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The relationship between $\dot{V}O_2$ and muscle deoxygenation kinetics and upper body repeated sprint performance in trained judokas

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Abstract

Judo is a sport characterized by short periods of maximal-intensity activity, interceded with short periods of rest, akin to repeated sprint efforts. Moreover, it relies heavily on upper body musculature. The maintenance of performance over the course of several repeated sprint efforts seems to be related to the ability to recover quickly in between efforts, which has been linked to muscular oxidative capacity. The measurement of oxygen uptake kinetics ($\dot{V}O_2$ Kinetics) and muscle haemoglobin/myoglobin deoxygenation kinetics ([HHb] kinetics) constitute non-invasive parameters that provide a surrogate of muscular oxidative capacity. The purpose of this study was to determine if an association could be established between $\dot{V}O_2$ Kinetics, [HHb] kinetics, and other parameters of aerobic fitness and upper body repeated sprint (RSA) performance, and if these associations were observed in a separate group of trained judo athletes (JT) and in a group of untrained individuals (UT).

Fifteen participants, consisting of eight judo athletes (age 21,1 ± 3,0 yr, height 172,3 ± 4,5 cm, body mass 71,5 ± 7,1 kg) and seven healthy individuals untrained in upper body exercise modalities (age 22,6 ± 1,0 yr, height 172,71 ± 4,5 cm, age 64,29 ± 5,8 kg) were recruited as participants for the study. Each participant completed an arm crank incremental test to determine peak oxygen consumption (peak $\dot{V}O_2$), maximal aerobic power (MAP) and the first ventilatory threshold (VT_1). On a subsequent day, two heavy-intensity square-wave exercise transitions of 6 minutes at 20% Δ (20% the workload ranging from VT_1 to MAP) were performed to determine $\dot{V}O_2$ and [HHb] kinetics. On a following session, participants performed an upper body RSA test (4 sprints x 15:45-s work: rest), where [HHb] parameters were monitored, along with peak (PPO) and mean (MPO) power output, total work performed (W) over the course of each sprint. During all testing sessions, pulmonary gas exchange variables were measured breath-by-breath and [HHb] data of the triceps brachii was monitored by near-infrared spectroscopy (NIRS).

All correlations were established for the JT and UT groups, separately, and for a group of heterogenous fitness level consisting of the whole sample.

No significant correlations were found between $\dot{V}O_2$ and [HHb] kinetics and upper body RSA performance both in the JT group and the UT group. However, a strong negative correlation was found between the MAP and the decrease in PPO (↓PPO) between the first and last sprint ($r = -0,74$, $p = 0,002$) and a strong positive correlation was found between the MAP and the accumulated work (ΣWork) throughout the four sprints ($r = 0,83$, $p < 0,001$) when we consider the whole sample as a heterogenous group and in the UT group. A strong negative correlation was found between the peak $\dot{V}O_2$ and the ↓PPO between the first and last sprint ($r = -0,81$, $p < 0,001$) and a strong positive correlation was found between the peak $\dot{V}O_2$ and the ΣWork throughout all sprints ($r = 0,70$, $p = 0,004$) when we consider the whole sample as a heterogenous group and in the UT group. Significant strong negative correlations were observed between the peak $\dot{V}O_2$ and the ↓PPO ($r = -0,83$; $p = 0,022$), the decrease in mean power output (↓MPO) ($r = -0,80$; $p = 0,030$) and the decrease in work (↓Work) performed between the first and fourth sprints ($r = -0,80$; $p = 0,030$) when we consider the whole sample as a heterogenous group and in the UT group. A linear regression found Maximal [HHb] amplitude (Max. A[HHb]) in the fourth sprint and peak $\dot{V}O_2$ to be significant predictors of ΣWork throughout all sprints. When each group is analysed separately, no significant predictors of ΣWork throughout the upper body RSA test were found.

We can conclude that $\dot{V}O_2$ and [HHb] kinetics are not associated with an increased upper body RSA. However, other variables of aerobic fitness seem to be associated with increased upper body RSA performance in a group of individuals with heterogeneous fitness level. This suggests that aerobic fitness variables may play an important role in upper body RSA performance. However, once a certain level of upper body aerobic fitness is attained, other physiological and fitness variables may play a more important role in determining upper body RSA performance.

Keywords: $\dot{V}O_2$ kinetics, Judo, Upper body, Arm crank, Near-infrared spectroscopy, Repeated sprint.

Resumo

O judo é uma modalidade desportiva caracterizada pela execução de períodos de atividade de intensidade máxima, intercedidos por períodos de pausa/recuperação reduzidos, de forma análoga a um regime de trabalho em sprints repetidos. Para além disso, é uma modalidade que solicita de forma preponderante os grupos musculares dos membros superiores. A capacidade de manter o nível de desempenho ao longo de uma sequência de esforços de sprints repetidos parece estar associada à capacidade de recuperar rapidamente entre esforços, o que parece estar em parte dependente da capacidade oxidativa dos grupos musculares implicados no esforço. A determinação da cinética de consumo de oxigénio (cinética do $\dot{V}O_2$) e da cinética da desoxigenação da haemoglobina/mioglobina (cinética de [HHb]) constituem formas não-invasivas de estudar a capacidade oxidativa muscular.

O propósito do presente estudo foi determinar o grau de relação entre variáveis da cinética do $\dot{V}O_2$, cinética da [HHb] e/ou outras variáveis de desempenho aeróbio e variáveis de desempenho em *sprints* repetidos (RSA) de membros superiores (MS), e se estas associações se verificam num grupo de atletas de judo treinados (JT) e num grupo de participantes não-treinados (UT).

Quinze participantes, consistindo num grupo de oito atletas de judo e sete participantes não treinados em modalidades de exercício envolvendo os membros superiores, foram recrutados. Cada participante realizou um teste progressivo máximo num ergómetro de braços para determinação do pico de consumo de oxigénio (peak $\dot{V}O_2$), a potência aeróbia máxima (MAP) e o primeiro limiar ventilatório (VT_1). Numa avaliação subsequente, realizaram duas transições de exercício de carga constante de seis minutos, num domínio de intensidade pesada, a 20% Δ (20% da carga que dista entre a carga ao VT_1 e a MAP), para determinação da cinética do $\dot{V}O_2$ e a cinética de [HHb]. Numa outra avaliação, foi realizado um teste de sprints repetidos de MS (4 sprints, 15-s trabalho, 45-s repouso), em que foram monitorizadas variáveis da cinética de [HHb], bem como o pico de potência (PPO) e potência média (MPO) obtidos em cada sprint, o trabalho total (Work) realizado, e as alterações nas variáveis de desempenho ao longo do teste. As variáveis cardiorrespiratórias foram obtidas com recurso a um sistema de análise de gases *breath-by-breath*, e as variáveis da cinética de [HHb] do tricípite braquial com recurso a espectroscopia de infravermelho-próximo (NIRS).

Todas as correlações foram estabelecidas para o grupo JT e UT, de forma separada, e também considerando a amostra completa como um grupo com estado de treino heterogéneo.

Não parecem existir correlações significativas entre variáveis da cinética do $\dot{V}O_2$ e de [HHb] e o desempenho em sprints repetidos de MS no grupo JT ou no grupo UT. No entanto, foi observada uma correlação negativa forte entre a MAP e o decréscimo no PPO (↓PPO) entre o primeiro e o último sprint ($r = -0,74$, $p = 0,002$) e uma correlação positiva forte entre a MAP e o trabalho acumulado (ΣWork) ao longo dos quatro sprints ($r = 0,83$, $p < 0,001$) quer no grupo heterogéneo, quer no grupo UT. Verificou-se uma correlação negativa forte entre o peak $\dot{V}O_2$ e o ΣWork ao longo dos quatro *sprints* ($r = 0,70$, $p = 0,004$) quer no grupo heterogéneo, quer no grupo UT. Foram observadas correlações negativas fortes significativas entre o peak $\dot{V}O_2$ e o ↓PPO ($r= -0,83$; $p = 0,022$), o decréscimo na potência média obtida (↓MPO) ($r =-0,80$, $p = 0,030$) e o decréscimo no trabalho (↓Work) realizado ($r = -0,80$, $p = 0,030$) ao longo dos quatro sprints quer no grupo heterogéneo, quer no grupo UT. Uma regressão linear revelou que a amplitude máxima de [HHb] no quarto sprint e o $\dot{V}O_2$ pico correspondem a preditores significativos do ΣWork ao longo dos quatro sprints. Quando cada grupo é analisado de forma individual, não se verificaram nenhuns preditores significativos do ΣWork ao longo do protocolo de sprints repetidos.

Pelas observações feitas no presente estudo, é possível concluir que a cinética do $\dot{V}O_2$ e cinética da [HHb] parecem não estar associados a um melhor desempenho em sprints repetidos de MS. No entanto, outras variáveis de aptidão aeróbia parecem estar associadas a um melhor desempenho em sprints repetidos de MS num grupo de participantes com um nível heterogéneo de aptidão física. Tal observação sugere que variáveis de aptidão aeróbia possam ter um papel importante no desempenho de sprints repetidos de MS. No entanto, parece que quando é atingido um determinado nível de aptidão aeróbia, outras variáveis fisiológicas assumem um papel mais importante no desempenho de sprints repetidos de MS.

Palavras-chave: Cinética de $\dot{V}O_2$, Judo, Ergómetro de braços, Membros superiores, Espectroscopia de infravermelhos próximo, *Sprints* repetidos

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%	Percentage
Δ	Difference
20% Δ	20% of the difference between the maximal aerobic power and the workload corresponding to the first ventilatory threshold
20% Δ W	Workload corresponding to 20% of the difference between the maximal aerobic power and the workload corresponding to the first ventilatory threshold
A	Absorbance
A	Amplitude
$A_{phase\ II}$	Amplitude of the primary phase of $\dot{V}O_2$ kinetics
A_{sc}	Amplitude of the slow component phase of $\dot{V}O_2$ kinetics
A'_{sc}	Effective amplitude of the slow component
A[HHb]	Amplitude of response of haemoglobin/myoglobin deoxygenation
ADP	Adenosine diphosphate
ATP	Adenosine triphosphate
A.U.	Arbitrary units
CaO_2	O_2 concentration in arterial blood
CP	Critical Power, the horizontal asymptote of the power output-exercise duration relationship
Cv	O_2 concentration in venous blood
CK	Creatine kinase
CO_2	Carbon dioxide
DO_2	Diffusion coefficient of oxygen
EE $\dot{V}O_2$	Average oxygen consumption at the end of the 6-minute square-wave transition
$\mathcal{E}$	Molar absorptivity
G	Gain, the increase in $\dot{V}O_2$ per increase in external workload (mL $O2.Kg^{-1}.min^{-1}.W^{-1}$)
JT	(trained) Judo athletes
J	Joule

