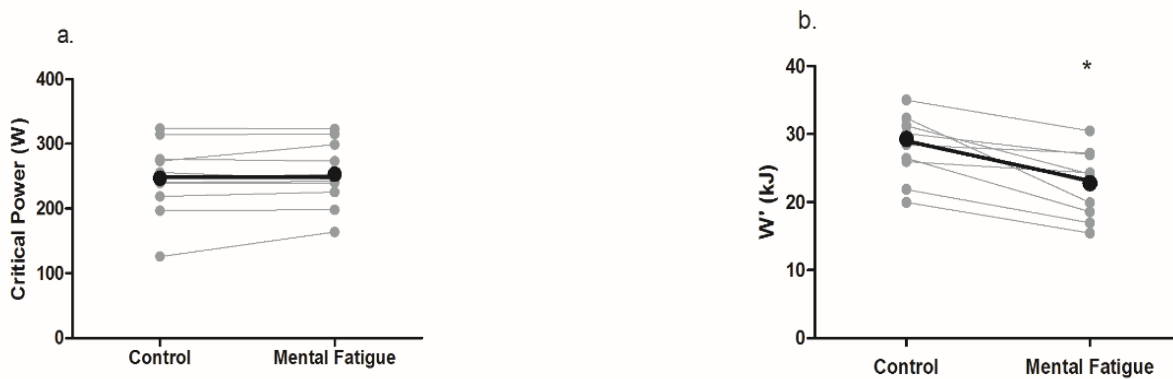


Figure 13: Effect of Mental Fatigue and Control Conditions on a) CP and b) W' calculated using the work-time model. \* = significant effect of condition (P <0.05).

*Effects of experimental condition on blood lactate, PE and Heart rate*

There was no condition x intensity interaction effect for the pre-post change in blood lactate measured from each TTE trial ( $P = 0.36$ ). However, a significant main effect was evident, with blood lactate concentrations being significantly lower in the MF compared to the CON condition ( $P = 0.03$ ; Figure 14). There was no significant effect of TTE intensity ( $P = 0.62$ ).

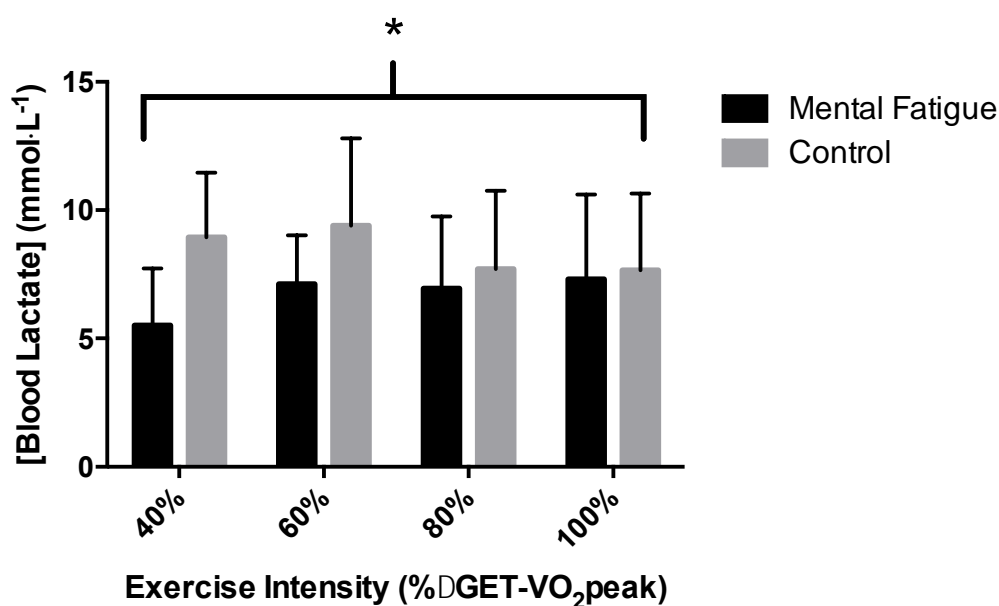


Figure 14: Blood lactate response during time to exhaustion trials used in the determination of CP and AWC. \* = significant effect of condition ( $P < 0.05$ ).

A significant interaction effect of condition x intensity was evident for the PE slope ( $P = 0.03$ ; Figure 15). The slope of the PE response was significantly greater in the MF vs CON condition ( $P = 0.03$ ).

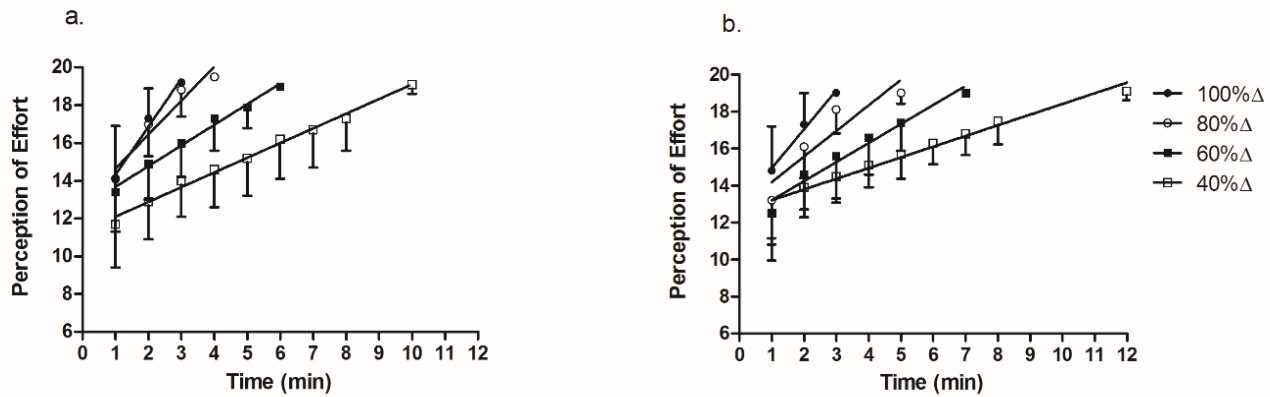


Figure 15: Slope of RPE response during time to exhaustion trials used in the determination of CP and AWC following a. mental fatigue and b. the control condition.

There was no interaction effect between condition x intensity for the slope of the HR response ( $P = 0.40$ ). The slope of the HR response was not significantly different between the MF and CON conditions ( $P = 0.31$ ). The maximal HR and PE recorded during each TTE were not different between conditions ( $P > 0.05$ ).

## V. Discussion

To our knowledge this is the first study to demonstrate that the  $W'$  can be altered by purely psychological factors. Specifically, we used a prolonged cognitive task to induce mental fatigue, and thus reduced time-to-exhaustion during fixed intensity cycling trials used to construct the work-time relationship. The shorter time to exhaustion did not alter the slope of the relationship, hence CP was not affected, but the intercept was lower following MF, resulting in a reduced  $W'$ .

The current study utilised a period of 30 mins of the incongruent Stroop task. The participant's greater perception of MF following the Stroop task compared to the CON task (Figure 12a) attests to its more demanding nature. This manipulation check suggests that the 30 min Stroop task was successful in inducing a higher level of mental fatigue, without changing perceived physical fatigue (Fig 12b), compared to the CON condition. Supporting the findings of previous research (Marcora et al., 2009; Pageaux et al., 2014), we found a significant impairment of exercise performance with MF in our group of trained cyclists. Specifically, time-to-exhaustion was reduced by an average of ~15% across exercise intensities equivalent to  $\Delta 40$ ,  $\Delta 60$ ,  $\Delta 80$ , and 100%  $\dot{V}O_{2\text{peak}}$  (see table 2). These findings are also in agreement with the findings of Bray et al. (2008), who demonstrated that as little as 220 s of mental exertion involving response inhibition could reduce the endurance capacity of the muscles involved in a hand grip exercise. However, even though we found a reduced time to exhaustion performance, this did not alter the slope of the work-time relationship, used to estimate CP (Fig 13a). From a mathematical perspective, this is likely due to the reductions in time to exhaustion being proportional across the different time to exhaustion durations. From a physiological perspective, it is plausible to suggest that MF does not affect the purportedly 'aerobic' component of the work-time relationship (i.e. the CP). However, the reduced time to exhaustion did affect the  $W'$  (intercept of the work-time relationship, Fig 13b), due to participants disengaging from the TTE trial sooner in the MF vs. CON condition (Fig 13). Previous research suggests that this disengagement was not due to mental fatigue altering the underlying physiological processes of muscle fatigue associated with time-to-exhaustion exercise (Marcora et al., 2009; Pageaux et al., 2014), rather the subjects experiencing a higher than normal perception of effort in the MF condition (see Figure 15). Thus, the higher than normal PE likely reduced the fixed amount of work that participants could perform above the CP before task disengagement occurred, regardless of the rate at which the work was done.

The level of participant potential motivation was not significantly different immediately prior to the TTE trials in both the MF and CON conditions. Therefore, to understand the negative effect of the MF intervention on the short-term high intensity cycling TTE performance, it is important to consider the PE. In this regard, findings from previous research have demonstrated impairment in endurance performance without alternations in cardiorespiratory, metabolic and neuromuscular responses to the exercise (Marcora et al., 2009; Pageaux et al., 2013; Pageaux et al., 2014). These studies suggest that following a period of prolonged cognitive exertion (such as the incongruent Stroop task), adenosine accumulation in the anterior cingulate cortex of the brain provides a mechanism for increased higher perception of effort and reduced exercise performance in subsequent self-paced TTE trials (de Morree et al., 2012). Indeed, results of the current study demonstrate that the slope of the PE response increased significantly over the different TTE intensities in both conditions, but were greatest following MF (Fig 15a and b). Mentally fatigued participants experienced a quicker rise in PE, meaning that they reached their maximal level of perceived exertion sooner and disengaged from the TTE trial earlier than in the CON condition. Indeed, in support of Brehm's theory of motivation (Wright, 2008), participants withdrew effort (i.e. disengaged) when the task was perceived to be too difficult. Therefore the increased PE and reduced TTE are likely to impact upon the mathematical work-time relationship used to estimate the CP. Interestingly, Nakamura et al. (2005) propose that PE and CP are "indirectly" related, and that a "PE threshold" (derived from linear extrapolation of the relationship between exercise intensity and perceived exertion rate), can be used to determine CP derived from a series of TTE trials. Therefore altering the work-time relationship by increasing PE (due to MF) is also likely to change the estimate of CP. However, our results do not support the correlational findings of Nakamura et al. (2005), in that we did not find any change in CP by using MF to experimentally manipulate the PE. Instead, participant early disengagement from the TTE trial following MF meant that they were unlikely to have

expended their entire  $W'$ , even though they exercised until volitional exhaustion. This hypothesis is supported by the blunted blood lactate response seen during the TTE trials following MF. Therefore, by increasing the PE and reducing TTE, it is possible to affect the critical power concept that has previously only been described using physiological parameters. As a consequence, this data suggests that models of endurance performance, which purely focuses on peripheral mechanisms (as in the CP concept), fail to encapsulate all of the determinants of performance during prolonged or sustained exercise. Moreover, because 30 min of mental exertion increased the perception of effort and reduced TTE performance, scientists, athletes and coaches should avoid cognitive tasks involving response inhibition prior to assessing critical power. In addition, endurance athletes should avoid prolonged periods of mental exertion prior to training and competition.

## **Conclusions**

The critical power concept is defined by the mathematical relationship between work done and TTE within the severe-intensity exercise domain, and represents a threshold above which the individual progressively expends the  $W'$ . However, results from this study suggest that the parameter of  $W'$  can be altered by prior MF. This reduced  $W'$  was associated with a shorter time to exhaustion during the constant intensity exercise trials used in the determination of the critical power. Furthermore, this research study demonstrates that the higher than normal PE due to MF reduced the participant's TTE, as they reached the maximal PE in a shorter period of time. Thus, notwithstanding any MF induced alteration in the normal muscle fatigue processes associated with TTE testing, it is possible that participants disengaged from the constant work rate trials prior to fully expending their  $W'$ . The findings of our research



therefore suggest that attempting to constrain the mathematical work-time relationship by physiological parameters seems problematic, and overly restrictive.

## **CHAPTER 5**

# **THE RELIABILITY OF RATING OF PERCEIVED EXERTION VIA CAFFEINE INGESTION DURING HIGH-INTENSITY INTERVAL TRAINING**

## **THE RELIABILITY OF RATING OF PERCEIVED EXERTION VIA CAFFEINE INGESTION DURING HIGH-INTENSITY INTERVAL TRAINING**

### **I. Abstract**

Introduction: Athletes often use caffeine supplementation in an attempt to enhance exercise performance. However, whilst there is evidence documenting its ergogenic properties, no research exists that has considered the reliability of the effect of caffeine during self-paced exercise performance.

Methods: On three separate occasions, nine recreationally active male participants (age:  $26.5 \pm 7.8$ /yrs; body mass  $76.3 \pm 4.4$ /kg;  $\dot{V}O_{2peak}$   $53.2 \pm 8.6$ / ml.kg<sup>-1</sup>.min<sup>-1</sup>; PO:  $322 \pm 41.3$ /W) ingested caffeine (3 mg.kg<sup>-1</sup>), one hour before completing four bouts of self-paced exercise equivalent to a rating of perceived exertion (RPE) of 16, 17, 18, and 19, of 4 min each, with 3 min active recovery between each. Power output, HR and cadence were continuously recorded throughout all exercise trials.

Results: Statistical analysis demonstrated that participants were able to reliably reproduce self-paced exercise equivalent to RPEs of 16, 17, 18 and 19. Specifically, following caffeine ingestion a mean coefficient of variation (CV) across all RPE intensities of 2.10% (05% CL = 1.03 - 3.61%) was found for cycling power output, 1.6% (95% CL = 0.60 – 3.77%) for cycling cadence, and 1.40% (95% CL = 0.51 - 2.51%) for heart rate. A repeated measures ANOVA demonstrated no significant difference in power output, cadence or heart rate across the three repeated trials across all RPE intensities.

Discussion: The results of this study demonstrate that following caffeine ingestion, cycling power output and cadence used during self-paced exercise at fixed RPEs can reliably be

reproduced. Moreover, the reliability of the self-paced work rate is reflected in a good reliability of the associated exercise heart rate response.

## **II. Introduction**

The rating of perceived exertion is a concept that is widely accepted as a method to estimate exercise intensity due to its strong positive association with physiological variables such as oxygen uptake, heart rate, and blood lactate. On the basis of empirical evidence, early studies by Skinner et al. (1973) and Stamford (1976) are often presented as evidence that supports the reliability of the RPE scale. Data from these works report test-retest correlations of 0.71 to 0.90, depending on the exercise mode and whether the protocol was continuous or incremental. However, more recent research has begun to question the efficacy of using RPE during exercise (Lamb et al., 1999; Whaley et al., 1997). Indeed, Lamb et al. (1999), investigated the test-retest reliability of incremental treadmill running in trained male athletes, and reported that 95% of limits of agreement differed by as much as three RPE units. Given this conflicting evidence in the literature, it is perhaps advisable to investigate the test-retest reliability of the use of RPE prior to an experimental study reliant on self-paced exercise at a fixed RPE.

A central aspect of the work in this thesis is the manipulation of the perception of effort. In the previous chapter, the perception of effort was increased via the use of experimentally induced mental fatigue. In the following chapter, caffeine was used to reduce the perception of effort during self-paced exercise, or more specifically to increase power output at a fixed RPE. Indeed, previous research has demonstrated that regardless of intensity, mode, or duration of exercise, caffeine ingestion is able to alter an individual's perceptual response. This alteration has been evident as either a reduced perception of effort during fixed intensity exercise

(Birnbaum and Herbst, 2004; Costill et al., 1978; Doherty et al., 2002, 2004; Giles and Maclaren, 1984; MacIntosh and Wright, 1995), or an increased work output at a fixed RPE (Casal and Leon, 1985; Cole et al., 1996; Ivy et al., 1979; Plaskett and Cafarelli, 2001).

Caffeine acts as a stimulant for both physical and mental performance (Delbeke and Debackere, 1984; Graham, 2001), and has been used as an ergogenic aid by many athletes within the endurance sports community (Burke, 2008; Desbrow and Leveritt, 2006). Indeed, the literatures suggests that supplementation with moderate ( $\sim 3\text{-}6 \text{ mg}\cdot\text{kg}^{-1}$ ) and high doses ( $\sim 6\text{-}13 \text{ mg}\cdot\text{kg}^{-1}$ ) of caffeine can reduce the perception of effort and enhance moderate to high intensity endurance exercise performance (Burke, 2008; Doherty and Smith, 2004; Graham, 2001). As well as reportedly exerting an effect at the local muscle level (Erickson et al., 1987; Essig et al., 1980; Spriet et al., 1992), caffeine has been shown to influence nervous system function (Cole et al., 1996). Indeed, caffeine has been shown to reduce the perception of effort during constant load exercise (Cole et al., 1996), and attenuate central fatigue by inhibition of central adenosine receptor activation, thereby increasing the dopamine serotonin ratio in the brain (Davis et al., 2003; Fredholm, 1995; Lynge and Hellsten, 2000). In this regard, a meta-analysis has established that caffeine consumption reduces perceived exertion by an average of 5.6% compared with a placebo during constant load exercise, resulting in an improved exercise performance of 11.2% (Doherty and Smith, 2005). Regression analysis suggested that perceived exertion during exercise accounted for 29% of the variance in the improvement in exercise performance induced by caffeine supplementation. However, the 95% confidence interval for the improvement in performance established via Doherty and Smith's meta-analysis was 4.6 to 17.8%, suggesting a range of variability in the way in which individuals respond to caffeine ingestion (Graham et al., 2001). To the author's knowledge, there is also no

evidence concerning the reliability of the ergogenic effect of caffeine during exercise. Therefore, the aim of the present study was to examine the reliability of self-paced exercise at a fixed rating of perceived exertion during high intensity interval exercise, following the ingestion of 3 mg.kg<sup>-1</sup> caffeine.

### **III. Methods**

Nine recreationally active male participants (age: 26.5 ± 7.8 yr; body mass 76.3 ± 4.4 kg;  $\dot{V}O_{2peak}$  53.2 ± 8.6 ml.kg<sup>-1</sup>.min<sup>-1</sup>; PO: 322 ± 41.3 W) volunteered to take part in the study. Following institutional ethical approval of the study (protocol number 140-2014\_2015), participants were provided with written informed consent to participate. Prior to the start of the study, a list of food and drinks containing caffeine which were to be avoided in the 48 hours before each test was given to the participants. The participants were also asked about their habitual caffeine use. Prior to testing, the participants were reminded of the instructions on the use of the RPE scale (Noble and Robertson, 1996). The subjects had to:

1. Understand the definition of RPE and receive an explanation of the nature and use of the scale.
2. “Anchor” the top and bottom perceptual ratings to previously experienced sensations of the easiest and most difficult exercise encountered.
3. Ensure they gave an “all-over,” integrated rating, which included both muscular and cardiorespiratory sensations.

#### *Experimental Study Design*

Each participant visited the laboratory on 3 separate occasions to undertake an interval cycling protocol (see *Experimental Procedures* below). All visits were scheduled at the same time of day for each participant, with a minimum of 2 days separating the test sessions. For each

subject, all tests were completed within a 3-week period. One-hour prior to the start of exercise, the participants consumed 3 mg.kg<sup>-1</sup> mass of caffeine contained within a gelatine capsule.

### *Experimental Procedures*

The effort-based cycling trials were completed on an air-braked cycle ergometer (Wattbike Trainer, UK), which allowed participants to continually vary their PO throughout the session. This was achieved by the participant manually adjusting the air brake in order to produce a level of resistance that allowed them to match the target RPE for each interval. The participants initially completed a standardised warm-up consisting of a 5-min warm-up at 100 W. The participants then completed the cycling trial, which consisted of 4 x 4-min intervals, with 3 min unloaded active recovery between each interval (total exercise time of 25-min), where for each interval, the participants were able to continuously vary their PO, however, the RPE (Borg's 6-20 scale; Borg, 1988) was fixed to a level for each interval of 16, 17, 18, and 19, following an incremental format. Changes in power output were facilitated by the participants manually. The participants were blinded to the actual power output that they were cycling at during each visit, in order that they were not able to either consciously or subconsciously try to match power output and cadence during each visit. Cycling power output, cadence and heart rate (S810i, Polar, Kempele, Finland) were monitored continuously throughout the cycling bouts during each visit.

### *Statistical Analysis*

The individual typical error was expressed as a coefficient of variation (CV) and Intra-class correlation coefficient (ICC), calculated separately from the power output, cadence and heart rate data, for each participant at each RPE intensity across each of the three visits. The mean data across all three trials at each RPE intensity was assessed using the root mean square error. The confidence intervals (95% CI) of the CV and ICC, and 95% limits of agreement were calculated per participant to assess the variability of the repeated tests (Hopkins, 2000). Comparisons of the power output, cadence and heart rate across repeated tests were assessed using a repeated measures ANOVA. Statistical significance was set at 95% confidence ( $P < 0.05$ ). All data are presented as means  $\pm$  standard deviation (SD) unless stated otherwise.

## **VI. Results**

### *Power output*

The mean power output results for the group of participants were  $154 \text{ W} \pm 6 \text{ W}$ ,  $165 \text{ W} \pm 5 \text{ W}$ ,  $175 \text{ W} \pm 4 \text{ W}$ , and  $191 \text{ W} \pm 5 \text{ W}$  for work rates of self-paced exercise at fixed ratings of perceived exertion equivalent to 16, 17, 18, and 19 on the Borg 6-20 scale, respectively. Reliability data are presented in Table (3). A repeated measures analysis of variance at each RPE level revealed a non-significant value at power output, heart rate, and cadence ( $P > 0.05$ ). Because there was no evidence of a change in power output across the three repeated trials, a single CV was derived using the root mean square error from the ANOVA. This process provided a typical error expressed as a CV of 2.1% (95% CL = 1.03 – 3.61%), with limits of agreement being  $\pm 10.75\%$ . Figure (16) illustrates the agreement in power output between the



three repeated trials across all RPE intensities following caffeine supplementation. On a basis of an RPE of 17, limits of agreement were  $\pm 8\%$ .

*Table 3: Reliability statistics for power output across all RPE intensities, and three repeated tests for all participants (n = 9). Data are mean, plus a 95% confidence interval in parenthesis.*

RPE	ICC	CV (%)	95LoA
16	0.92 (0.80-0.98)	3.43 (1.18-5.11)	16 (-13-18)
17	0.98 (0.94-0.99)	1.71 (0.91-2.62)	8 (-7-8)
18	0.96 (0.88-0.99)	2.07 (1.32-3.44)	9 (-7-10)
19	0.96 (0.88-0.99)	1.64 (0.74-3.27)	10 (-9-11)

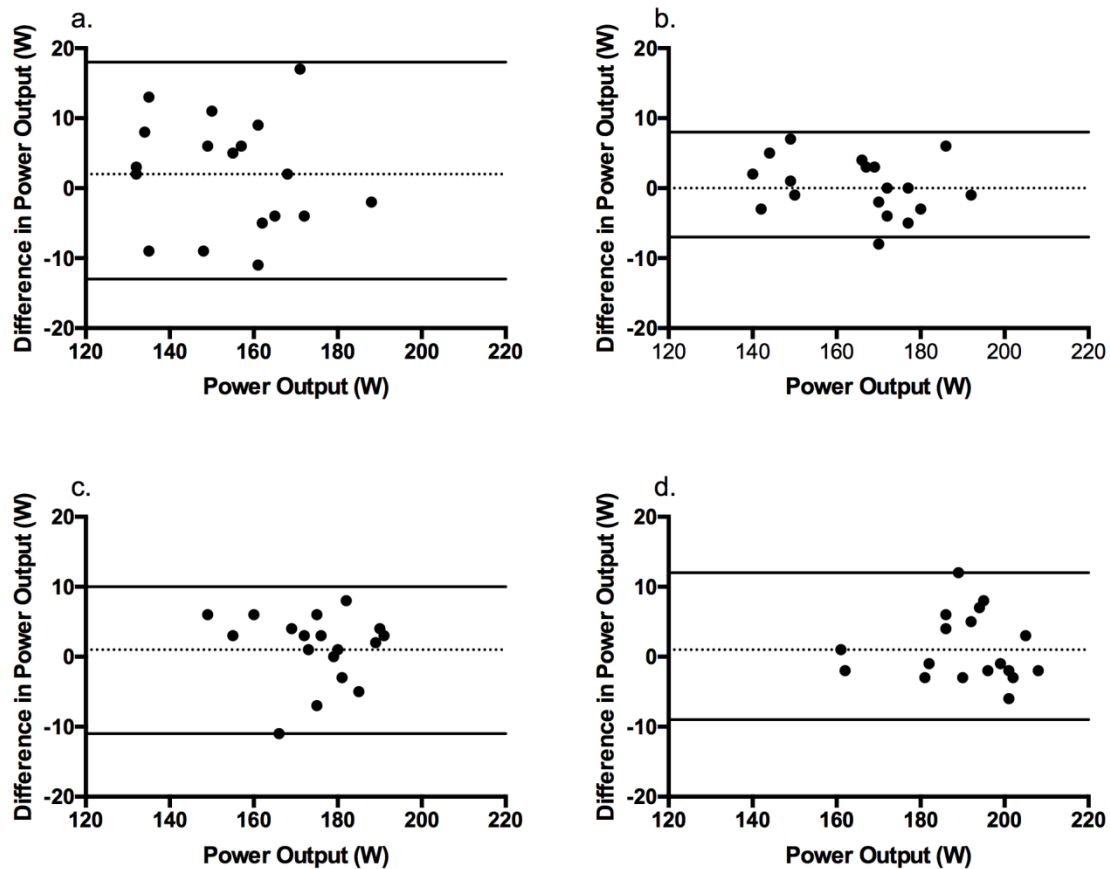


Figure 16: Agreement between repeated tests of PO at RPE intensities of: a. 16; b. 17; c. 18; d. 19.