ℓ	Optical pathlength
MLSS	Maximal lactate steady state
HR	Heart rate
H^+	Proton (Hydrogen ion)
HbO_2	Oxyhaemoglobin/myoglobin
Hb_{tot}	Total haemoglobin/myoglobin
[HHb]	Deoxyhaemoglobin/myoglobin
Hz	Hertz
I	Intensity of reflected light, detected by the probe
I_0	Intensity of the light beam emitted by the spectrophotometer/oximeter
Kg	Kilogram
km/h	Kilometre per hour
L/min	Litre per minute
$\dot{V}O_2$ max.	Maximal oxygen consumption rate
Max. [HHb] Sprint 1	Maximal amplitude of haemoglobin/myoglobin deoxygenation attained in the first sprint
Max. [HHb] Sprint 2	Maximal amplitude of haemoglobin/myoglobin deoxygenation attained in the second print
Max. [HHb] Sprint 3	Maximal amplitude of haemoglobin/myoglobin deoxygenation attained in the third sprint
Max. [HHb] Sprint 4	Maximal amplitude of haemoglobin/myoglobin deoxygenation attained in the fourth sprint
MAP	Maximal aerobic power
MPO	Mean power output
MinPO	Minimal power output
↓PPO	Decrease in peak power output or difference in peak power output
↓MPO	Decrease in mean power output or difference in mean power output
min.	Minute
mL. kg^{-1}.$min.^{-1}$	Mililitre per kilogram per minute
mmol/L	Milimole per litre
MRT	Mean response time
N	Sample size

NADH	Reduced form of the nicotinamide adenine dinucleotide
NIRS	Near-infrared spectroscopy
nm	Nanometre
O_2	Oxygen
PPO	Peak power output
Peak $\dot{V}O_2$	Peak oxygen consumption
PCr	Phosphocreatine
Pi	Inorganic phosphate
Peak HR	Peak heart rate achieved during the incremental test
$PmvO_2$	Partial pressures of O_2 in the microvasculature
$PmitoO_2$	Mitochondrial partial pressures of O_2
$\dot{Q}$	Cardiac output
RSA	Repeated-sprint ability
Rel. VT_1_W	Workload corresponding to the onset of the first ventilatory threshold relative to the maximal aerobic power
SIT	Sprint interval training
s	Second
t	Time
τ	Time constant
τ'[HHb]	Effective time constant, the sum of time delay and time constant of [HHb] kinetics
$\tau_{\text{phase II}}$	Time constant of the preimary phase of $\dot{V}O_2$ kinetics
τ_{SC}	Time constant of the slow component phase of $\dot{V}O_2$ kinetics
TD	Time delay
$TD_{\text{phase II}}$	Time delay of the pimary phase of $\dot{V}O_2$ kinetics
TD_{SC}	Time delay of the slow component phase of $\dot{V}O_2$ kinetics
UT	Untrained participant
$\dot{V}CO_2$	Volume of expired carbon dioxide
$\dot{V}E$	Minute ventilation
$\dot{V}E/\dot{V}CO_2$	Ratio between minute ventilation and volume of expired carbon dioxide
$\dot{V}E/\dot{V}O_2$	Ratio between minute ventilation and volume of oxygen consumption
$\dot{V}O_2$	Rate of oxygen consumption
$\dot{V}O_{2baseline}$	Baseline oxygen consumption
$\dot{V}O_{2SC}$	Oxygen consumption rate during the slow componente phase of $\dot{V}O_2$ kinetics

$\dot{V}O_2$ max.	Maximal oxygen consumption
VT_1	First ventilatory threshold
$VT_1\_\dot{V}O_2$	Oxygen consumption rate at the onset of the first ventilatory threshold
VT_1_W	Workload at the onset of the first ventilatory threshold
W	Workload
Work	Work performed
↓Work	Decrease in work performed or difference in work performed, corresponding to the difference between the work performed in first sprint and the work performed in the last sprint
%↓Work	Difference between the work performed in first sprint and the work performed in the last sprint relative to the work performed in the first sprint
ΣWork	Accumulated work

Chapter I – Introduction

1.1 Thesis' purpose and scope

During exercise, the rate of conversion of chemical energy into mechanical energy at the skeletal muscle level determines the exercise intensity which can be sustained. In order to maintain a given exercise intensity, energy must be supplied at the same rate as it is utilized to power muscular contraction. During supramaximal exercise, the rate of utilization of energy reaches its maximal level (Saltin & Gollnick, 1983). During such type of activities, energy, in the form of adenosine triphosphate (ATP), is mainly resynthesized by the anaerobic energy systems (Hargreaves & Spriet, 2006). During short-duration, supramaximal intensity exercise, the high-energy phospate system plays a particular role in ATP resynthesis, particularly phosphocreatine (Poole & Jones, 2012). Over the course of short term exercise, muscle phosphocreatine content can be depleted by 69% compared to resting levels (Norman, Sollevi, Kaijser & Jansson, 1987).

Even though several physical and sport endevours involve a single effort, most sport activities rely on the ability to repeat several efforts over time. The ability to perform such kind of activity repeatedly over time is dependent on the ability to quickly restore phosphocreatine levels, ensuring that high rates of muscular work can be maintained over the course of several high-intensity bouts (Bogdanis, Nevill, Boobis, & Lakomy, 1996). The ability to restore phosphocreatine stores back to near-resting level seems to be dependent on muscular oxidative capacity (Sahlin, 1991). Moreover, as these short-duration efforts are repeated over time, the contribution of the oxidative energy system seems to increase (Gaitanos,Williams, Boobis & Brooks, 1993). These two phenomena highlight the potential contribution of muscle oxidative capacity to the performance of short-duration, supramaximal exercise.

Oxygen uptake ($\dot{V}O_2$) kinetics is the area of research which studies the physiological mechanisms underlying the $\dot{V}O_2$ response to exercise of different intensities (Poole & Jones, 2012). It can also be considered the field which studies the factors that govern the rate at which gas exchange takes place upon imposition of a given workload. Pulmonary oxygen uptake kinetics, measured breath-by-breath using fast response gas analysers, has been considered a surrogate of muscle oxygen uptake kinetics at the onset of exercise (Barstow & Mole, 1987; Grassi et al., 1996). The measurement of $\dot{V}O_2$ kinetics might provide information regarding an athlete's ability to perform and recover from repeated high-intensity efforts. In fact, faster $\dot{V}O_2$ kinetics have been associated with a lesser degree

of metabolic perturbation upon the onset of exercise (Demarle et al., 2001) and during recovery (Rossiter et al., 2002).

More recently, near-infrared spectroscopy (NIRS) has been used to determine the concentration and oxygenation status of biological tissues (Balaban, Mootha & Arai, 1996). Near infrared (NIR) electromagnetical radiation (wavelength of ~700 –900 nm) is potentially able to penetrate several millimeters or more into biological tissues, allowing the detection of the oxygenation status of the main absorbing chromophores in skeletal muscle - haemoglobin (Hb), myoglobin (Mb), and cytochrome oxidase (cytox). Such technology has been used to study tissue oxygen dynamics upon the onset of exercise (Ferrari, Muthalib, & Quaresima, 2011).

Over the course of the 20th century and early 21st century, several studies have been published regarding the kinetics of $\dot{V}O_2$ response in the context of several exercise modalities such as cycle ergometer (Grassi et al., 2003a), arm-cranking (Koppo, Bouckaert & Jones, 2002), canoeing (Inbar, Faina, Demarie & Whipp, 2012), and running (Carter, Pringle, Jones & Doust, 2002), both in healthy untrained individuals (Murias, Kowalchuk & Paterson, 2010) and in trained athletes (Damasceno et al., 2011). These exercise modes/sports activities, involving reproducible motor patters, have been easier to study from a physiological standpoint. However, there are many other sports which are characterized by different movement patterns and activity profile. One such sport is Judo. Up to date, no studies have sought to investigate the characteristics of $\dot{V}O_2$ kinetic response in judo athletes and attempt to determine if there is any degree of association between $\dot{V}O_2$ kinetics response and performance parameters. Such studies would provide a greater understanding of the potential factors that may limit performance over the course of the intermittent-nature efforts that characterize a judo match. Given that judo-specific movements rely extensively on the use of upper body musculature (Franchini, Del Vecchio, Matsushigue & Artioli, 2011), it is of interest to study the $\dot{V}O_2$ kinetics response in the context of upper body exercise – namely, arm cranking.

The aim of the present thesis was to determine if an association could be established between pulmonary $\dot{V}O_2$ kinetics, haemoglobin/myoglobin deoxygenation kinetics ([HHb]), and other parameters of aerobic fitness and upper body repeated sprint performance. Moreover, the present study also sought to investigate which factors might predict upper body repeated sprint performance (expressed in terms of accumulated work),

both in a group of JT and in a group active, but non-specifically upper body trained individuals (UT).

In the second chapter of the present thesis, the review of the literature is presented, providing background information and the context within which this thesis was developed. Several key topics are covered. The main parameters that characterize the kinetics of $\dot{V}O_2$ response to exercise are introduced, along with the characteristics of $\dot{V}O_2$ kinetic response under different exercise-intensity domains. The principles and applications of near-infrared spectroscopy are also introduced. This chapter also covers the characteristics of the kinetics of $\dot{V}O_2$ response to upper body exercise, and the physiology of Judo performance.

The third chapter details all of the methodological considerations and procedures that were used to collect the relevant data for the present thesis, as well as the statistical analysis techniques utilized to analyse the data.

The fourth chapter presents the main results that were observed.

The fifth chapter introduces the critical analysis and discussion of the main results that were observed, establishing a link between the observed results and the initial hypothesis, presents limitations associated with the work that was conducted and presents several suggestions for future investigations.

The final and sixth chapter presents the main conclusions that could be drawn from the present thesis.

Following the sixth chapter, the references that were cited throughout the study are presented.

Chapter II – Literature review

2.1 Historical aspects of the study of skeletal muscle metabolism

The study of $\dot{V}O_2$ kinetics is intimately linked to the study of skeletal muscle metabolism and circulation, as these two aspects play a crucial role in determining how $\dot{V}O_2$ rises in response to a given workload (Hughson, 2009).

The first modern studies on skeletal muscle metabolism date back to the 19th century, when swedish chemist Jons Berzelius (1779-1848) discovered lactate in the skeletal muscles of hunted animals (Von Muralt, 1950).

The 20th century saw the rise of many studies which sought to enlighten several aspects regardings muscular function and metabolism. In a classical paper, Walter Fletcher (1873-1933) and Frederick Hopkins (1861-1947) (1907) reported that resting lactic acid content in amphibian muscle was small but it increased 10 times the resting value when stimulated to contract to fatigue and disappeared when the skeletal muscle was perfused with oxygen.

The collective work of Archibald Vivian Hill (1886-1977), William Hartree (1887-1958) and Otto Meyerhof (1884-1951) led to the lactic acid theory of muscle contraction. According to this theory, the conversion of glycogen to lactic acid would be the major source of energy to power muscular work, and oxygen would be used during recovery to convert lactic acid back to glycogen. Such work contributed to establishing the thermodynamical and biochemical basis of muscular work, and resulted in the awarding of the Nobel Prize in Physiology to Archibal Hill and Otto Meyerhof in 1922.