### *Cadence*

The mean cadence results for the group of participants were  $86 \pm 3 \text{ rev}\cdot\text{min}^{-1}$ ,  $91 \pm 1 \text{ rev}\cdot\text{min}^{-1}$ ,  $92 \pm 1 \text{ rev}\cdot\text{min}^{-1}$ , and  $94 \pm 1 \text{ rev}\cdot\text{min}^{-1}$  for a self-paced exercise work rate equivalent to the fixed RPEs of 16, 17, 18, and 19, respectively. Table (4) illustrates reliability coefficients and limits of agreement for cadence data. A repeated measures ANOVA demonstrated that there was no significant difference in the cadence measures across trials ( $P > 0.05$ ). A mean group CV of 1.6 % (95% CL = 0.60 % – 3.77 %) across all four RPE intensities was found. Figure

(17) illustrates the agreement in cadence between the three repeated trials across all RPE intensities following caffeine supplementation.

*Table 4: Reliability statistics for cycling cadence across all RPE intensities, and three repeated tests for all participants (n = 9). Data are mean, plus a 95% confidence interval in parenthesis.*

RPE	ICC	CV (%)	95LoA
16	0.84 (0.89-0.96)	1.38 (0.63-4.95)	4 (-5-4)
17	0.88 (0.71-0.96)	1.81 (0.62-2.79)	5 (-5-5)
18	0.81 (0.55-0.94)	1.67 (0.61-3.37)	5 (-5-5)
19	0.84 (0.62-0.95)	1.57 (0.57-4.00)	5 (-5-5)

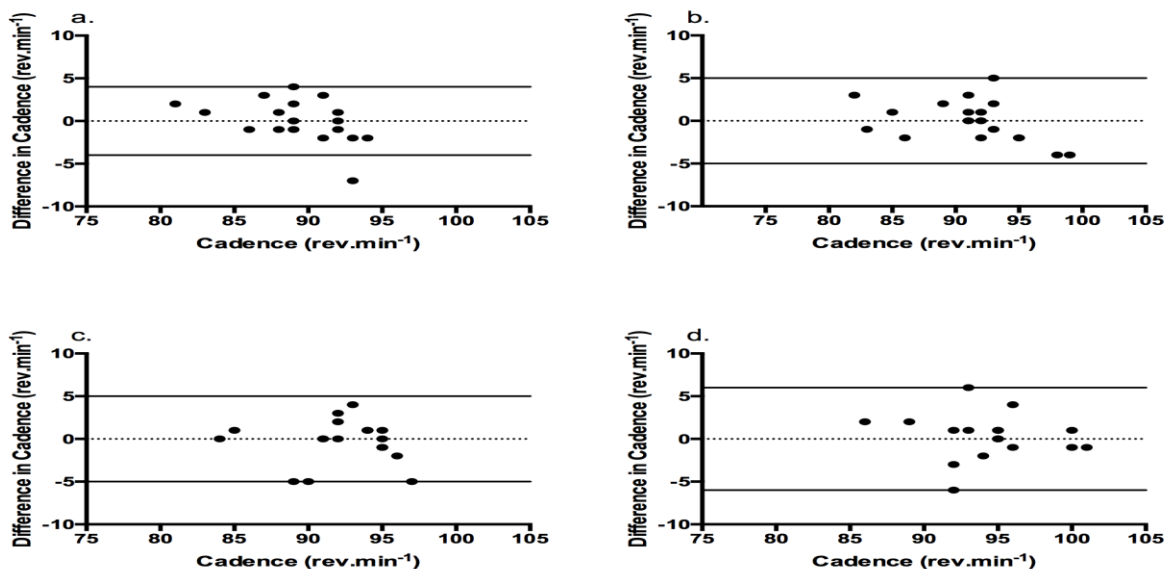


Figure 17— Agreement between repeated tests of cycling cadence at RPE intensities of: a. 16; b. 17; c. 18; d. 19.

*Heart rate*

The mean heart rate results for the group of participants were  $153 \pm 4$  beats.min<sup>-1</sup>,  $162 \pm 4$  beats.min<sup>-1</sup>,  $170 \pm 3$  beats.min<sup>-1</sup>, and  $176 \pm 6$  beats.min<sup>-1</sup> during self-paced exercise at fixed RPEs of 16, 17, 18, and 19, respectively. Table (5) illustrates reliability coefficients and limits of agreement for heart rate data. A repeated measures analysis of variance ANOVA demonstrated that there was no significant difference in the heart rate measures across trials ( $P > 0.05$ ). A mean group CV of 1.40% (95% CL = 0.51% – 2.51%) was found across all four RPE intensities. Figure (18) illustrates the agreement in heart rate between the three repeated trials across all RPE intensities following caffeine supplementation.

*Table 5: Reliability statistics for heart rate across all RPE intensities, and three repeated tests for all participants (n = 9). Data are mean, plus a 95% confidence interval in parenthesis.*

RPE	ICC	CV	95LoA
16	0.96 (0.98-0.99)	1.85 (0.4-2.48)	9 (-9-9)
17	0.96 (0.90-0.99)	1.4 (0.34-2.64)	7 (-6-8)
18	0.98 (0.93-0.99)	1 (0.56-1.69)	5 (-4-5)
19	0.91 (0.77-0.97)	1.33 (0.8-3.22)	7 (-7-8)

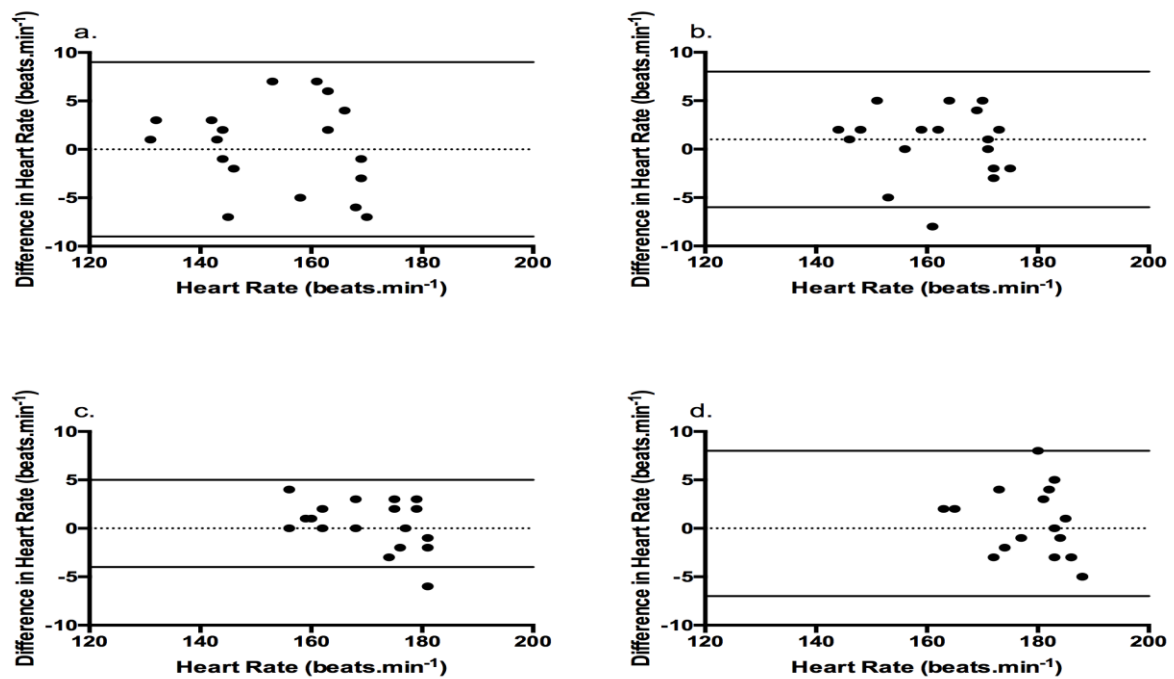


Figure 18— Agreement between repeated tests of heart rate at RPE intensities of: a. 16; b. 17; c. 18; d. 19.

## V. Discussion

The purpose of the current study was to examine the reliability of self-paced exercise based upon fixed RPE intensities following  $3\text{mg}\cdot\text{kg}^{-1}$  of caffeine ingestion. Previous literature has demonstrated a clear intra-individual variation in the magnitude of ergogenic response to caffeine (Astorino and Robertson, 2010), in that some individuals respond to caffeine, whereas others do not, although the mechanisms explaining this are unknown. The reliability coefficients in Tables 3, 4, and 5 present a total within-subject variation of 2.1% for power output, 1.6% for cadence and 1.4% for heart rate. The novel aspect of this study was that the exercise response following caffeine ingestion was reliable (Hopkins, 2000).

The reliability of power output at fixed RPEs is lower than previously reported by Wallace et al. (2014), who investigate the reliability of the RPE at fixed power outputs equivalent to 35, 50 and 65% of maximal cycling power output from an incremental cycling test. They found a mean within subject CV of 8.8% (95% CI = 7.4 – 10.8%). Moreover, in contrast to the work of Lamb et al. (1999), the current research did not find a reduction in the level of reliability as absolute exercise intensity increased across the different stages of the test. Adopting a “worst case scenario” approach to interpreting the LoA analyses (Nevill and Atkinson, 1997), an individual in this study reporting cycling at an RPE of 17 in trial 1, could possibly have produced a power output as high as (8/W) or as low as (-7/W) during the same stage a few days later.

In terms of test-retest correlations, the current study also reports higher values than are previously evident in the literature at each RPE intensity for power output (RPE 16:  $r = 0.99$ , RPE 17:  $r = 0.98$ , RPE 18:  $r = 0.78$  and RPE 19:  $r = 0.67$ ). For example, during repeated time to exhaustion treadmill running trials, Doherty et al. (2001) reported a coefficient of variations in the RPE of between 4.4 and 6.0%, with ICCs of between 0.78 and 0.87. Lamb et al. (1999) report an overall test-retest correlation coefficient of  $r = 0.86$  and Skinner et al. (1973), present a lower value of  $r = 0.80$ . Skinner et al. (1973) suggest that their findings reflect a “sufficiently high reliability” of RPE during incremental exercise. However, as pointed out by Noble and Robertson (1996), a correlation coefficient of 0.80 still suggests that this is approximately 36% of unexplained variance in the relationship. Stamford (1976) investigated the reliability of RPE during treadmill walking and jogging, cycling and stool stepping at various intensities. Stamford suggested that there was a good test-retest reliability in his data by reporting correlations of  $r = 0.90$  during cycling. However, it does not seem that the data were collected in an identical fashion during the repeated trials in each exercise mode, and reliable data is not

presented for all modes. It is therefore difficult to fully interpret and compare the findings of Stamford's study to the current investigation. It is difficult to provide a reason as to why results from the current study suggest a higher reliability than those previously reported in the research literature. One possible reason is due to the difference in study design between the current study, and those outlined above. Specifically, the current study fixed exercise intensity at a given RPE and measured the reliability of cycling power output, cadence and heart rate. By contrast, the previously published literature fixed the external work rate, and then asked participants to rate their perceived exertion. It is possible that this subtle difference in study design might have contributed to the improved test-retest reliability established in the current study.

To date, only 2 studies have used a test-retest design to investigate the consistency of the effects of caffeine, although both were from an exercise performance perspective (Astorino et al., 2012; Paton et al., 2010). Paton et al. (2010) required male cyclists to perform repeated 30 second sprints following the ingestion of caffeine (240 mg as a chewing gum), or a placebo. On two repeated trials in each condition, data revealed a lower decline in mean cycling power output in the caffeine vs. placebo trial. Astorino et al. (2012) investigated the repeatability of the ingestion of  $5 \text{ mg}\cdot\text{kg}^{-1}$  caffeine in a drink on 2 separate occasions vs. a placebo on a 10 km cycling time trial performance. In both caffeine trials, caffeine significantly increased cycling performance (+1.6% and +1.9%), compared to the placebo condition ( $P = 0.02$ ). The heart rate was consistently higher in the caffeine condition ( $P < 0.01$ ), although the RPE was similar ( $P = 0.65$ ). Unfortunately, no reliable statistics are provided for any of the physiological or performance variables (except performance time:  $\text{ICC} = 0.97$ ) measured in Astorino's study, so

it is difficult to provide a comparison to the current study, except to say that the ergogenic effect of caffeine was consistent across repeated trials.

Unfortunately, it was not possible to obtain blood samples to quantify the plasma caffeine concentration prior to each visit. Moreover, it was not possible to ensure that participants had abstained from caffeine consumption (as requested), prior to each visit. These are two clear limitations of the current study, and must be taken into consideration when interpreting the results. However, the good level of reliability demonstrated in the data suggests that the participants did prepare consistently prior to each testing visit, and the caffeine absorption was likely similar following ingestion across the repeated trials. It has been previously suggested that nutritional status may affect the potential ergogenic effects of caffeine by changing the absorption rate (Skinner et al., 2010). However, dietary intake and exercise status were carefully controlled in this study to ensure that participants were in an identical state before all trials.

## **Conclusion**

The results from the current study demonstrate that power output, cadence and heart rate show high levels of reliability during self-paced exercise at fixed RPE intensities when identical doses of caffeine are ingested on separate days. The implications of this finding are that the 6-20 RPE scale may be a useful tool for prescribing and self-regulating high intensity exercise following caffeine ingestion.



## **CHAPTER 6**

# **DOES CHRONIC USE OF CAFFEINE REDUCE ITS ACUTE ERGOGENIC EFFECTS DURING HIGH INTENSITY INTERVAL TRAINING?**

## **DOES CHRONIC USE OF CAFFEINE REDUCE ITS ACUTE ERGOGENIC EFFECTS DURING HIGH INTENSITY INTERVAL TRAINING?**

### **I. Abstract**

Introduction: Many athletes use caffeine before and during competitions however, the chronic use of caffeine during training is not well understood. Therefore, the aim of this study was to investigate the acute effects of caffeine on high intensity interval training (HIIT) and how these were affected by chronic caffeine intake.

Methods: Using a randomized, counterbalanced and double-blinded placebo-controlled design, twenty recreational male endurance athletes (age  $33 \pm 9$  years;  $\dot{V}O_{2\max}$   $55.3 \pm 8.9$  ml.kg<sup>-1</sup>.min<sup>-1</sup>) ingested either caffeine (3 mg.kg<sup>-1</sup> body mass), or a placebo one hour before a HIIT session. HIIT consisted of 4 x 4 min bouts (RPE 16, 17, 18, and 19) of exercise, with 3 min active recovery between each. Power output, HR and blood lactate were recorded throughout each HIIT session. Subsequently, participants were randomly allocated to either ingest caffeine (3 mg.kg<sup>-1</sup> body mass) or placebo capsules three times a week, one hour before regular training sessions. After 4 weeks of chronic supplementation, all participants performed a follow-up HIIT assessment using the same study design, as outlined above.

Results: During HIIT sessions, caffeine increased power output, HR and blood lactate at both the baseline and follow-up assessments ( $P < 0.05$ ). After 4 weeks of caffeine or placebo ingestion, there were no differences between groups in their power output, HR or blood lactate responses during HIIT following acute caffeine or placebo ingestion ( $P > 0.05$ ).

Discussion: Caffeine ingestion one hour prior to HIIT acutely increases power output, HR, and blood lactate for exercise at the same RPE. The frequent ingestion of caffeine during training does not reduce its ability to enhance power output for the same level of effort during HIIT.

## **II. Introduction**

Caffeine is widely used in sport due to its ability to improve both aerobic endurance (Costill et al., 1978, 1979, 1980; Ivy et al., 2009) and anaerobic sprint-type (Astorino, 2011) exercise performance. Purported mechanisms of caffeine include effects on plasma norepinephrine (Fisher et al., 1986), reduction in muscle glycogen (Erickson et al., 1987; Essig et al., 1980; Spriet et al., 1992), and increased fat utilisation (Essig et al., 1980; Flinn et al., 1990; Ivy et al., 1979). However, there are also a significant number of studies that do not support the notion of carbohydrate sparing and the increased fat utilisation effect of caffeine (e.g. Cox et al., 2002; Graham and Spriet, 1991; Laurent et al., 2000). Indeed, following caffeine ingestion, improved high intensity exercise performance has been demonstrated where muscle glycogen depletion is not the primary cause of fatigue (Jackman et al., 1996; McNaughton, 1986). It is therefore likely that caffeine also exerts effects on performance via its effect on nervous system function. Indeed, caffeine has been shown to reduce the perception of effort during constant load exercise (Cole et al., 1996), and attenuate central fatigue by the inhibition of central adenosine receptor activation and thereby increase the dopamine serotonin ratio in the brain (Davis et al., 2003; Lim et al., 2001). Alternatively, caffeine may reduce the neuron activation threshold (Waldeck, 1973), meaning that a larger number of motor units are recruited and thereby spread the tension requirement across a larger muscle mass (Van Handel, 1983). In turn, this greater distribution of work would alter the perception of effort during constant work exercise.

Regardless of the exact mechanism, a reduction in the perception of effort has been demonstrated by an increase in the rate of work output during exercise at a fixed rating of perceived exertion (RPE) (Cole et al., 1996; Ivy et al., 1979; Plaskett and Cafarelli, 2001), or more naturally, a reduction in RPE during fixed work rate exercise (Birnbaum and Herbst, 2004; Casal and Leon, 1985; Costill et al., 1978; Doherty et al., 2002, 2004; Giles and Maclaren, 1984; MacIntosh and Wright, 1995). Thus, in the context of high intensity interval training (HIIT), it is possible that caffeine use has the potential to increase the amount of work that an individual could perform within a given time period.

However, there is debate over the effects of chronic caffeine intake, and whether there is an increased tolerance to the pharmacological effects of caffeine following prolonged use. Indeed, an early study by Dodd et al. (1993) demonstrated no differences in the effect of caffeine on exercise performance between habitual and naïve caffeine users. More recently Goncalves et al. (2017), have also demonstrated that 30 min cycling time trial performance following acute caffeine ingestion (vs. a placebo) was not affected by the habitual level of caffeine consumption by their study participants. In contrast, Bell et al. (2002), confirmed that time to exhaustion at 80%  $\text{VO}_{2\text{max}}$  was significantly longer following caffeine ingestion in non-habitual vs. habitual caffeine users. However, these cross-sectional studies are limited in their methodological approach due to their arbitrary divisions of study participants (e.g. low vs. high, or low vs. moderate vs. high) based on the level of habitual caffeine use. Although, interestingly, a recent study (Beaumont et al., 2016) using a longitudinal study design has demonstrated that 4 weeks of caffeine ingestion ( $1.5\text{-}3.0 \text{ mg}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$ ; titrated) in habitually low caffeine users, resulted in a reduction in the acute ergogenic effect of caffeine during a 30 min cycling performance trial. However, unfortunately the study did not incorporate a post-supplementation placebo trial, and so a direct comparison between caffeine and placebo

conditions after the period of chronic supplementation is not possible. Thus, it is not possible to rule out changes in participant fitness throughout the study period, and whether this was the reason for the change in performance. Nevertheless, there is general acceptance that athletes should refrain from using caffeine chronically during training, or within their diet, as it will diminish the ergogenic effects experienced during competition (Fisher et al., 1986; Sokmen et al., 2008).

Therefore, in light of the contrasting findings and methodological limitations of previous research, this study aimed to investigate the effect of chronic caffeine intake on exercise performance in the form of a HIIT session. The study had two objectives; firstly, to investigate whether caffeine ingestion can acutely increase the amount of work possible during exercise at fixed RPE; and secondly to investigate whether the acute effects of caffeine are affected following a 4-week period of caffeine intake. We hypothesised that during exercise at a fixed RPE, caffeine ingestion would increase the amount of work that can be performed during a given period of time, but this effect would be reduced after a 4 week period of chronic caffeine use.

### **III. Methods**

Twenty recreationally active male participants (age:  $33 \pm 9$  yrs; body mass  $78.3 \pm 9.3$  kg;  $\dot{V}O_{2\text{peak}}$   $55.3 \pm 8.9$  ml.kg<sup>-1</sup>.min<sup>-1</sup>; PO:  $333 \pm 49$  W) volunteered to take part in the study. Following institutional ethical approval of the study (protocol number 140-2014\_2015), the participants were provided with written informed consent forms to participate. All participants were instructed to continue their normal daily activities, and to refrain from starting any new training until the end of the study. In addition, participants were told to maintain their usual

diets over the period of the study. Prior to the start of the study, a list of caffeine containing food and drinks to be avoided in the 48 hours before each test was given to the participants. The participants were also given the modified version 1988 Caffeine Consumption Questionnaire (CCQ) of Landrum (1988) about their habitual caffeine use.

### Experimental Study Design

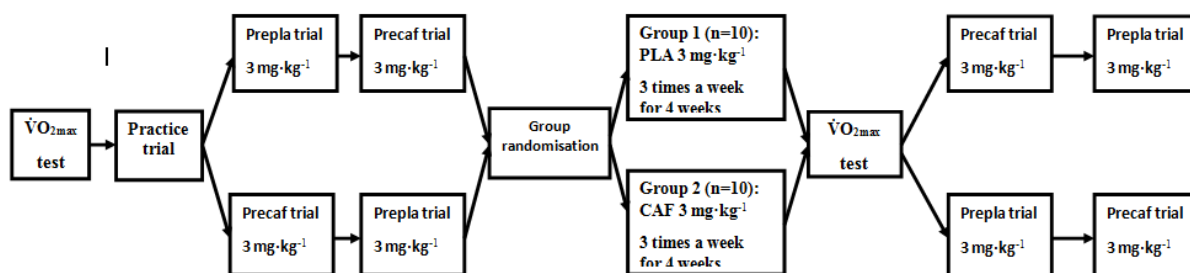


Figure 19: An overview of the study protocol

Each participant visited the laboratory on 7 separate occasions, the first of which was for the determination of  $\dot{V}O_{2peak}$  and to familiarize themselves with the use of the RPE scale (Borg 6-20 scale; Borg 1998). Visit 2 consisted of a full familiarization of the HIIT protocol to be used during the subsequent experimental trials. Then, using a double-blinded, placebo-controlled, cross-over design, participants were randomised to either caffeine or placebo conditions for visit 3, with visit 4 being the opposite condition. One-hour prior to the HIIT session (*see effort-based HIIT session*), participants consumed either 3 mg.kg<sup>-1</sup> body mass of caffeine, or a dextrose placebo (3 mg.kg<sup>-1</sup> body mass), contained within a gelatine capsule to disguise taste and consistency.

Following visit 4, participants were randomized into either a caffeine (CAF) or placebo (PLA) group and consumed either caffeine (3 mg.kg<sup>-1</sup> body mass) or dextrose placebo (3 mg.kg<sup>-1</sup>

body mass) capsules, 3 times per week (1 hour prior to exercise training), for 4 weeks. During this 4-week period, participants were instructed to continue their normal habitual physical activity routine, without introducing any new activity. Following the 4-week period of caffeine or placebo ingestion, participants returned to the laboratory to complete another  $\dot{V}O_{2\max}$  test (visit 5), and a caffeine and placebo HIIT session (visits 6 & 7) using a double blind, placebo-controlled, cross-over design as outlined above.