The lactic acid theory would subsequently be challenged by the work of several researchers. Philip Eggleton (1903–1954), Grace Eggleton (1901–1970) along with Cyrus Hartwell Fiske (1890-1978) and Yellapragada Subbarow (1895-1948), in the mid 1920's, discovered that a phosphagen compound, later determined to be phosphocreatine, decreased during a set of muscular contractions, setting a stage for a new era in the field of muscular metabolism (Mahajan, Saxena & Sarma, 2018).

In the 1930's, the experiments of the Danish physiologist Einar Lundsgaard (1899–1968) and McKeen Cattell (1891-1983), using skeletal muscle preparations injected with iodoacetate, an inhibitor of glycolysis, showed that the conversion of glycogen to lactic did not directly supply energy for muscle contraction. This work was critical in that it brought down the theory proposed by Meyerhof and Hill (Cattell & Lundsgaard, 1933; Brooks &

Gladden, 2003). In another series of works, Lundsgaard observed that skeletal muscle preparations placed in oxygen were able to produce work for longer periods when stimulated to contract, and that phosphagens underwent a smaller decrease in concentration. Such work suggested that the repletion of phosphagens would be brought about by oxidative processes.

In 1929, Karl Lohman (1898-1978), discovered adenosine triphosphate (ATP), and in 1934 proposed that the breakdown of this compound would be responsible for supplying the required energy for muscular contraction. Lohman proposed that ATP would be the compound involved in supplying the direct energy required to power muscular work, and that the breakdown of phosphocreatine by the enzyme creatine kinase would result in the conservation of free energy via the formation of ATP from ADP. This work proved to be of great significance as it linked the hydrolysis of ATP with the breakdown of phosphocreatine, and highlighted that phosphagen transfer was crucial for the conservation of energy, a concept that had a major impact on the fields of physiology and biochemistry, much beyond the study of muscular metabolism (Langen & Hucho, 2008).

2.2 The development of the field of Human gas exchange and oxygen uptake kinetics

The technique of indirect calorimetry, by which metabolic rate – the rate at which the body produces heat – is quantified based on the volume of oxygen consumed and carbon dioxide produced, was a major breakthrough that allowed the study of gas exchange and metabolic demands associated with several types of Human physical tasks (Macfarlane, 2001). This would culminate in major developments in methods and techniques available for studying gas exchange and metabolic rate, which would eventually form the basis of the field of $\dot{V}O_2$ kinetics.

The history of research in the field of indirect calorimetry and gas exchange dates back to the 18th century, when French chemist Antoine Lavoisier (1743 - 1794) along with his collegue Armand Seguin (1767 – 1835), taking advantage of the recent discoveries by Joseph Priestly (1733-1804) and Carl Scheele (1742-1786), which showed that oxygen was necessary for the survival of animals, performed a series of experiments on gas exchange

and respiration in animals and Humans in the 1780's. Lavoisier and associates, however, thought that oxidation and respiration took place in the lungs (Brooks, 2012).

In the 19th century, the work of the german physiologists Heinrich Magnus (1802 - 1870), Hermann von Helmholtz (1821 – 1894) and Eduard Pfluger (1829 – 1910), in which peripheral blood gases and muscular heat production were measured, led to the proposal that the process of respiration – the production of energy through the utilization of oxygen -, would take place in peripheral tissues.

The field of indirect calorimetry and gas exchanged made further progress in the 19th century, as several technical advances made it possible to measure oxygen consumption of exercising Humans. Nathan Zuntz (1847 – 1920), german physiologist, made use of these advancements to study gas exchange in Humans, and study how different diets influenced metabolism and the ability to perform work. At the same time the german school of physiology was involved in the study of gas exchange and muscular metabolism, several british scientists were also becoming proiminent in this field, of which John Scott Haldane (1860 -1936), Claude G. Douglas (1882 – 1963), Frederick Hopkins (1861 – 1947), Walter Fletcher (1873 – 1933), Hartley Lupton (1892 – 1924) and Archibald Hill (1886 – 1977) were the most notable (Barnard & Holloszy, 2003).

In the late 19th century, American scientist Francis Benedict (1870 – 1957), working with Wilbur Atwater (1844 – 1907), measured oxygen consumption during constant workload exercise of varying intensity, and reported that oxygen consumption would rise as a function of an increase in workload (Barnard & Holloszy, 2003).

A major contribution to the *would-be* field of $\dot{V}O_2$ kinetics came from scandinavia. Professor August Krogh (1874 -1949) and Johannes Lindhard (1870-1947) built the first ever electronic cycle ergometer (Krogh & Lindhard, 1913), and a more accurate apparatus to quantify oxygen uptake response to exercise (Krogh & Lindhard, 1920). In 1913, Krogh and Lindhard published one of the first papers detailing the response profile of $\dot{V}O_2$ upon the onset of constant-workload exercise of various intensities, which demonstrated that oxygen consumption seemed to increase first at smaller magnitude, upon the onset of exercise, and then increase at a greater magnitude over several minutes, until a *steady state* is achieved. It was suggested that this second phase of rise in $\dot{V}O_2$ might be attributed to the arrival of deoxygenated blood at the lungs.

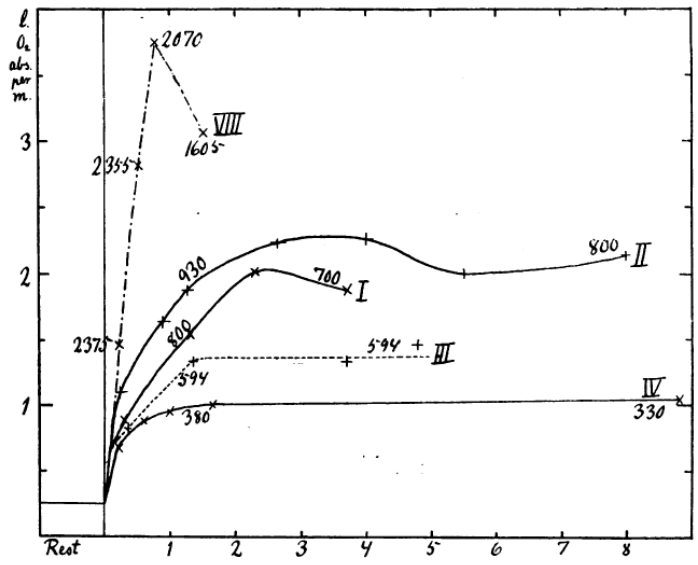

Figure 1. profile of oxygen consumption over time, in response to several workloads. Adapted from Krogh & Lindhard, 1913.

In 1923, Archibald Vivian Hill and Lupton observed that there seemed to be a plateau in $\dot{V}O_2$ at very high running speeds, upon observing that $\dot{V}O_2$ did not rise further after an increase in speed subsequent to a period of prolonged work. Hill and associates (Hill & Lupton, 1923) (Hill, Long & Lupton, 1924) also observed that, after the onset of moderate intensity constant-workload exercise, the rise in pulmonary $\dot{V}O_2$ over time displayed a exponential response, described according to the following equation:

$$\dot{V}O_2(t) = \dot{V}O_{2ss} (1 - e^{-k/\tau}),$$

Where t is the time, $\dot{V}O_{2ss}$ is the increase in steady-state $\dot{V}O_2$ above baseline, *K* is the rate constant of a change in an exponential process (Thomas, Hass, Heil & Wier, 2014). Owing to the exponential nature of the rise in $\dot{V}O_2$ in response to an exercise workload, there is a difference between the required value of $\dot{V}O_2$ and the actual $\dot{V}O_2$.

Hill and Lupton (1892-1924) (1923) proposed that a plateau in $\dot{V}O_2$ suggested that oxidative metabolism could contribute no further to energy production, and that anaerobic energy production would have to make up for the energy required to run at higher running

speeds. Such findings were important for several reasons: 1) it brought about the concept of *maximal oxygen uptake*; 2) It presented a new perspective on the interaction between aerobic and anaerobic metabolism during exercise; 3) It introduced the question regarding what would constitute the limit to the maximal amount of oxygen that could be consumed and utilized.

The development of breath-by-breath gas analysers in the second half of the 20th century led to the rise in the most extensive contributions to the field of study of $\dot{V}O_2$ response to exercise, which took place in the late 1960's and 1970's, when Dr. Karlman Wasserman, Dr. Brian Whipp, and William Beaver layed the foundation for the study of $\dot{V}O_2$ kinetics as it stands today (Jones & Poole, 2005).

Whipp (1971) and Whipp and associates (1982) published several works in which a model fit was applied to the oxygen consumption data, describing the kinetics of the response of $\dot{V}O_2$ to moderate exercise according to a monoexponential function. The rise in $\dot{V}O_2$ was descibed and modeled according to the following equation:

$$\dot{V}O_2(t) = \dot{V}O_{2ss}.(1 - e^{-t/\tau}),$$

where τ is the time constant, equivalent to 1/*k*, and is defined as the time necessary to reach 63% of the final output response (Burnley & Jones, 2007).

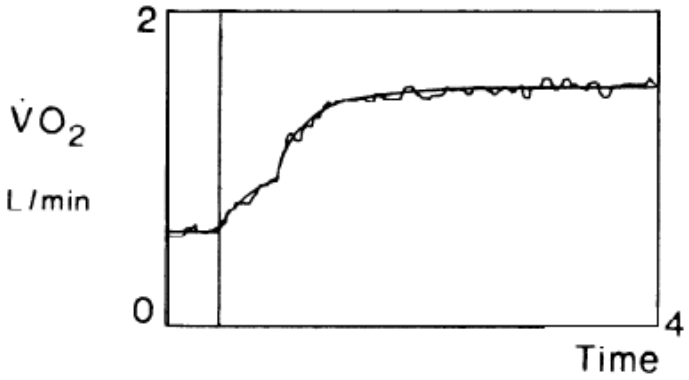

Figure 2. Average $\dot{V}O_2$ response to 8 exercise transitions of 4 minutes of duration, from a workload of 0 watts to 100 watts, during cycloergometer exercise. Adapted from Whipp et al., 1982.

Research work by Knight and associates (1992) and also Poole and associates (1992), would lead to finding a tight coupling between pulmonary $\dot{V}O_2$ and leg $\dot{V}O_2$, suggesting

that pulmonary $\dot{V}O_2$ kinetics reflected muscle $\dot{V}O_2$ kinetics to a great extent. Such findings were later supported by Grassi and associates (2003), which observed a tight coupling between the pulmonary $\dot{V}O_2$ response and mean-response time (MRT) of muscular myoglobin and capillary haemoglobin deoxygenation. Several research observations have supported such findings, showing that $\dot{V}O_2$ of the contracting muscles reflects changes of muscle(s) $\dot{V}O_2$ and phosphagen stores (Rossiter et al., 2002a; Bangsbo, 2000; Krustrup, Jones, Wilkerson, Calbet, & Bangsbo, 2009; Rossiter et al., 2002b).

A fast response in the rise of $\dot{V}O_2$ at the onset of exercise – fast kinetics of $\dot{V}O_2$ response - , as been suggested to be a crucial aspect for human performance, as it results in a smaller O_2 deficit, and hence lesser perturbation in cellular energetic homeostasis for any given rise in workload (Korzeniewski & Zoladz, 2006). Thus, the study of the field of $\dot{V}O_2$ kinetics presents considerable research interest for the study of aspects underlying exercise tolerance and Human performance.