## Experimental Procedures

### *$\dot{V}O_{2\max}$ test*

Upon arrival at the laboratory, participants had their height and mass measured (Seca, Hamburg, Germany), and the electromagnetically braked cycle ergometer (Corival, Lode, Groningen, The Netherlands) was adjusted to their requirements with the setting recorded to allow reproduction at each subsequent visit. Participants were then given standard instructions for the overall rating of perceived exertion using the 6-20 Borg scale (Borg, 1998). Following a 5 min warm-up at 50 W, participants completed an incremental cycling test to volitional exhaustion using a 25 W per minute ramp rate. Exhaustion was determined as the point at which pedal cadence dropped below 60 RPM for more than 5 seconds, despite strong verbal encouragement (Andreacci et al., 2002). Expired gases were continuously measured throughout the test using an online breath-by-breath gas analyser (CORTEX Biophysik GmbH, Leipzig, Germany). HR was also continuously recorded using a chest strap and watch (S810i, Polar, Kempele, Finland).  $\dot{V}O_{2\text{peak}}$  was determined as the highest  $\dot{V}O_2$  attained during a 60 s period in the test. Maximal cycling power output was calculated as the highest 60 s power output obtained during the incremental cycling using the formulae:

$$W_{\max} = WE + (25W/t \times tE)$$

*Effort-based HIIT Sessions*

The effort-based HIIT sessions were completed on an air-braked cycle ergometer (Wattbike Trainer, Wattbike, UK), which allowed participants to continually vary their power output (PO) throughout the session. Participants initially completed a standardised warm-up consisting of 5-min at 100 W. Participants then completed the HIIT session, which consisted of 4 x 4 min intervals, with 3 min unloaded active recovery between each interval (total training time of 25 min). During each interval, participants were required to cycle at a fixed rating of perceived exertion, but were able to continuously vary their PO by manually adjusting the cycle ergometer air brake to ensure the target RPE was maintained. The exercise intensity that each participant was required to maintain during the 4 intervals was determined by RPEs of 16, 17, 18 and 19 (Borg's 6-20 scale; Borg, 1998), following an incremental format.

During the HIIT sessions,  $\dot{V}O_2$  and HR were continuously measured as outlined above. A finger-tip capillary blood sample was taken 1 min into the recovery following each interval to determine blood lactate concentrations (C-Line, Biosen, EKF Diagnostics, Cardiff, UK). Surface EMG was recorded via the use of surface electrodes (Bagnoli, Delysis Inc, Massachusetts, USA) placed on the VL of the right leg and a reference electrode placed on the patella of the same leg. The skin was prepared by carefully shaving and cleaning the area. The EMG was recorded at a sampling frequency of 1000 Hz. A 30 s average of EMG signals from each stage of the HIIT was then normalized to the maximum EMG obtained during the HIIT session.



## Statistical Analysis

Initially normality was assessed using the Shapiro-Wilk test. Between-group comparisons for age, stature, body mass, self-reported habitual caffeine use,  $\dot{V}O_{2\text{peak}}$  and maximal cycling power output were assessed using independent t-tests. A mixed-design factorial ANOVA was used to assess the effect of the group (CAF vs PLA), condition (caffeine vs placebo), test time point (pre- vs. post- 4 week supplementation period), and the 4 effort based exercise intensities used during HIIT (RPE intensity) for the variables of power output, cadence, heart rate, and blood lactate concentration. If a significant interaction was revealed, the main effect of condition was not considered, and tests of simple main effects of condition were reported using the Holm-Bonferroni method (Holm, 1979). All statistical analysis was conducted using SPSS (SPSS, Chicago, Illinois, USA) with significance levels accepted at 95% ( $p < 0.05$ ).

## IV. Results

### *Baseline measures*

An independent samples t-test was conducted to examine the differences in baseline measures of age, stature, body mass, habitual caffeine use,  $\dot{V}O_{2\text{peak}}$  and maximal cycling power output between caffeine and placebo groups. The equality of variance showed no differences ( $p=.846$   $F=.040$ ). Results indicated that the caffeine group ( $m=131.01$ ,  $SD=117.92$ ) was greater than the placebo group ( $m=122.78$   $SD=108.25$ ) across all variables,  $t(10)=-.126$ ,  $p>0.05$  Cohen's  $D=.07$ ).

*The effect of 4-weeks' caffeine supplementation on the effects of caffeine during effort-based HIIT*

*Table 6: Presents the grand mean results of caffeine and placebo groups in both pre- and post-tests, before and after the 4 weeks of training.*

	<b>PRE PLA</b>	<b>PRE CAF</b>	<b>POST PLA</b>	<b>POST CAF</b>
<b>CAF GROUP</b>				
PO (W)	236 ± 34	249 ± 37	237 ± 34	248 ± 34
CAD (rev.min <sup>-1</sup> )	85 ± 12	90 ± 11	91 ± 11	92 ± 11
HR (beats.min <sup>-1</sup> )	156 ± 15	159 ± 14	157 ± 17	158 ± 16
LT (mmol.L <sup>-1</sup> )	7.0 ± 3.0	7.6 ± 3.0	6.7 ± 2.0	7.4 ± 3.0
<b>PLA GROUP</b>				
PO (W)	213 ± 39	224 ± 35	211 ± 39	224 ± 38
CAD (rev.min <sup>-1</sup> )	90 ± 13	91 ± 13	91 ± 11	91 ± 8
HR (beats.min <sup>-1</sup> )	162 ± 13	163 ± 13	165 ± 12	168 ± 13
LT (mmol.L <sup>-1</sup> )	7.0 ± 3.0	7.6 ± 3.0	6.7 ± 3.0	7.7 ± 4.0

Table 6: Grand means of caffeine and placebo groups from HIIT sessions undertaken pre- and post- 4-week supplementation period in both caffeine and placebo conditions.

*Effect of caffeine use on cycling power output and work done during HIIT*

Figure (20) displays the effects of acute caffeine and placebo ingestion on cycling power output during the HIIT session pre and post the 4-week supplementation period. Statistical

analysis demonstrated an overall interaction effect between acute treatment, intensity, chronic treatment and time point ( $P=0.020$ ;  $F=3.55$ ). However, there was no interaction between acute treatment and time point ( $P=0.020$ ;  $F=3.55$ ). However, there was no interaction between acute treatment, chronic treatment and time point ( $P=0.54$ ;  $F=0.71$ ), suggesting the effects of caffeine were the same regardless of when the test took place. The significant main effects were found for acute treatment ( $P < 0.001$ ;  $F=90.34$ ), and intensity ( $P < 0.001$ ;  $F=25.63$ ), showing that caffeine increased power output at a fixed RPE intensity. In addition, as the results showed, there were no differences in power output at each RPE intensity pre to post training, between caffeine/placebo groups (within subjects), and the work done was greater following caffeine ingestion compared to the placebo. Therefore, figure (21) displays the effects of acute caffeine and placebo ingestion on averaged work done means in kilojoules of caffeine and the placebo bar per RPE intensity.

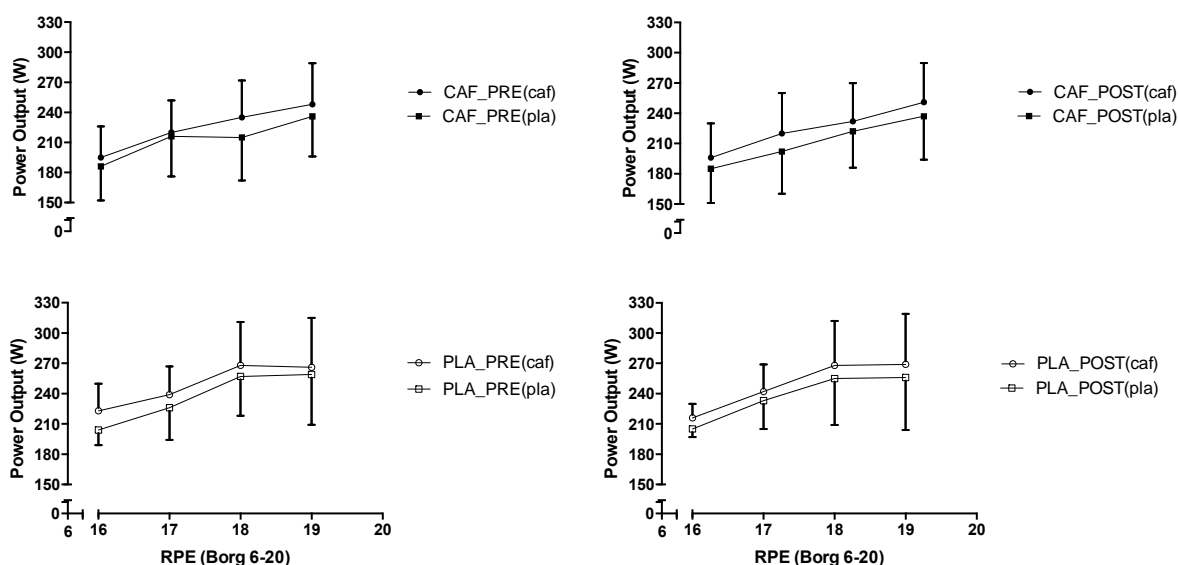


Figure 20: Mean power output during exercise at RPE 16-19 in CAF and PLA groups, pre- and post-4 weeks of training in acute CAF and PLA conditions.

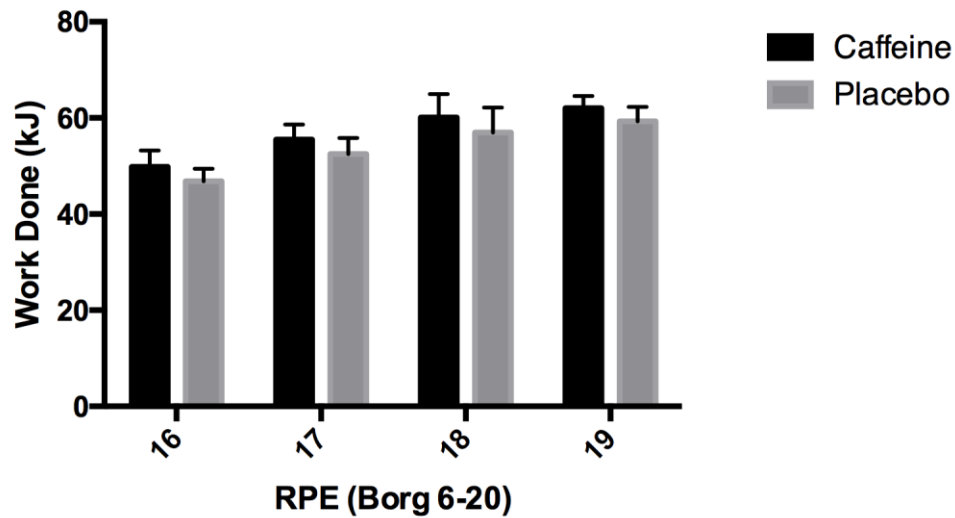


Figure 21: An averaged mean of pre and post-4 weeks of training work done during exercise at RPE 16-19 in acute CAF and PLA conditions.

### *Cadence*

Figure (22) displays the effects of acute caffeine and placebo ingestion on cycling cadence during HIIT pre and post the 4-week supplementation period. There was no overall interaction effect between acute treatment, intensity, chronic treatment and time point ( $P=0.328$ ;  $F=1.09$ ). However, a significant interaction was evident between chronic treatment and time point ( $P=0.018$   $F=6.826$ ).

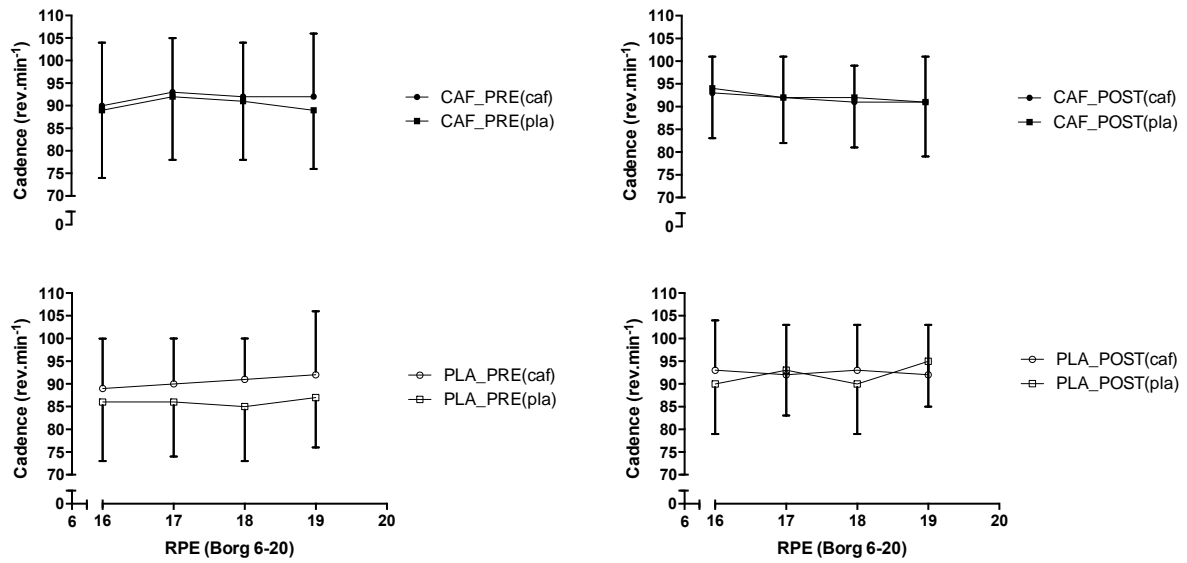


Figure 22: Mean cadence during exercise at RPE 16-19 in CAF and PLA groups, pre- and post-4 weeks of training in acute CAF and PLA conditions.