2.3 The profile of $\dot{V}O_2$ under different exercise-intensity domains

The nature of the response of $\dot{V}O_2$ to the onset of constant workload exercise is dependent on the exercise intensity domain over which the individual performs the square-wave transitions, and will determine distinct metabolic responses (Davis, 1985; Gaesser & Poole, 1996; Krustrup et al., 2009).

The profile of rise in $\dot{V}O_2$ upon the onset of constant-workload exercise is characterized by distinct dynamic components. During moderate intensity exercise, coresponding to workloads performed below the intensity corresponding to the first ventilatory threshold (VT_1), pulmonary $\dot{V}O_2$ increases within the first seconds of exercise. This early increase in $\dot{V}O_2$ corresponds to the cardiodynamic component or phase I of $\dot{V}O_2$ kinetics, which lasts circa 15-20 seconds, corresponding to blood flow transit time from the exercising muscle belly to the right atrium. Over this period, the $\dot{V}O_2$ response does not reflect skeletal muscle metabolic demands. Following this initial rise in $\dot{V}O_2$, there is an exponential increase of $\dot{V}O_2$, corresponding to the primary component or phase II of $\dot{V}O_2$ kinetics.

$\dot{V}O_2$ then rises more gradually, until a steady state $\dot{V}O_2$ is attained, corresponding to Phase III of $\dot{V}O_2$ kinetics.

Under the moderate exercise intensity domain, the overall response may be modeled as a time delay phase (TDp, 10-20 s) followed by an exponential phase (Whipp, Davis, Torres & Wasserman, 1981), described according to the following algebraic expression:

$$\dot{V}O_2(t) = \dot{V}O_2 \text{ baseline} + A.(1 - e^{-(t-TDp)/\tau p}),$$

Where $\dot{V}O_2$ baseline corresponds to the oxygen consumption observed before the imposition of a given workload, A corresponds to the amplitude of the response – the difference between baseline $\dot{V}O_2$ and the output $\dot{V}O_2$ response -, TDp corresponds to the time delay which preceeds the increase in pulmonary $\dot{V}O_2$, and τp corresponds to the time constant which represents the time necessary for 63% of the final output response to be complete.

The cardiodynamic component of the $\dot{V}O_2$ response seems to be driven by an increase in pulmonary blood flow in spite of appreciable changes in blood gases, and the quick nature of this phase seems to be related to the fast increase in cardiac output following the onset of exercise, mediated by vagal withdrawal and the "mechanical pump mechanism" of skeletal muscles which increases venous return (Lador et al., 2006), eliciting a rise in stroke volume and heart rate through the Frank-Starling and Bainbridge mechanisms.

During moderate-intensity, constant-workload cycle ergometry exercise, the gain (*G*) of $\dot{V}O_2$, expressed in ml $O_2.min^{-1}.watt^{-1}$, seems to remain fairly constant (Gaesser & Brooks, 1975; Hansen, Casaburi, Cooper, & Wasserman, 1988;Mallory et al., 2002; Poole, Gaesser, Hogan, Knight, & Wagner, 1992).

During heavy intensity exercise, which corresponds to all exercise workloads performed between the exercise intensity corresponding to the VT_1 and the the maximal lactate steady state (MLSS) or the Critical Power (CP) - the horizontal asymptote of the power output – exercise duration relationship (Jones, Vanhatalo, Burnley, Morton & Poole, 2010) -, the $\dot{V}O_2$ response profile becomes more complex. Besides the arise in the cardiodynamic and primary components of the $\dot{V}O_2$ response, a slow rise in $\dot{V}O_2$ above the expected steady-state value is observed after circa 2-3 minutes, which has been termed the slow component of $\dot{V}O_2$ response ($\dot{V}O_{2SC}$)

(Henson, Poole & Whipp, 1989; Poole, Barstow, McDonough & Jones, 2008; Roston et al., 1987).

The MLSS corresponds to the maximal exercise intensity which can be sustained over a prolonged period of time without a sustained rise in blood lactate over time (Billat et al., 2003). The workload corresponding to the MLSS also seems to be the highest workload for which $\dot{V}O_2$ and phosphocreatine (PCr) breakdown can attain a steady state (Beneke & Von Duvillard, 1996; Smith & Jones, 2001).

During heavy intensity exercise, the model fit of the $\dot{V}O_2$ response to exercise can be described by an equation that accounts for the manifestation of the slow component:

$$\dot{V}O_2(t) = \dot{V}O_2 \text{ baseline} + A.(1 - e^{-(t-TDp)/\tau p}) + Asc.(1 - e^{-(t-TDsc)/\tau sc})$$

Where (TDsc) and (τsc) correspond to the time delay and time constant of the $\dot{V}O_2$ slow component ($\dot{V}O_{2SC}$).

The $\dot{V}O_{2SC}$ corresponds to an additional O_2 cost of performing work and represents a reduction in work efficiency, resulting in a delayed attainment of a steady-state $\dot{V}O_2$ for workloads performed within the heavy-intensity domain, leading to a greater metabolic perturbation (Poole, Ward, Gardener & Whipp, 1988). The rise of the SC seems to be related to changes in neuromuscular and metabolic processes, such as the recruitment of lower efficiency motor units. These fibers, which are described as having reduced mechanical-coupling efficiency (less work produced per energy cost, expressed in $Watts.kJ^{-1}$ or $Watts.Kcal^{-1}$), less efficient respiratory coupling (lower ATP phosphorylation per unit of oxygen reduced during oxidative phosphorylation), and lower oxidative potential, contribute to the manifestation of a delayed $\dot{V}O_2$ steady-state, which is of higher magnitude relative to that achieved during moderate intensity exercise conditions (Sahlin, 1991; Tonkonogi, Walsh, Tiivel, Saks & Sahlin, 1999).

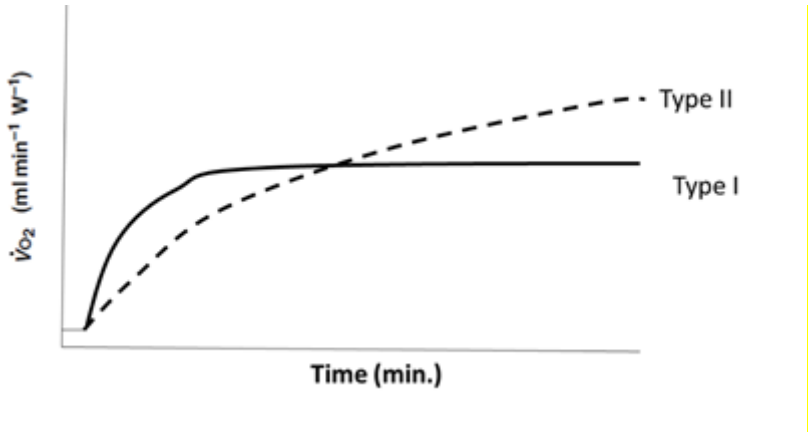

Figure 3. The development of the $\dot{V}O_{2SC}$ as a consequence of the superimposition of the $\dot{V}O_2$ response of type II fibers, recruited at higher magnitudes of exercise intensity, over the $\dot{V}O_2$ response of type I fibers. The slower response kinetics of type II fibers account for the delayed onset of the $\dot{V}O_{2SC}$ and the lower effiency accounts for the "additional $\dot{V}O_2$ cost" of supporting a given workload.

Rossitier and associates (2002), in a series of studies in which Phosphorus Nuclear Magnetic Resonance Spectroscopy (P-31 NMR) was used to investigate skeletal muscle bioenergetics, observed that the manifestation of the $\dot{V}O_{2SC}$ during high-intensity single-leg knee extensions was associated with a progressive decrease in muscle [PCr], which was associated with an increased recruitment in type II fibers (Rossiter et al., 2002).

Several authors have proposed other factors as mediators of the $\dot{V}O_2$sc, such as an elevation in body temperature and increased cathecolamine release (Casaburi, Storer, Ben-Dov & Wasserman, 1987), increased energy cost of cardiac and ventilatory work (Hagberg et al., 1978) and lactate production (Wasserman et al., 1986). However, studies in which catecholamine infusion during exercise was employed as experimental intervention failed to induce a rise in $\dot{V}O_2$ during heavy exercise (Gaesser, Ward, Baum & Whipp, 1994). Koga and associates have also demonstrated that muscle temperature elevation before the onset of exercise does not seem to influence either the phase II response or the slow component of muscle $\dot{V}O_2$ kinetics (Koga, Shiojiri, & Kondo, 1998). The O_2 cost of ventilatory work during heavy exercise has also shown great interinvidual variability (Aaron, Johnson, Seow & Dempsey, 1992). Poole and associates, infusing lactate in isolated dog muscle preparations that were submitted to electrical stimulation, found that lactate did not induce a rise in muscle $\dot{V}O_2$ (Poole, Gladden, Kurdak, & Hogan, 1994). Therefore, these factors have been considered minor putative mediators of the $\dot{V}O_{2SC}$

relative to phemona related to skeletal muscle energetics and neuromuscular function (Poole et al., 1991; Rossiter et al., 2002).

When exercise is performed in the severe intensity domain, corresponding to all workloads performed between the MLSS and the workload eliciting the maximal oxygen consumption - $\dot{V}O_2$ max. -, the profile of $\dot{V}O_2$ response at the onset of exercise is characterized by a cardiodynamic and primary components. However, under such exercise conditions, no steady-state in $\dot{V}O_2$ is attained, and the rise in $\dot{V}O_2$ associated to the slow component may increase over time until $\dot{V}O_2$ max. is attained. Such situation is associated with a continuous reliance on substrate-level phosphorylation for ATP resynthesis which seem to be associated with progressive manifestation of fatigue and exercise intolerance (Hill, Poole & Smith, 2002).

For exercise workloads performed above the intensity corresponding to $\dot{V}O_2$ max., termed supramaximal intensity exercise workloads, the magnitude of the external workload is such that it leads to the quick depletion of phosphocreatine stores and other metabolic events which impair muscular function, leading to the quick onset of fatigue, which might manifest itself before $\dot{V}O_2$ max. can be attained (Jones et al., 2011).

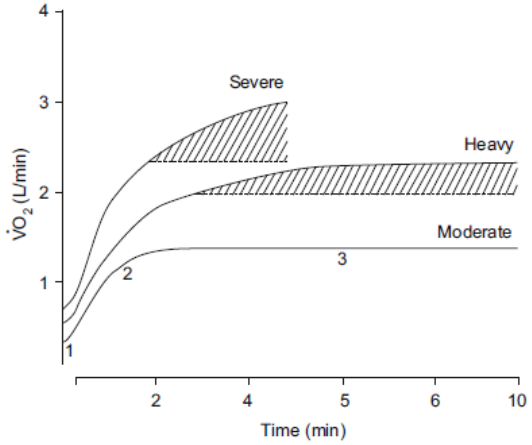

Figure 4. $\dot{V}O_2$ kinetic response during different exercise intensity domains. Adapted from Xu & Rhodes, 1999.

2.4 Factors influencing the control of $\dot{V}O_2$ kinetics

One aspect that has been a source of debate is whether the adjustment of $\dot{V}O_2$ kinetics at the onset of exercise is controlled by oxygen delivery to the active skeletal muscles, or if it is

mainly controlled by changes in intracelular energy charge or phosphorylation potential ([ATP]/[ADP]x[Pi]), reduction-oxidation potential ([NADH/[NAD]), and enzyme activity – namely pyruvate, isocitrate and α-ketoglutarate dehydrogenase (Delp, 1999; Grassi, 2000; Grassi, 2005; Hughson, Tschakovsky & Houston, 2001; Tschakovsky & Sheriff, 2004).

Extensive research work has been carried out in order to understand where the limitations to the control of $\dot{V}O_2$ kinetics may lie (Hughson, Tschakovsky & Houston, 2001; Mcdonough, Behnke, Padilla, Musch, & Poole, 2005).

Oxygen is a crucial substrate for oxidative metabolism, as can be understood from the following balanced chemical equation:

$$6\ ADP + 6\ Pi + 2\ (NADH + H^{+}) + O_2 \rightarrow 6\ ATP + 2\ NAD + 2\ H_2O.$$

A reduction in the rate of oxygen supply can potentially decrease the rate of muscle oxygen consumption (Jensen-Urstad et al., 1995) . Some physiologists propose that there is a limitation in O_2 delivery that slows the rise of $\dot{V}O_2$. Studies have shown that when O_2 delivery is impaired by certain interventions such as artificial hypoxia (Engelen et al., 1996), changes in posture (Hughson, Cochrane & Butler, 1993), prior exercise (Hughson & Morrissey, 1982), as well as beta-adrenergic blockade, which attenuates cardiac output (Hughson, 1984), $\dot{V}O_2$ kinetics seems to be impaired.

Moreover, in conditions of potentially reduced muscle O_2 delivery, such as during arm exercise or leg exercise done in a prone position, increased muscle vasodilatation and oxygen dissociation brought about by prior exercise, is associated with an accelaration of the primary component $\dot{V}O_2$ kinetics (Jones, Berger,Wilkerson & Roberts, 2006; Koppo & Bouckaert, 2005; Fukuba et al., 2004; Scheuermann & Barstow, 2003; Scheuermann, McConnell & Barstow, 2002).

Other group of physiologists proposed that the limits to the control of $\dot{V}O_2$ kinetics lie at the cellular level, and substantiate their position on the basis that several studies have demonstrated that the kinetics of arterial blood flow are appreaciably faster than the kinetics of muscular O_2 consumption (Greenhaff et al., 2002; Grassi et al., 1996; MacDonald, Shoemaker, Tschakovsky & Hughson, 1998), that cardiac output kinetics might be accelerated independently of $\dot{V}O_2$ kinetics (Yoshida, Kamiya & Hishimoto,

1995), and the fact that increased O_2 availability does not seem to increase the speed of $\dot{V}O_2$ kinetics *in situ* (Grassi, Gladden, Stary, Wagner & Hogan, 1998; 2002).

In muscles composed predominantely of slow-twitch fibers, microvascular oxygenation does not fall immediately at the onset of contractions; instead, it decreases exponentially to steady state following a 10- to 20-s delay or exhibits a slight increase (Behnke, Kindig, Musch, Koga & Poole, 2001; McDonough, Behnke, Padilla, Musch, & Poole, 2005), which is suggested as evidence for a metabolic limitation in the rise of $\dot{V}O_2$.

The work of several researchers has provided some evidence that there might be a close coupling between muscle oxygen consumption ($\dot{Q}O_2$) (Haseler, Kindig, Richardson, & Hogan, 2004; Mccreary et al., 1996; Rossiter et al., 2002a; Rossiter et al., 2002b) muscle phosphocreatine hydrolysis kinetics and $\dot{V}O_2$ kinetics during both moderate- and heavy-intensity exercise. It is not clear whether other metabolic processes might be involved in the control of the rate of $\dot{V}O_2$ under other exercise conditions, even though this view is supported by some authors (Zoladz & Korzeniewski, 2003; Robergs, 2014).

Even though these two fields of thought might appear to be in conflict, recent research work as shown that the site of control of $\dot{V}O_2$ kinetics might change as a function of exercise conditions.

Grassi and associates (1998), using isolated dog muscle preparations, reported that setting muscle blood flow at the required level to sustain a given power output did not induce changes in $\dot{V}O_2$ kinetics during moderate and heavy-intensity exercise (Grassi et al., 1998). However, it was observed that such intervention resulted in faster $\dot{V}O_2$ kinetics during severe intensity exercise (Grassi et al., 2000). However, studies in which participants inspired a hyperoxic gas mixture did not result in an acceleration of primary component of $\dot{V}O_2$ kinetics during severe exercise (Wilkerson, Berger & Jones, 2006).

It has been observed that $\dot{V}O_2$ kinetics tend to become slower upon the transition from moderate to heavy-intensity exercise domain workrates. This slowing of $\dot{V}O_2$ kinetics might reflect an increased recruitment of type II muscle fibers (Pringle et al., 2003), whose response might be more dependent on O_2 delivery, due to the lower diffusive oxygen delivery observed in these fibers (Behnke, McDonough, Padilla, Musch & Poole, 2003;

Mcdonough, Behnke, Pdailla, Musch & Poole, 2005). Such findings reinforce the idea that the O_2 delivery and metabolic limitation theories are not conflicting, but rather synergistic.

Poole and Jones (2012) have proposed that there seems to be a 'tipping point' for which $\dot{V}O_2$ kinetics become dependent on either blood flow or "metabolic inertia" – which is a term that encompasses phosphorylation potential, redox potential and enzyme activity. Age, training state, workload and exercise modality seems to determine whether $\dot{V}O_2$ kinetic response is O_2-delivery dependent or limited by metabolic factors.

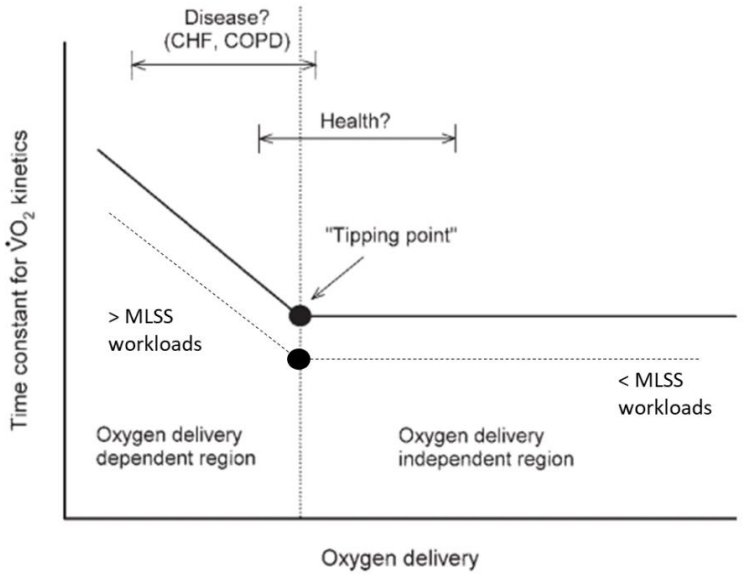

Figure 5. Relationship between O_2 delivery and the time constant for oxygen uptake ($\dot{V}O_2$) kinetics, a *prox*y for the quickness of $\dot{V}O_2$ response. According to this model, $\dot{V}O_2$ kinetics will depend on O_2 delivery under conditions left of the "tipping point", and will be independent of O_2 delivery to the right. Note that besides pathological conditions, which influence this relationship, so too does the magnitude of the exercise workload, with workloads performed above the MLSS dwelling into the O_2 delivery dependent zone, and workloads below the MLSS dwelling into the O_2 delivery independente zone. Adapated from Burnley (2008).

2.5 $\dot{V}O_2$ kinetics during upper body exercise

The cycloergometer has traditionally been the mostly used exercise modallity to study the response of $\dot{V}O_2$ kinetics to the onset of exercise under different conditions (Linnarsson, Karlsson, Fagraeus & Saltin, 1974; Whipp & Wasserman, 1972), and characterise its parameters (Whipp, Davis, Torres & Wasserman, 1981). Nevertheless, research work has

been conducted using other forms of exercise modalities such as treadmill running (Billat, Binsse, Petit & Koralsztein, 1998; Jones & McConnell, 1999), rowing (Ingham, Carter, Whyte & Doust, 2007), swimming (Caporaso & Scurati, 2008) and smaller muscle mass exercise such as arm cranking (Casaburi, Barstow, Robinson & Wasserman, 1992; Koga, 1996; Koppo Bouckaert & Jones, 2002) and knee-extension exercise (Kurstrup et al., 2009; Shoemaker, Hodge & Hughson, 1994). The comparison of the $\dot{V}O_2$ response to different kind of exercise modallities has been used to gain further insight on the different mechanisms of control of $\dot{V}O_2$ kinetics under different conditions.

Given the heterogenity in muscle fiber composition found between the lower and upper body musculature (Barstow, Jones, Nguyen, & Casaburi, 1996; Soderlund & Hultman, 1991; Gollnick, Armstrong, Saubert, Piehl, & Saltin, 1972), and the differences in hemodynamic response (Sawka, 1986) the $\dot{V}O_2$ response to upper body exercise presents considerable differences compared to $\dot{V}O_2$ response to lower body exercise.

It has been reported that the time constant of the primary component of $\dot{V}O_2$ kinetic response to the onset of upper body exercise is slower when compared to lower body exercise (Koppo, Bouckaert & Jones, 2002), both during moderate and heavy exercise-intensity domains (Casaburi, Barstow, Robinson & Wasserman, 1992). Research work conducted by Koppo and associates (2002), where the $\dot{V}O_2$ response to arm cranking and leg cycling were compared during severe exercise, reported that both the time constant of the primary phase of $\dot{V}O_2$ kinetics and the gain (G), expressed in litres of $O_2.Kg.min^{-1}.W^{-1}$, were higher in arm ergometer exercise compared to cycle ergometer exercise, resulting in a slower rise in $\dot{V}O_2$ and revealing a lower efficiency of the muscular groups involved.

During upper body exercise, namely during arm cranking, the arms are closer to the heart, reducing the gravitational effect on hydrostatic pressure, which might also contribute to a delay in convective (bulk flow) blood delivery to the working muscles. It has been shown that both supine and prone exercise, where such gravitational effect is removed, results in slower $\dot{V}O_2$ kinetic response (Convertino, Goldwater & Sandler, 1984; Hughson, Cochrane & Butler, 1993; MacDonald, Shoemaker, Tschakovsky & Hughson, 1998) when compared with upright exercise. Moreover, because the arms are composed predominantely of fibers with a lower capillary density, and therefore a lower vascular cross-sectional area, during high-intensity exercise, due to the high relative forces generated by the small muscle groups of the arms, the vascular cross-sectional area may be further reduced, which may

compromise blood flow delivery (Sawka, 1986). Some authors have observed a shorter mean transit time of blood flow through the microvasculature of the arm muscles, which may further contribute to delayed oxygen delivery to the mitochondria of these muscle groups (Calbet et al., 2005). Koga and associates (1996) have also demonstrated that cardiac output kinetics were slower during both moderate and heavy intensity arm cranking exercise, when compared to cycle ergometer exercise.