### *Heart rate*

Figure (23) displays the effects of acute caffeine and placebo ingestion on HR during HIIT pre and post the 4-week supplementation period. There was no overall interaction effect between acute treatment, intensity, chronic treatment and time point ( $P=0.719$ ;  $F=3.00$ ). A main effect evident for the effect of the acute treatment ( $P = 0.011$ ;  $F=1.00$ ).

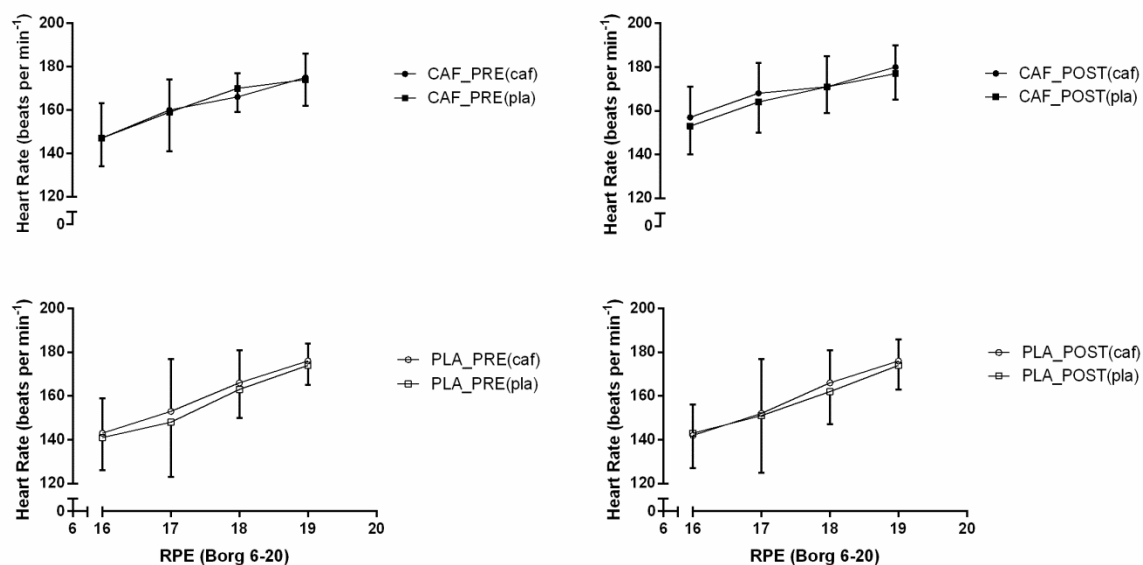


Figure 23: Mean heart rate response during exercise at RPE 16-19 in CAF and PLA groups pre- and post- 4 weeks of training in acute CAF and PLA conditions.

### *Blood lactate*

Figure (24) displays the effects of acute caffeine and placebo ingestion on blood lactate concentration during HIIT pre and post the 4-week supplementation period. There was no overall interaction effect between acute treatment, intensity, chronic treatment and time point ( $P=0.220$ ;  $F=1.519$ ). However, there was a main effect for acute treatment ( $P=0.012$ ;  $F=7.913$ ), with the CAF condition displaying a greater blood lactate than the PLA condition. There was no interaction effect between acute treatment and time point, suggesting that the lactate response was similar between test 1 and 2 conducted pre and post the 4 week training period.

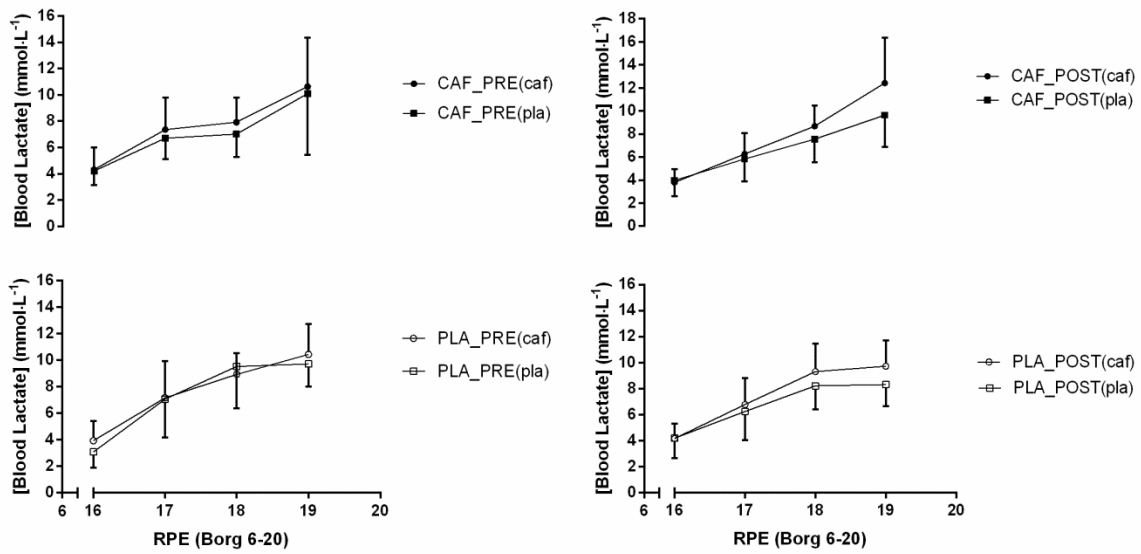


Figure 24: Blood lactate following exercise at RPE 16-19 in CAF and PLA groups, pre- and post-4 weeks of training in acute CAF and PLA conditions.

### *Electromyography (EMG)*

Figure (25) displays the effects of acute caffeine and placebo ingestion on Vastus Lateralis EMG during HIIT pre and post the 4-week supplementation period. There was no overall interaction effect between acute treatment, intensity, chronic treatment and time point ( $P=0.547$ ;  $F=1.390$ ). However, there was a significant main effect of the acute treatment ( $P = 0.004$ ;  $F = 1.00$ ), and of exercise intensity ( $P = 0.049$ ;  $F = 1.773$ ).

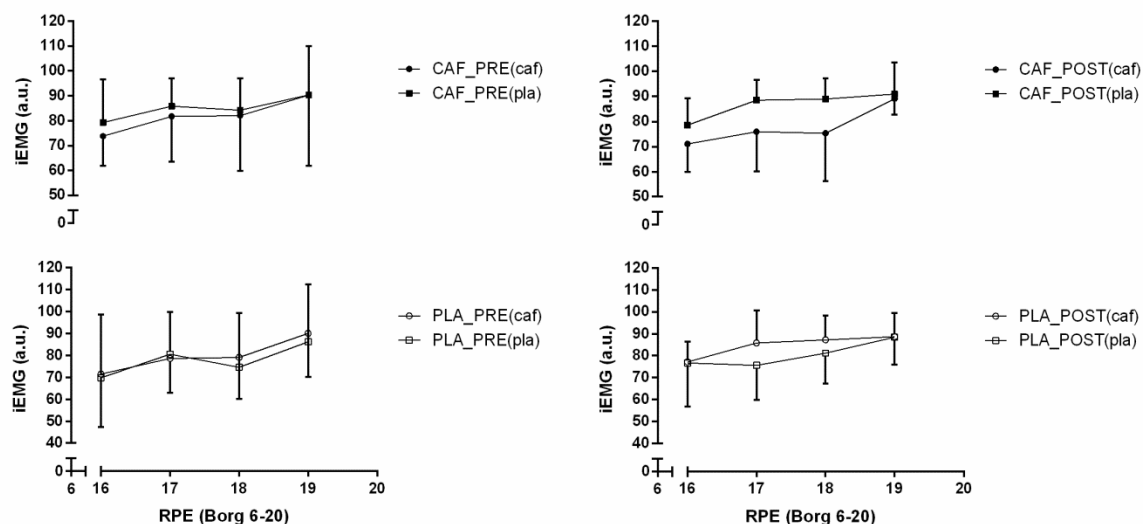


Figure 25: iEMG following exercise at RPE 16-19 in CAF and PLA groups, pre- and post-4 weeks of training in acute CAF and PLA conditions.

## I. Discussion:

This study utilised a randomized, double-blinded, placebo controlled study design to investigate the effects of chronic caffeine ingestion on effort-based HIIT. The main findings of the study were that caffeine exerts an ergogenic effect during HIIT at exercise intensities corresponding with the rating of perceived exertion of 16, 17, 18 & 19 on the Borg 6-20 scale. Specifically, the results of this study demonstrate that caffeine ingestion increases power output, HR and blood lactate concentration for the same level of RPE. Moreover, the frequent use of caffeine, taken three times a week for four weeks, does not reduce the acute ergogenic effects of caffeine during HIIT.



*Acute effects of caffeine ingestion*

In agreement with previously published works, the consumption of 3 mg.kg<sup>-1</sup> caffeine in this study led to an increased work output rate during HIIT at fixed RPEs (Cole et al., 1996; Duncan, 2009; Falk et al., 1989; Flinn et al., 1990; Ivy et al., 1979). The higher work output at fixed RPE levels on the caffeine condition demonstrates that the participant has the ability to sustain higher total exercise intensities without significant peripheral fatigue occurring during exercise. From the data available, it is difficult to explain why participants could sustain a higher work output at the same perceived work rate following caffeine ingestion. As the HIIT was non-exhaustive and for a short duration, it is unlikely that muscle glycogen depletion or other metabolic factors significantly affected the performance measurements. Even though the exact mechanism is unclear, it is likely that the effects observed following the acute administration of caffeine are a result of the direct stimulatory effect on the CNS (de Morree et al., 2014). It is plausible that caffeine ingestion led to a rise in circulating dopamine and noradrenaline, which changed neuronal excitability, due to its antagonizing action on adenosine A<sub>1</sub> and A<sub>2</sub> receptors (Fredholm et al., 1999). Indeed, Davis et al. (2003) have suggested that caffeine may reduce perception of effort by inhibition of brain adenosine receptors, which would otherwise inhibit the export of excitatory neurotransmission within the motor cortex. An increase in adenosine, along with a concomitant increase in 5-HT, have been associated with central fatigue during endurance exercise (Davis and Bailey, 1997). The blocking of the adenosine receptors is therefore likely to allow a higher work rate to be sustained and a delayed fatigue (Kovacs et al., 1998) during the HIIT protocol used within the current study. In addition, caffeine may have exerted a peripheral effect in terms of reducing the excitation threshold of motor neurons, thus facilitating motor unit recruitment (Waldeck, 1975). The results of the current study demonstrate a significantly higher iEMG in the CAF condition vs

PLA, suggesting an increase in muscle recruitment at the same fixed RPE may have contributed to the higher work output observed. However, the study outcomes in support of this hypothesis are mixed, with a recent study by Wardle et al. (2012) demonstrating that effortful decision making (high cost vs. high reward tasks) was unchanged following caffeine ingestion.

#### *Effects of chronic caffeine ingestion*

The findings of the current study demonstrate that chronic caffeine supplementation does not reduce its ergogenic effect during a subsequent acute dose. This finding concurs with previous cross-sectional studies of habitual and naïve caffeine consumers, which report no difference in exercise performance following acute caffeine consumption, between high and low habitual caffeine consumers (Dodd et al., 1993; Goncalves et al., 2017). However, in contrast, other studies have identified a tolerance to the ergogenic benefits of caffeine supplementation in high habitual consumers (Bell and McLellan, 2002; Robertson et al., 1981). However, the divergent results from these previously published studies are likely, due to their cross-sectional study design, with the need to stratify participants into arbitrary sub-groups based upon their habitual caffeine intake. The wide ranges used to characterise habitual caffeine consumption in these studies may have contributed to the divergent results. In contrast, the current study employed a longitudinal experimental study design to directly test whether chronic caffeine ingestion resulted in a tolerance to its acute ergogenic effects on exercise performance. Only one previously published study has examined the effects of a prolonged period of controlled caffeine intake on exercise performance (Beaumont et al., 2016). Beaumont et al. (2016) investigated the effects of an acute dose of 3 mg.kg<sup>-1</sup> caffeine on a 30 min cycling performance trial performance before and after 4 weeks of chronic caffeine supplementation. The performance benefit seen by a comparison to a placebo condition prior to the 4-week supplementation period was not evident at the post-trial. Beaumont et al. (2016) subsequently

concluded that chronic ingestion of caffeine resulted in a tolerance being developed by the participants. However, the study design did not include a post-supplementation placebo trial as a comparator for the post-supplementation caffeine trial, which unfortunately limits the ability to interpret the findings of the study. By contrast, the current study did not find any association between the different levels of habitual caffeine intake (caffeine vs placebo groups), and increases in power output during effort-based HIIT with caffeine supplementation. In this scheme, caffeine directly enhances the activity of enzymes that break down fat into fatty acids or caffeine increases circulating levels of epinephrine (EPI). Several studies suggest that chronic caffeine use dampens the EPI response to exercise and to caffeine, but does not affect indirect markers of fat metabolism during exercise (Bangsbo et al., 1992; Van Soeren et al., 1993). However, these alterations do not appear to dampen the ergogenic effect of 6-9 mg/kg caffeine. Endurance performance increased in all subjects in two studies in which both habitual and naïve users of caffeine were tested; the users refrained from caffeine for 48-72 h prior to the experiments (Graham and Spriet, 1991; Spriet et al., 1992).

Unfortunately, we were not able to measure blood caffeine concentrations during the acute or chronic phases of the study. However, previously published studies where similar dosages of caffeine have been ingested 60 min prior to exercise have demonstrated significant increases in plasma caffeine concentration (Beaumont et al., 2016; Bell and McLellan, 2002; Graham and Spriet, 1995). Therefore, it is assumed that the 3 mg.kg<sup>-1</sup> supplementation used in the current study was sufficient to increase blood caffeine concentrations. As part of the protocol of the current study, participants were required to refrain from caffeine in the 48 hours prior to the exercise tests. This could be suggested to be a confounding influence on the results of the current study, as it may diminish the effects of supplementation in the caffeine group. However, previous research has demonstrated that 4 days of caffeine withdrawal does not

influence the exercise responses to an acute 3 mg.kg<sup>-1</sup> caffeine dose in habituate caffeine users (Irwin et al., 2011). As a consequence, it is believed that the withdrawal of caffeine supplementation in the 48 hours prior to the exercise testing sessions did not influence the subsequent exercise responses during the self-paced HIIT.

## **Conclusion**

In conclusion, this study demonstrates that caffeine ingestion 1 hour prior to a HIIT session can increase work done in a defined period of time for the same perceived effort. Moreover, these exercise responses were not affected by chronic caffeine consumption over a 4-week period, refuting the opinion that habitual caffeine supplementation during training will negatively affect the ergogenic effects of caffeine use during subsequent competitive performance.

# Chapter 7

## General Discussion

## **General discussion**

The aim of this general discussion is to summarize and compare the main findings of the experimental studies within this thesis with reference to the proposed link between the perception of effort and endurance performance. This chapter will also consider the limitations of the thesis and suggestions for future research.

Previous researchers have investigated on the link between perception of effort and endurance exercise performance (Enoka and Stuart, 1992; Marcora et al., 2008; Marcora et al., 2009). However, these studies have been confined to the use of TTE testing as an index for endurance performance. Indeed, some researchers have established a linear relationship between exercise duration and the perception of effort (Noble et al., 1973; Parfitt and Eston, 1995; Wenos et al., 1996). However, prior to this thesis there were a limited number of investigations that considered whether the perception of effort was a key predictor of self-paced endurance exercise performance (Abbiss et al., 2015; Joseph et al., 2008; McCormick et al., 2015).

The experimental study documented in Chapter 3 aimed to investigate the relationship between endurance exercise performance (in the form of a 30 min cycling time trial), and the proposed physiological determinants of endurance performance including  $\dot{V}O_{2\max}$ , LT and cycling efficiency. In an attempt to determine whether the perception of effort was an important determinant of endurance performance in this context, a novel test of perceptual efficiency was used, whereby the slope of the increase in RPE over time at a fixed exercise intensity was used. Following regression analysis, results demonstrated that  $\dot{V}O_{2\max}$  ( $L \cdot \text{min}^{-1}$ ) was the best predictor of 30 min laboratory cycling time trial performance. Given the moderately trained cohort of participants within the study, and the wide range of  $\dot{V}O_{2\max}$  values recorded, it is unsurprising that a strong correlation was established with time trial performance. However, numerous previous studies have published significant relationships between physiological

parameters (e.g. Maximum minute power output,  $\dot{V}O_{2\max}$ , lactate threshold, and efficiency) and endurance cycling time-trial performance (Balmer et al., 2000; Hawley and Noakes, 1992; Hoogeveen and Hoogsteen, 1999).

The findings of the study in Chapter 3 demonstrate that perceptual efficiency did correlate with cycling time trial performance ( $r = -0.66$ ,  $p < 0.05$ ), but this only explained a small proportion (31.4%) of the overall variation in performance within the cohort of participants. The study used a heterogeneous group of recreationally active males and females that might have biased the relationship between endurance exercise performance and  $\dot{V}O_{2\max}$ , leading to stronger correlations between the two parameters than if a group more homogenous for  $\dot{V}O_{2\max}$  (i.e. trained athletes), was used. Indeed, using a cohort of well-trained cyclists, Joseph et al. (2008) demonstrated that perception of effort is related to time trial cycling performance, regardless of distance. Marcora and colleagues (2008, 2009 and 2010) provide a model by which perception of effort is suggested to regulate endurance performance. Marcora's model suggests that the relationship between the RPE and endurance exercise performance is underpinned by various cognitive and motivational factors i.e. potential motivation, knowledge of the distance/time to cover and that remaining, and previous experience of perceived exertion during exercise of varying intensity and duration. When the perceived exertion exceeds the motivation of the individual to keep exercising at a given pace, they reduce their intensity.

The experimental study documented in Chapter 4 investigated the effect of mental fatigue on endurance performance in the form of repeated TTE tests. TTE tests are commonly used within the sports science domain, including in the determination of the CP and W'. In agreement with previous research, the study hypothesised that mental fatigue would increase the perception of effort and reduce TTE performance (Marcora et al., 2009), therefore decreasing CP and W'

without affecting their purported skeletal muscle physiological determinants. The results of the study demonstrated that as hypothesised, mental fatigue did indeed increase the perception of effort and reduce TTE. As a consequence, the intercept of the work-time relationship was reduced when participants were mentally fatigued. However, the slope of work-time relationship was not affected by mental fatigue, meaning that CP was unchanged.

It is unlikely that the prolonged mental exertion from the Stroop Tasks used in this study affected any of the local muscular physiological processes that are thought to underpin CP and W' (Jones et al., 2008; Rossiter et al., 2002). Nevertheless, the fixed amount of 'anaerobic' work that participants could perform above the CP, prior to exhaustion was reduced, regardless of the rate at which the work was performed. According to the psychobiological model of endurance performance, the faster progressive increase in the perception of effort over time in the MF condition caused premature exhaustion during the tests as, due to the nature of the TTE protocol, participants could not choose to cycle at a lower power/torque. As a consequence, the only decision they could make was to terminate the exercise trial. This early termination was likely prior to participants expending all of their W', hence also explaining the lower blood lactate concentrations observed in the MF condition. The implications of the findings of this study are that scientists, athletes and coaches should avoid prolonged cognitive tasks prior to a laboratory assessment of physiological parameters. In addition, endurance athletes should avoid prolonged periods of mental exertion prior to training and competition.

The experimental study in chapter 5 aimed to investigate the reliability of self-paced exercise, regulated by the perception of effort. Establishing the reliability of the intermittent exercise protocol developed in Chapter 5 was important, as it was to be utilised as both a testing and training protocol in the experimental study in Chapter 6. Previous studies have demonstrated



that the RPE displays a reliable and positive linear response during exercise testing at a variety of different intensities (Doherty et al., 2001; Eston and Williams, 1988; Smutok et al., 1980).

As the focus of this thesis is concerned with the manipulation of the perception of effort, caffeine was used in Chapter 6 in order to reduce the RPE. Therefore, as an extension to the previous research that has investigated the reliability of the RPE, the experimental study in Chapter 5 did so following 3 mg.kg<sup>-1</sup> of caffeine ingestion. The findings of the study demonstrated that participants were able to reliably reproduce self-paced exercise equivalent to RPEs of 16, 17, 18 and 19. Specifically, following caffeine ingestion a mean coefficient of variation (CV) across all RPE intensities of 2.10% (05% CL = 1.03 - 3.61%) was found for cycling power output, 1.6% (95% CL = 0.60 - 3.77%) for cycling cadence, and 1.40% (95% CL = 0.51 - 2.51%) for heart rate. This high level of reliability for the intermittent high intensity cycling protocol meant that it was able to be utilised as part of the intervention protocol in Chapter 6.

The experimental study in Chapter 6 investigated the acute effects of caffeine ingestion on the high intensity intermittent cycling protocol, and how these were affected by chronic caffeine intake. Using a randomized, counterbalanced and double-blinded placebo-controlled design, twenty recreational male endurance athletes ingested either caffeine (3 mg.kg<sup>-1</sup> body mass), or a placebo one hour before exercise. In line with previous research (Cole et al., 1996; Duncan, 2009; Falk et al., 1989; Flinn et al., 1990; Ivy et al., 1979), the study found that caffeine acutely increased power output, HR and blood lactate during high intensity self-paced cycling. The experimental study in Chapter 6 also established that the acute effects of caffeine ingestion were conserved after 4 weeks of chronic caffeine ingestion (3 mg.kg<sup>-1</sup> body mass, 3 x per week). Indeed, in agreement with the findings of this thesis, but using a cross-sectional study

design, the ergogenic effect of caffeine has previously been reported to be consistent between habitual and non-habituated caffeine consumers (Dodd et al., 1991; Goncalves et al., 2017).

Only one previously published study has experimentally examined the effects of a prolonged period of controlled caffeine intake on exercise performance (Beaumont et al., 2016). Beaumont et al. (2016) investigated the effects of an acute dose of 3 mg.kg<sup>-1</sup> caffeine on a 30 min cycling performance trial performance before and after 4 weeks of chronic caffeine supplementation. The performance benefit seen by a comparison to a placebo condition prior to the 4-week supplementation period was not evident at the post trial. Beaumont et al. (2016) subsequently concluded that chronic ingestion of caffeine resulted in a tolerance being developed by the participants. However, the study design did not include a post-supplementation placebo trial as a comparator for the post-supplementation caffeine trial, which unfortunately limits the ability to interpret the findings of the study. By contrast, the study in Chapter 6 did not find any association between the different levels of habitual caffeine intake (caffeine vs placebo groups), and increases in power output during effort-based HIIT with caffeine supplementation. Therefore, the conclusions drawn from Chapter 6 are that the frequent ingestion of caffeine during training does not reduce its ability to enhance power output for the same level of effort during subsequent acute exercise, or competition.

## **I. Theoretical Implications**

Perceived exertion is suggested to be a key determinant of endurance exercise performance (Abbiss et al., 2015; McCormick et al., 2015). Indeed, as demonstrated in Chapter 3 of this thesis and other studies (e.g. Marcora et al., 2009; Mauger et al., 2010; Watson et al., 2005) following experimental intervention, it is possible to manipulate the perception of effort during subsequent submaximal exercise trials (e.g. a time trial), and affect resultant performance. This has also been demonstrated during TTE testing at a fixed exercise intensity, where participants stop exercising once their perceived exertion exceeds their motivation for the trial. Indeed, this is demonstrated by the experimental study in Chapter 4 of this thesis, where participants were required to exercise for as long as possible, with and without mental fatigue. The participants rated their perception of effort similar in both conditions at the start of the trial, but the rate of increase in RPE was steeper in the mental fatigue vs control condition, meaning that they disengaged from the task earlier following mental fatigue. However, participants in the mental fatigue condition still rated their perception of effort as being maximal prior to disengagement. This earlier disengagement appears to be related to the subjective feelings of “tiredness” and “lack of energy” (Boksem and Tops, 2008) caused by mental fatigue. In support, previous research has demonstrated the negative effect of mental fatigue induced by prolonged mental exertion (30-90 min) on endurance performance during both whole-body (Marcora et al., 2009) and single-joint exercise (Pageaux et al., 2013). These studies demonstrated that a higher perception of effort is observed independently of any change of the neuromuscular, cardiorespiratory, and metabolic responses to exercise and support the psychobiological model of endurance performance in which perceived exertion has an important role in limiting endurance performance (Marcora and Staiano, 2010).