Given that the $\dot{V}O_2$ response of type II fibers has been shown to be more dependent on blood flow, and given the greater proportion of type II fibers in upper body musculature, blood flow might constitute a limiting factor in the response of $\dot{V}O_2$ during arm cranking and other forms of upper body exercise.

2.6 NIRS and muscle deoxygenation kinetics

Energy production by oxidative metabolism is dependent on adequate delivery of oxygen ($\dot{Q}O_2$) to meet the metabolic demands ($\dot{V}O_2$) of working tissues. Delivery of oxygen to cytochrome C oxidase, the enzyme that catalyzes the reduction of oxygen in the electron transport chain, can be described by Fick's perfusive O_2 delivery equation and the diffusive O_2 delivery equation (Barstow, 2019):

$$\dot{V}O_2 = Q \times (Ca - C\nu)\, O_2$$
$$\dot{V}O_2 = DO_2 \times (PmvO_2 - PmitoO_2)$$

where $\dot{Q}$ is blood flow; Ca and Cv are O_2 concentrations in arterial and venous blood, respectively; DO_2 is the diffusivity of O_2; and $PmvO_2$ and $PmitoO_2$ are the partial pressures of O_2 in the microvasculature and mitochondria, respectively. Both Fick equations provide insight into the relative balance between $\dot{Q}O_2$ and $\dot{V}O_2$ to skeletal muscle.

The determination of muscular $\dot{Q}O_2$ and $\dot{V}O_2$ has been acessible only through invasive methodology, such has catheterization. Near-infrared spectroscopy (NIRS) has provided a noninvasive methodology for determining relative or absolute global tissue oxygenation, and thus has been able to provide further insights about muscular O_2 consumption dynamics.

Here a brief review is presented regarding the principles of NIRS. Spectroscopy is the study of the interaction of electromagnetic radiation with matter. Matter is composed of subatomic particles – namely protons, neutrons and electrons. Upon incidence of radiation, electrons can be excited and transition between energy levels, emitting energy, which can be quantified as a photon (Newman, 2008).

When infrared, visible, or ultraviolet light incides over a sample of material, some small fraction of the incident photons will be absorbed, which can be detected using absorption spectroscopy. If the incident intensity on the sample is I_0, then the intensity detected after travelling a distance x through the sample with molar concentration c of absorbing molecules is given by the equation:

$$I = I_0 \, e^{-\varepsilon x c},$$

Where I_0 is the incident intensity of light, I is the intensity of light detected after light has travelled x distance, and ε is the molar absorptivity, corresponding to a measure of light absorption at a particular wavelength of the incident electromagnetic radiation. When rewritten taking the logarithm using base 10 as $\log (I/I_0)$, the attenuation of light travelling through material can be related to the concentration of a given analyte according to the Beer-Lambert Law:

$$A = \log (I/I_0) = \varepsilon . [C] . \ell ,$$

Where A is absorbance, ε is the molar absorptivity for a given material, characterizes how easily a volume of material can be penetrated by a beam of light, and ℓ is the light path length. Visible light, with a wavelength of 400–650 nm is unable to penetrate deep tissues. On the other hand, near infrared light (NIR), with a higher wavelength (~700–900 nm), is potentially able to achieve a greater depth, eventually reaching the tissues, where the main absorbing chromophores in skeletal muscle - haemoglobin (Hb), myoglobin (Mb), and cytochrome oxidase (Cox.) - readily absorb radiation at such wavelengths. The concentration of cytochrome oxidase is reported to be insignificant in mammalian muscle when compared with that of haemoglobin and myoglobin (Davis & Barstow, 2013), suggesting the primary sources of the NIRS signals are haemoglobin and myoglobin heme.

It has been reported that in human skeletal muscle, the ratio of Hb to Mb concentration is above 5 (Mancini et al., 1994), therefore, it has been suggested that the main NIRS signal derives from changes in haemoglobin's oxygenation status.

The type of spectrometer which was used in the present thesis is a continuous-wave tissue oximeter. This type of spectrometer cannot account for the extended path length due to scattering, nor losses of light absorbance due to scattering. In continuous wave spectrometery, a light source of constant intensity is transmitted by an emission probe and light intensity is detected by a photon detector, providing only changes in light attenuation. The emission probe emits infrared light at three wavelengths (685, 850, and 980 nm).

The NIRS signal allows for the determination of changes in four main variables:

1. Oxyhaemoglobin/myoglobin [HbO_2] – the relative changes in oxygenated haemoglobin/myoglobin;

2. Deoxyhaemoglobin/myoglobin [HHb] – the relative changes in deoxygenated haemoglobin/myoglobin;

3. Total Heme [tHb] - which is the sum of [HbO2] and [HHb];

4. And Tissue Oxygenation Index – which is the quotient between [HHb] and [tHb].

NIRS has been used to examine the relative matching of oxygen delivery (as $\dot{Q}O_2$) with tissue oxygen utilization ($\dot{V}O_2$) during constant-workload exercise transitions (Ferreira, Poole & Barstow, 2005) (Grassi et al., 2003).

[HbO_2] and [HHb] are variables inversely related to each other, as decreases in tissue [HbO_2] and increases in [HHb] are associated with relative deoxygenation. This holds true if regional blood volume remains constant. Since arterial blood is mostly oxygenated, changes in regional arterial flow are reflected by parallel changes in [HbO_2] if the rate of oxygen consumption remains constant. Because [HHb] is closely associated with changes in venous oxygen content and is less sensitive to arterial blood flow than [HbO_2] is, [HHb] is believed to be a more sensitive measurement of relative tissue deoygenation (DeLorey, Kowalchuk & Paterson, 2003; Ferreira, Koga & Barstow, 2007; Saitoh et al., 2009). The resulting [HHb] signal is indicative of the balance between $\dot{Q}O_2$ and $\dot{V}O_2$ of the probed tissue.

The measurements obtained from NIRS provide insight regarding the level of oxygenation at the microvascular level and allows for an estimate of $\dot{Q}O_2$ and muscle $\dot{V}O_2$ (DeLorey et al., 2003; Mancini et al., 1994). An observed decrease in [HHb] for a given workload suggests an increase in O_2 delivery. Conversely, an increase in [HHb] suggests a greater reliance on O_2 extraction relative to delivery to meet metabolic demands (Grassi et al., 2003).

The [HHb] data has been modelled in a similar way to O_2, in order to determine the kinetics of relative tissue oxygen extraction. Single- and bi-exponential models have been used to fit the [HHb] data for moderate and heavy intensity exercise, respectively.

The parameters obtained from the modelling of [HHb] kinetics include a time-delay (TD), a time constant (τ) for the exponential phase in the rise of [HHb] signal, and an amplitude value of the signal (A). Several authors sum the TD and τ values as an effective time constant (τ') in order to describe the overall [HHb] response dynamics in the primary phase of the response. The [HbO_2] and [TotHb] are usually not reported as the dynamics of the signal does not seem to approximate an exponential function (DeLorey, Paterson & Kowalchuk, 2007).

The dynamics of [HHb] are related to pulmonary $\dot{V}O_2$ kinetic response, with faster [HHb] kinetics relative to $\dot{V}O_2$ kinetic response being associated with a mismatch between muscle O_2 extraction relative to muscle O_2 delivery. (Grassi et al., 2003; Murias, Spencer, Kowalchuk, & Paterson, 2011).

All types of NIRS spectrometers used seem to be universally affected by some errors. One potential aspect that affects the magnitude of the signal is the concentration of melanin in the skin. Melanin content in the skin is directly related to a reduction of [HbO_2] signal (Zonios, Bykowski & Kollias, 2001).

Adipose tissue thickness is also a factor that potentially interferes with the strength of the NIRS signal. An increase in adipose tissue thickness will reduce the relative contribution of the underlying skeletal muscle to the NIRS signal by reducing the effective light penetration into muscle (DeLorey et al., 2003).

2.7 Effects of training on $\dot{V}O_2$ and [HHb] kinetics

Hickson and associates (1978) conducted one of the first studies showing the effects of training on the profile of $\dot{V}O_2$ reponse. Following 10 weeks of an endurance training program consisting of sessions of either cycling or running exercise performed 6 days per week, $\dot{V}O_2$ rose faster to steady state at the same absolute workload and relative workload compared to before the exercise program intervention. Phillips and associates (1995) have also shown that endurance exercise results in a decrease in the mean response time (MRT) in the rise of $\dot{V}O_2$ over the course of 30 days.

Exercise training has also been shown to induce a reduction in the $\dot{V}O_{2SC}$ (Jones et al., 2011). Womack and associates (1995) reported that in a group of 7 participants which participated in a 6-week training program consisting of cyclergometer exercise training performed at an intensity corresponding to 70% of $\dot{V}O_2$ peak, for 40 minutes, 4 days per week, changes in $A\dot{V}O_{2SC}$ were seen after 2 weeks of exercise training. Such changes seemed to be independent of changes in blood lactate concentration. Given that a rise in blood lactate concentration results from an imbalance between lactate release from muscle and the its rate of oxidation in the skeletal muscle as well as other tissue beds (Brooks, 1991), these early changes in $\dot{V}O_{2SC}$ might be independent of metabolic factors, such as changes in mitochondrial density and oxidative enzyme content (Holloszy & Coyle, 1984).

Training may also induce changes in fiber capilarization (Andersen & Saltin, 1985), which may also underlie this initial reduction in the $A\dot{V}O_{2SC}$. More recently, Murias et al. (2010) found strong correlations (r = 0,93 in old participants; r = 0,96 in young participants) between changes in τ of the primary phase of $\dot{V}O_2$ kinetics and changes in local tissue deoxygenation, and proposed that improved matching of blood flow delivery to local utilization might be associated with the observed speeding of $\dot{V}O_2$ kinetics. Such changes might improve the profussion of less oxidative muscle fibers and therefore result in a lesser metabolic demand placed on these fibers (Barstow, Jones, Nguyen, & Casaburi, 2000).

Womack et al. (1995) also proposed that the changes reported in their study regarding the decrease in $\dot{V}O_{2SC}$ might result from changes in muscle fiber recruitment. Thus, it is

possible that changes in tissue blood flow might result in a lesser recruitment of type II muscle fibers, resulting in a concomitantly faster $\tau_{\text{phase II}}$ and a reduced $\dot{V}O_{2SC}$.

Training induced reductions in $\dot{V}O_{2SC}$ have been associated with decreased PCr utilization and a greater exercise tolerance (Jones, Wilkerson, Berger & Fulford, 2007).

While the mentioned studies have used heavy-intensity, continous-exercise training protocols to study training-induced changes in $\dot{V}O_2$ kinetics, other research groups have sought to understand the impact of severe intensity training on the metabolic response to exercise.