The exact cognitive process involved in generating the increased perception of effort during exercise performance after mental exertion is yet to be fully elucidated. Of particular interest is response inhibition, which is a cognitive process related to the inhibition of unwanted/inappropriate or emotional responses (Mostofsky and Simmonds, 2008), and it is an important component in decision-making during volitional tasks (Haggard, 2008). Cognitive tasks inducing response inhibition (as used in the experimental study in Chapter 4), are thought to activate the anterior cingulate cortex (ACC) and the pre-supplementary motor area (Mostofsky and Simmonds, 2008). Activity in these cortical areas has been associated with perceived exertion (de Morree et al., 2012; Williamson et al., 2001, 2002), and impairment to the ACC is recognised to impact effort-based decision-making in animals (Rudebeck et al., 2006; Walton et al., 2003, 2006). Thus, it is biologically reasonable to suggest that prior mental exertion involving response inhibition would influence the effort-based decision-making procedure assumed to regulate endurance exercise performance (Marcora, 2010). However, in both experimental conditions in Chapter 4, the time to exhaustion task required both physical exertion, as well as mental exertion, which are likely to have interacted to impair endurance performance. Indeed, as both physical and mental fatigue are known to negatively influence perceived exertion (Mehta and Parasuraman, 2013; Pageaux et al., 2014), it is probable that the combined load of both mental and physical exertion contributed to increasing the perception of effort and ultimately limited the time to exhaustion endurance exercise.

In contrast to the effects of mental fatigue being used to increase the perception of effort, caffeine is a psychoactive substance that has been shown to reduce the perception of effort during exercise (Berglund and Hemmingsson, 1982; Costill et al., 1978; Essig et al., 1980; Flinn et al., 1990). Caffeine has been shown to have a central role in altering the release,

requirement, or activity of neurotransmitters in the brain, and in this manner influencing the perception of effort. Even though the exact mechanism is unclear, it is likely that the effects observed following the acute administration of caffeine are a result of the direct stimulatory effect on the CNS (de Morree et al., 2014).

It is plausible that caffeine ingestion led to a rise in circulating dopamine and noradrenaline which changed neuronal excitability due to its antagonizing action on adenosine A<sub>1</sub> and A<sub>2</sub> receptors (Fredholm et al., 1999). Indeed, Davis et al. (2003) have suggested that caffeine may reduce perception of effort by inhibition of brain adenosine receptors, which would otherwise inhibit the export of excitatory neurotransmission within the motor cortex (Davis et al., 2003). An increase in adenosine, along with a concomitant increase in 5-HT, have been associated with central fatigue during endurance exercise (Davis and Bailey, 1997). Blocking of the adenosine receptors is therefore likely to allow a higher work rate to be sustained, and postpone fatigue (Kovacs et al., 1998) during high intensity exercise.

Moreover, the effect of caffeine blocking brain adenosine in the anterior cingulate cortex (ACC) of the brain has important implications for the control of muscle contraction and the perception of effort during intensive or prolonged exercise (Pageaux et al., 2015). The result is that an individual's perception of effort may be reduced for the same level of physical effort or work (Rudebeck et al., 2006; Walton et al., 2003, 2006), or to increase physical effort or work output at a fixed rating of perceived exertion (Cole et al., 1996; Ivy et al., 1979; Plaskett and Cafarelli, 2001). Indeed, results of Chapter 6 in this thesis, and that of others (Tarnopolsky et al., 1989; Trice and Haymes, 1995) clearly demonstrate this effect. In addition, caffeine may have also exerted a peripheral effect in terms of reducing the excitation threshold of motor neurons, facilitating motor unit recruitment (Cole et al., 1996; Waldeck, 1975). Results from the experimental study in Chapter 6 demonstrate a significantly higher iEMG in the CAF

condition vs PLA, suggesting an increase in the muscle recruitment at the same fixed RPE may have contributed to the higher work output observed.

## **II. Practical implications**

The rating of perceived exertion is often used as a marker of intensity and of homeostatic imbalance during exercise. It is usually monitored through exercise tests to assess other measures of exercise intensity. As such, RPE is used to predict exercise capacity, assess alterations in training status, and describe changes in endurance performance and pacing strategy. Therefore, assuming a constant potential motivation, any psychological or physiological manipulation that reduces the perception of effort and increases endurance exercise performance, for example, as shown in chapter 4, mental fatigue increases perception of effort and reduces time to exhaustion during high-intensity cycling exercise, whilst as shown in chapter 6, caffeine ingestion has the opposite effect.

Indeed, several studies have reported that the rate of increase in RPE through self-paced competitive tasks varies during constant-load or distance tasks, where the subjects exercise to volitional exhaustion, and this is in direct relation to the length of time that the participant continues to exercise (Baden et al., 2005; Joseph et al., 2008). The increase in perception of effort over time is then set as a consequence of this subconscious teleoanticipatory calculation of the safe duration of endurance exercise, with some ongoing adjustments based on continuous afferent feedback. In short, using a TTE model of endurance performance, the TTE and perception of effort are both based on a subconscious decision taken at the beginning of endurance exercise. However, TTE is limited by a conscious decision to stop exercise, taken at the end of endurance exercise on the basis of reaching a maximal perception of effort (Marcora and Staiano, 2010).

Alterations in pace during exercise can be clarified by a constant internal negotiation of momentary RPE associated with a pre-planned “ideal rate of RPE development” pattern, which accounted for the portion of duration covered and the expected end point. Thus, as suggested in Chapter 3, physical performance may also be assessed using RPE, for example, the rate of increase in the perception of effort over time at a fixed exercise intensity, or during incremental exercise testing and the work rate at which a peak RPE was achieved. Indeed, the perception of effort has been correlated with several physiological aspects such as oxygen consumption, blood lactate, heart rate, electromyography, and rectal and skin temperature (Davies et al., 2008; Eston and Williams, 1988) suggesting that it is related to the physiological process which occurs during exercise.

The findings from Chapter 4 of this thesis along with those of Marcora et al. (2009) and Pageaux et al. (2014) provide important data demonstrating a significant reduction in endurance exercise performance following a prolonged period of cognitive work. Regardless of the exact mechanisms, these experiments provide evidence that athletes should avoid mental exertion prior to training and competition. Specifically, the ability to appropriately self-pace exercise is an important aspect of athletic performance (Foster et al., 1993), and determines how an athlete regulates their power output during exercise when physiological homeostasis is challenged (Lander et al., 2009; St Clair Gibson et al., 2006). A higher perception of effort is therefore likely to reduce endurance performance, due to a conscious decision made by the athlete to compensate for the negative effect of mental fatigue on the perception of effort (i.e. run at a slower pace to match desired perception of effort).

Indeed, if athletes did not choose to exercise at a lower work rate, the progressive increases in perception of effort over time would likely cause premature exhaustion, as observed during the fixed intensity trials used in Chapter 4 of this thesis. As not finishing a race is a more negative

outcome for an athlete than completing their performance in a longer time, reducing the average performance power output is likely to be the most appropriate behavioural response.

Caffeine ingestion has been used widely among athletes during training and competitions, and extensive research has been conducted on the effect of caffeine ingestion (Bangasbo et al., 1992; Doherty and Smith, 2005; Spriet et al., 1992), and at different doses (Bell and McLellan, 2002). In this regard, the ingestion of caffeine has been shown in chapter 6 of this thesis, and by others (e.g. Cole et al., 1996), as a useful strategy to increase the work rate during a certain exercise task; i.e. participants who ingested caffeine produced higher power outputs for the same perception of effort compared to a placebo. As athletes seem to utilise overall effort and accumulated fatigue to dictate their high intensity interval training sessions (Seiler et al., 2013), it would seem intuitive that athletes take caffeine prior to training in order to exploit its ergogenic effect.

Thus, as demonstrated by the results of Chapter 6, following low dose caffeine ingestion, individuals have the potential to complete more work for the same overall session effort, and potentially realise greater training gains; although further research is required to verify this hypothesis. A potential confounding effect for the chronic use of caffeine during training is that individuals might become habituated to the pharmacological effects of caffeine and thus not realise the same ergogenic effects over time. Indeed, previous cross-sectional and longitudinal research has suggested that a tolerance to the ergogenic effects of caffeine is evident following prolonged use (Beaumont et al., 2016; Bell and McLellan, 2002; Robertson et al., 1981). However, limitations of cross-sectional experimental designs, and lack of control groups limit the ability to interpret the findings of these studies.



The randomized, double-blinded, placebo controlled study design of the experimental study in Chapter 6 is the first to provide objective evidence that the acute ergogenic effects of caffeine are conserved with chronic low dose ( $3 \text{ mg}\cdot\text{kg}^{-1}$  body mass) ingestion. Therefore, the findings of this thesis refute the opinion that habitual caffeine supplementation during training will negatively affect the ergogenic effects of caffeine use during a subsequent competitive performance. Nevertheless, future studies should be conducted to investigate the effect of longer term training ( $>4$  weeks) with chronic caffeine ingestion of higher doses (e.g.  $6 \text{ mg}\cdot\text{kg}^{-1}$  body mass). These studies should aim to investigate whether higher caffeine doses permit a greater overall training load to be maintained, and whether this affords greater training gains than lower doses of caffeine or placebo groups.

### **III. Conclusions and perspectives**

The aim of this thesis was to investigate the role of perception of effort on endurance performance. The role of perception of effort in relation to the physiological parameters such as  $\dot{V}O_2\text{max}$ , lactic threshold, and exercise economy was investigated in Chapter 3. The results suggested that although not the main predictor of cycling time-trial performance, the perception of effort was a significant correlate. Indeed, when perception of effort was increased via use of a mentally fatiguing task in Chapter 4, endurance cycling performance was reduced. In contrast, when the perception of effort is reduced during high intensity intermittent cycling via caffeine ingestion, it is possible to improve performance. Specifically, participants in Chapter 6 were able to produce more work in caffeine vs. a placebo condition. Moreover, these effects on performance were reliable on a test-rest basis (Chapter 5), and were still evident following a period of chronic caffeine ingestion (Chapter 6). This suggests that the use of caffeine during

exercise training could be a beneficial way to allow individuals to exercise at a higher intensity and potentially realise greater training gains in a given period of time. However, further research is required to verify this hypothesis. As there was no tolerance effect of caffeine evident, it is also possible to suggest that chronic caffeine ingestion during training would not affect the subsequent ergogenic effect that caffeine would have taken prior to competition.

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## **Appendix**

This appendix contains an example of the scales for rating of perceived exertion (used in all studies). An example of scale and instruction used for rating of perceived exertion during whole-body exercise (Borg, 1998).

**6 No exertion at all**

**7 Extremely light**

**8**

**9 Very Light**

**10**

**11 Light**

**12**

**13 Somewhat hard**

**14**

**15 Hard (heavy)**

**16**

**17 Very hard**

**18**

**19 Extremely hard**

**20 Maximal exertion**



## Borg's RPE Scale Instructions

While exercising we want you to rate your perception of effort, i.e. how hard, heavy and strenuous exercise feels to you. The perception of exertion depends on how hard driving your legs or arms, how heavy are your breathing, and the overall sensation of how strenuous exercise is. It does NOT depend on muscle pain, i.e. the aching and burning sensation in your leg or arm muscles.

Look at this rating scale; we want you to use this scale from 6 to 20, where 6 means “not exertion at all” and 20 means “maximal exertion”.

9 corresponds to “very light” exercise. For a normal, healthy person it is like walking slowly at his or her own pace for some minutes.

13 on the scale is “somewhat hard” exercise, but it still feels OK to continue.

17 “very hard” is very strenuous exercise. A healthy person can still go on, but he or she really has to push him-or herself. It feels very heavy, and the person is very tired.

19 on the scale is “extremely hard” exercise. For most people this is the most strenuous exercise they have ever experienced.

Try to appraise your feelings of exertion as honestly as possible, without thinking about what actual physical load is (heart rate, speed, power output, intensity level on the exercise machine). Don't underestimate your perception of exertion, but don't overestimate it either.

It is your own feeling of effort that's important, not how it compares to other people's. What other people think is not important either. Look carefully at scale and expressions, and then give a number.