Poole and Gaesser (1985) reported that high-intensity interval training (105% of power at $\dot{V}O_2$ max.) resulted in similiar changes in lactate and ventilatory thresholds when compared to moderate-heavy intensity exercise of continuous nature (70% of power at $\dot{V}O_2$ max.), indicating that both severe and moderate-heavy intensity training could induce similiar adaptations. Demarle and associates (2001) reported that following 8 weeks of a specific training program for trained runners, consisting of two sessions of interval training at 93% of the velocity at maximal oxygen consumption, significant decreases were observed for the $\tau_{\text{phase II}}$ of $\dot{V}O_2$ kinetics (mean 26-s pre-training vs. Mean 14-s post training) and in the magnitude of the oxygen deficit (2,27 L pre training vs. 1,52 L post training). These changes were associated with an increased time to exhaustion at $\dot{V}O_2$ max. (400-s pre training vs. 411-s post training).

Bailey and associates (2009) compared the effects of two weeks of two distinct exercise training protocols (30-s x 4 repetitions, all out intensity, known as sprint interval training (SIT) vs. work-matched duration continous training at an intensity equivalent to 90% of the gas exchange threshold (akin to VT_1) on $\dot{V}O_2$ kinetics and quadriceps [HHb] kinetics. Following the 6 sessions, both protocols resulted in decrease in $\tau_{\text{phase II}}$ of $\dot{V}O_2$ kinetics (28-s pre vs. 21-s post in SIT training protocol; 26-s pre vs. 24-s post in continuous training protocol). However, only SIT training protocol resulted in significant changes in muscle [HHb] kinetics following exercise, but only during severe intensity exercise. Interestingly, only the SIT training protocol resulted in significant changes in the $\dot{V}O_{2\ SC}$ during severe-intensity exercise, which might be associated with specific adaptations in certain fibers induced by this supramaximal-intensity training protocol (Iaia et al., 2009).

The authors attributed the observed changes to training-induced metabolic adaptations, as it has been reported by other research groups (Burgomaster et al., 2005), which have used such maximal power output, all-out, short duration protocols, that these protocols might induce significant changes in muscle respiratory capacity. The reduced duration of the intervention (2 weeks) might have been insuficient for training induced changes resulting from continuous exercise to manifest, as it has been shown that these occur over a longer time course (Phillips, Green, MacDonald, & Hughson, 1995; Carter et al., 2000).

More recently, several research groups have also studied the effects of endurance training on [HHb] kinetics. Several authors have reported that the main effect of endurance training (of continuous nature) seems to be the reduction in the mismatch between oxygen delivery and oxygen extraction, despite no significant changes in [HHb] kinetics (Grey et al., 2015; McKay, Paterson & Kowalchuk, 2009; J. M. Murias et al., 2016). However, Bailey and associates (2009) reported significant changes in τ' [HHb] and A[HHb] following a two-week SIT protocol, which the authors attributed to changes in muscle oxidative capacity.

Comparatively lesser work has been published regarding upper body responses to exercise training. In an early study regarding the response of the upper body muscles to exercise, Cerretelli and associates (1979) reported that elite swimmers and kayakers had a faster $\dot{V}O_2$ response during arm cranking exercise when compared with control participants. A later study by Pendergast and associates (1979) revealed that a group of arm-trained athletes (kayakers) displayed a faster $\dot{V}O_2$ response to variable workloads of 62,5 - 200 watts (W).

More recently, Mcnarry, Welsman and Jones (2011) reported that in a group of pubertal girls, the $\tau_{\text{phase II}}$ of $\dot{V}O_2$ kinetics and [HHb] kinetics were faster in a group of swimmers compared to a group of untrained girls, suggesting that training may influence upper body [HHb] kinetics, even though the characteristics of the exercise that may bring about such adaptations remain elusive.

2.8 The role of oxidative metabolism in maximal-intensity exercise performance

During severe and supramaximal exercise, demanding very high power outputs, the rate of energy utilization in skeletal muscle increases manyfold above its resting level (Hultman & Sjöholm, 1983). This increased rate of energy utilization accounts for the increased ATP utilization to power the cross-bridge cycle and the several pumps associated with calcium and ionic flux across the several muscle compartments.

The rate of ATP utilization is determined by the metabolic demands imposed on skeletal muscle, associated with the external workload which is being performed (Spriet, Söderlund, Bergström & Soderlund, 1987).

It has been estimated that the metabolic cost associated with performing intermittent muscular contractions is higher than that associated with a single continuous contraction (Spriet, Soderlund & Hultman, 1988), which has been associated with an increased Ca^{2+} - transport ATPase activity during relaxation and enhanced actomyosin ATPase activity during the early portion of each contraction. Therefore, the ability to quickly replenish ATP stores might be a more critical factor for determining exercise performance during maximal-intensity intermittent exercise (Bogdanis et al., 1996).

While there are several types of activities in which performance is determined by the production of maximal force or power output in a single effort event, such as the 100 m run, there are several activities, such as several competitive sports, which are characterized by the ability to perform several high power output efforts over time, such as Tennis (Kovacs, 2006), Soccer (Bangsbo, Mohr & Krustrup, 2006), and Judo (Franchini, Del Vecchio, Matsushigue & Artioli, 2011). It has been shown that ATP turnover rate decreases over the course of time when performing high-intensity intermittent activity (Gaitanos et al., 1993).

Gaitanos and associates (1993) observed a 64 % decrease in ATP turnover over the course of ten 6-s (seconds) maximal sprints, interceded with 30-s of rest in between.

This decrease in the rate of ATP turnover was associated with a 33,4 % decrease in peak power output and a 26,6 % decrease in mean power output, compared with the first sprint. The authors suggested that the ability to regenerate ATP was a crucial aspect to support high-intensity activity. Over the course of the 10 sprints, an 86,4 % decrease in ATP resynthesis from glycolytic metabolism was reported. The relative contribution of phosphocreatine to the total ATP resynthesis increased from 8,3 % to 13,3 %, suggesting that PCr resynthesis rate might play an important role in maintaining high ATP turnover rates over the course of several high-intensity workbouts. Furthermore, because power output decreased to a less extent when compared to the decrement in ATP turnover rate, the authors suggested that over the course of the 10 sprints there was an increase in the aerobic contribution to the resythesis of ATP, which prevented further decreases in work capacity.

Harris and associates (1976) studied the time course of PCr resynthesis following dynamic and isometric exercise, and found that PCr concentration remained depressed during a recovery period when leg blood flow was occluded, suggesting that oxygen delivery might play a key role in the resynthesis of PCr stores following exercise. Sahlin and associates (1979) also observed that, in isolated muscle fibers, virtually no PCr recovery was observed after 15 minutes when samples were placed in a nitrogen-rich enviroment (4% restored) compared to when samples were placed in an oxygen-rich enviroment (68% restored). These conclusions seem to be supported by researchwork which examined the timecourse of PCr resynthesis following isolated muscular exercise using Phosphorus Nuclear Magnetic Ressoance (Chance, Eleff, Banks, Leigh & Warnells, 1982; Hamaoka, Mccully, Niwayama & Chance, 2011; Sjöholm et al., 1983).

An important consideration when examining the recovery of PCr stores following intense exercise is the possible difference in PCr resynthesis between the two major fibre types (fast and slow twitch). Although PCr is decreased to a similar extent in both fibre types following a maximal 30-s sprint on a non-motorised treadmill (Greenhaff, Bodin, Soderlund, & Hultman, 1994), some researchers have suggested that PCr resynthesis may be slower in the fast, compared with the slow twitch muscle fibres.

Tesch and associates (1989) reported that muscle PCr stores were resynthesised to 50% of its resting value in fast twitch fibers and to 68% of the resting value in slow twitch fibers

60-s following 30 repetitions of maximal knee extension exercise. Soderlund and Hultman (1991) also found PCr concentration to be higher in slow twitch fibers compared to fast twitch fibers following electrical stimulation of the quadriceps for 30-s at a frequency of 20 Hz. These authors suggested that the higher mitochondrial density and capillarization of the slow twitch fibres might account for such findings (Soderlund & Hultman, 1991).

More recent studies have also lent support to the hypothesis that there is heterogeneity in PCr recovery between different fiber types following exercise, with type IIx (Karatzaferi, Haan, Fergunson, van Mechelen & Sargeant., 2001; Casey, Constantin-Teodosiu, Howell, Hultman & Greenhaff 1996; Yoshida, Abe, Fukuoka, 2013) displaying a lower PCr recovery following exercise. However, some research groups have also suggested that during high-intensity exercise and particularly in muscle groups with lower oxidative capacity, other mechanisms may play an important role in determining PCr recovery (Forbes, Paganini. Slade, Towse & Meyer, 2009), suggesting that glycogenolysis may play an important role in PCr recovery in muscles with lower oxidative capacity.

In spite of the noted diffrences, many studies underline the importance of aerobic metabolism in replenishing phosphocreatine stores following exercise (McMahon & Jenkins, 2002). Given that faster $\dot{V}O_2$ kinetics have been associated with faster [PCr] recovery kinetics (Rossiter et al., 2002), it seems that the rate with which an individual can increase $\dot{V}O_2$ towards its maximal level in a given exercise task can have an important role in determining performance in maximal intensity exercise.

Moreover, fast $\dot{V}O_2$ kinetics also seem an important determinant of the maximal power output that can be sustained without continuously depleting phosphocreatine stores and incurring in acidosis (Murgatroyd, Fergunson, Ward, Whipp & Rossiter, 2011), and in the magnitude of the oxygen deficit incurred (Jones & Burnley, 2009), two aspects that determine the magnitude of metabolic perturbation experienced during exercise.

2.9 Importance of $\dot{V}O_2$ kinetics for maximal intensity intermittent exercise performance

High-intensity exercise, particularly of short duration, relies primarily on the supply of ATP by substrate level phosphorylation. Even though it is often assumed that “anaerobic”

or substrate-level resynthesis of ATP is a key determinant of high-intensity exercise performance, it has been shown that the contribution of aerobic metabolism to energy release during high-intensity exercise is quite significant (Medbo & Tabata, 1989)

Given the limited energy supply that can be derived from non-oxidative sources, and that anaerobic ATP resynthesis is associated with a greater magnitude of loss of cellular homeostasis, leading to an earlier the onset of fatigue, the ability to attain a high rate of oxidative energy production quickly would seem important in reducing the reliance on substrate level phosphorylation – and therefore reduce oxygen deficit -, and in delaying the onset of fatigue, two crucial aspects for high-intensity exercise performance. The speed with which $\dot{V}O_2$ rises to attain a given value can be characterized by the τ of the primary component of $\dot{V}O_2$ kinetics ($\tau_{\text{phase II}}$).

In a study which sought to determine the contribution of the aerobic energy system to the performance of 30-s Wingate test, Kavanagh and Jacobs (1988) reported that the aerobic energy system contributed an average of 18,5% to the total work performed, and that the values of aerobic contribution were higher for individuals with higher $\dot{V}O_2$ max. Given that some authors (Powers, Dodd & Beadle 1985) have shown that there is an inverse relationship between the τ of the primary component of $\dot{V}O_2$ kinetics and $\dot{V}O_2$ max., it is possible that individuals with higher $\dot{V}O_2$ max (higher aerobic training status), and therefore faster $\dot{V}O_2$ kinetics, may accomplish the same work with a lower contribution of substrate level phosphorylation, due to a lower magnitude of oxygen deficit.

Several authors have studied the relationship between $\dot{V}O_2$ max. and repeated sprint (RSA) performance in running activities (Aziz, Chia, & Teh, 2000; Balsom, Ekblom, & Sjödin, 1994; Balsom, Gaitanos, Ekblom, & Sjödin, 1994; Dawson, Howarth, Tarnopolsky, Wong, & Gibala, 2003), and have observed that $\dot{V}O_2$ max is inversely associated with accumulated time and speed decrement, and suggested that there might be an important contribution of the aerobic energy system in maintaining performance throughout a repeated sprint exercise task, particularly: (1) in the later work periods of the RSA task, (2) and under conditions of a limited recovery period.

Dupont and associates (2005) studied the relationship between the $\tau_{\text{phase II}}$ of $\dot{V}O_2$ kinetics and RSA performance in a task involving fifteen 40 meter sprints interceded by 25-s of

active recovery, and observed a significant correlation (r = 0,64) between the magnitude of the $\tau_{\text{phase II}}$ and the % of decrement in speed across sprints. The authors suggested that fast $\dot{V}O_2$ kinetics might be associated with a faster rise of $\dot{V}O_2$ towards $\dot{V}O_2$ max., resulting in a greater aerobic contribution during each work period, and a reduction in the oxygen deficit, which might be related to a greater ability to maintain performance during RSA exercise.

Moreover, the authors suggested that the faster $\dot{V}O_2$ kinetics might also be related to a better ability to promote PCr resynthesis during recovery from each sprint. However, we should note that the relative high volume of sprints performed, the relatively short rest periods and the use of active recovery between sprints might have contributed to a greater demand placed in the oxidative energy system during the RSA task, which might not replicate the demands associated with RSA in other contexts, eluding our physiological interpretation of the importance of $\dot{V}O_2$ kinetics for RSA performance.

In contrast, Buchheit (2012), studying a large sample of team-sports athletes, and using shorter distance protocols, found only small correlations between % decrement in sprint performance and $\dot{V}O_2$ kinetics. Moreover, the author reported significant correlations between maximal sprinting speed and peak treadmill sprint in an incremental test and RSA performance indexes.

Given the relationship between peak treadmill speed and running economy reported by several authors (Mendez-Villanueva et al., 2011; Paavolainen, Numella & Rusko, 2000), the author suggested that locomotor function and maximal aerobic speed might be more associated with a greater RSA performance than $\dot{V}O_2$ kinetics parameters, which the author termed “metabolic control” factors.

In regards to upper body exercise, Price and associates (2014), studying the physiological responses to an upper body wingate test, reported that the aerobic energy system contributed an average of 43,5% to the total work performed, which seems unexpected due to the slower oxygen uptake kinetics usually observed in this form of exercise (Pendergast, 1989; Koppo, Bouckaert & Jones, 2002). The standard deviation of this value was relatively large (± 29,3%), and the authors further attributed the reported values to the lower absolute resistance applied throughout the test. Nevertheless, these results seem to

indicate that the aerobic energy system might play an important role in high-intensity exercise performance, including upper-body exercise.

2.10 Physiology of Judo performance and the importance of $\dot{V}O_2$ kinetics

Judo is a grappling sport, and is divided by weight categories, with 7 classes existing both for male and female competitors. The sport is characterized by being a technically and tactically demanding sport, involving several intermittent efforts of high intensity activity (Franchini et al., 2013), that last for 20-30 seconds, which are usually interceded by 5-10 seconds of rest. A Judo match may last up to 4 minutes, and a Judo athlete may perform up to 7 matches, incluiding preliminary rounds, main rounds and finals. While it has been suggested that the high intensity efforts that occur throughout a match are mainly supported by anaerobic ATP resynthesis, ATP resynthesis by the aerobic system may be crucial to the recovery process in between high-intensity efforts and between matches (Franchini et al., 2011).

It has been shown that, in a contest between elite judo athletes, contest winners have a higher activity profile over the course of a match, performing more offensive actions (attacks/techniques) per match (56 offensive actions/match in gold medallists vs. 49 offensive actions/match in silver medallists) (Boguszewski, 2016). These results highlight that for high-level athletes, the ability to perform multiple high-intensity actions over the course of a match may be a crucial aspect in determining a contest winner. Therefore, the study of the factors underlying the ability to perform more high intensity actions over the course of a match in this group of athletes seems to be of relevance.

Thomas and associates (1989) reported that in a group of elite Canadian Judokas, upper body relative peak power output, performed in an arm crank ergometer, averaged 80% of that achieved for the lower body test. These values are considerably higher than those reported by Price and associates (2014) in a group of healthy participants. The higher values in elite athletes reported by Thomas and associates might result from the greater involvement of the upper body muscles during sport specific activity. Relative lower body

peak and mean power outputs were similiar to those reported in a group of physically active males (Maud & Shultz, 1989).

The above-mentioned studies seem to point that anaerobic power and capacity, particularly of the upper body, play a key role during a Judo match. Nevertheless, given that Judo is an intermittent sport that requires athletes to perform several periods of high-intensity efforts throughout a match, each interceded with a very short recovery period, it is suggested that oxidative capacity of the skeletal muscle may play a key role in the maintenance of a high work capacity and activity profile throughout a match (Tomlin & Wenger, 2002), despite the fact that it has been reported that Judo athletes seem to have $\dot{V}O_2$ max. values of 50-60 $ml.kg^{-1}.min^{-1}$ for men and 40-50 $ml.kg^{-1}.min^{-1}$ for women, comparable to those of physically trained participants (Hawkins, Raven, Snell, Stray-Gunderse & Levine, 2007). Nevertheless, these $\dot{V}O_2$ max. values have been reported for graded treadmill testing, which might not be an adequate modality to evaluate the aerobic fitness of Judo athletes, given the higher upper body involvement in the sport activty.

Franchini and associates (2005) have reported a significant correlation between the percentage of $\dot{V}O_2$ max. at lactate threshold and mean power output achieved during an upper body wingate test which was performed 15 minutes following a simulated judo match. Such results might seem to suggest that aerobic power is important for power output recovery following a Judo match. Nevertheless, the lactate threshold values reported by Franchini and associates were obtained during a treadmill test, which might not reflect the predominant muscle groups engaged in the grappling activity associated with a judo match.

Given that muscular oxidative capacity seems to be correlated with a better ability to maintain high power during maximal-intensity exercise (Bogdanis, Nevill, Boobis & Lakomy, 1996), and that upper body musculature is highly engaged during a Judo match (Marques, Drago, Aoki & Moreira, 2017), it seems pertinent that more studies need to be conducted in order to determine the relationship between aerobic fitness parameters and the ability to achieve and maintain high power outputs during upper body exercise in this group of athletes.

Given the background information presented, the present work sought to investigate the relationship between $\dot{V}O_2$ and [HHb] kinetics, as well as other parameters of aerobic fitness, and performance in an upper body RSA test. It was hypothesized that in both regular participants and judo athletes alike lower $\tau_{\text{phase II}}$ and lower τ' [HHb] values would be associated with a better ability to maintain higher power output and accumulate more work over the course of an upper body repeated-sprint test. It was also hypothesized that other parameters of aerobic fitness, such and maximal aerobic power (MAP), might also be associated with a greater ability to maintain power throughout the test and/or accumulate more work.

Chapter III – Methods

3.1 Participants (sample)

For the present study, fifteen participants were recruited, eight of which were trained judokas of national and international level (JT), and seven were untrained individuals (UT).

Table 1 presents the descriptive data regarding the characteristics of the participants involved in the present study.

Table 1. Sample characteristics

Variables	Age (years)	Height (cm)	Body mass (kg)
Judo athletes (JT)	21,1 ± 3,0	172,3 ± 4,5	71,5 ± 7,1
Untrained (UT)	22,6 ± 1,0	172,71 ± 4,5	64,3 ± 5,8

For judokas to participate in the study, they were required to: (1) Have participated in national-level competitions at least 3 years prior to the study; (2) Have been training at least 4 days a week on a regular basis 3 years prior to the study. In order to participate in the test, untrained participants were required to: 1) Not be involved in any sort of upper body training modality – either rhytmic type exercise, such as swimming or kayking, or any sort of weight training for at least 18 months prior to the study.

The participants in both groups were required: (1) To have an age between 17-35 years old; (2) Not to be suffering from any upper body injuries, or recovering from any major upper body injury that occured in the past 12 months (Akoto et al., 2018); (3) Not be taking any medications on a regular basis that could affect the performance in the test (cardiovascular medications, opioids, relaxants, anesthetics, histamine, nitric oxide, asthma-treating drugs, endocrine drugs).

The number of participants atributed to each group was defined by performing a statistical power analysis, considering the study of Mcnarry and associates (Mcnarry et al., 2011). The number of participants was determined by obtaining a power of 1,875 and a bilateral alpha level of 0,05, which resulted in a group sample size of 7 participants. Given the different study conditions and to overcome a possible dropout, 8 participants for each group were recruited.

However, due to nationwide restrictions placed upon education institutions by the SARS-CoV-2 pandemic, one of the participants refrained from participating in the study.

3.2 Procedures and familiarizarion

Prior to the testing procedures, each participant was provided with a document outlining the experimental procedures which were to take place. The document also detailed the potential risks associated with each test. The participants were informed that the results of each test were to remain anonymous, and that the obtained data would be used to test the hypothesis proposed by the present thesis and could be potentially used for other academic purposes.

The participants were also informed that they were free to withdraw from the study at any point, with no prejudice to themselves. Additional explanations were provided to questions posed by the individuals participating in the study. If they agreed to every aspect presented for the study, they were obliged to sign a document giving their informed consent to participate in the study.

Prior to each evaluation or test, every participant was informed of the testing procedures, the risk associated with each procedure and the expected participant's commitment.

Besides the informed consent, each participant also completed a PAR-Q type questionaire before being accepted to participate in the study, in order to collect further information regarding the health status of each individual, and eventual factors/conditions which may have precluded the participation in the study.

3.3 Assessment procedures

Before each test, each participant was provided with practice trials to allow familiarization with the equipment, allowing both the JT and UT participants to become acquainted with the exercise technique, to the face mask and to the ergometer function. Such procedures also served the ocasion for the research team to provide further instruction and detail

regarding the required commitment by each individual, and other important aspects associated with the specific requirements of performing an upper body ergometer test (i.e. participants were to avoid squeezing the ergometer handles very tigthly and should try to remain stationary, only using their upper body to propel the handles).

The participants were asked to present themselves in the laboratory as hydrated and as rested as possible. Participants were required to refrain from drinking any sort of alchoolic beverage and from performing high intensity exercise at least twenty-four hours prior to each test session and to refrain from eating and from taking caffeine two-three hours prior to each test. All tests were conducted approximately at the same time of day (±2h), in order to avoid the effects of chronobiological variability on physiological response (Reilly & Waterhouse, 2009).

Untrained participants were asked to visit the laboratory on three non-consecutive days, whilst trained judokas were provided with more flexibility in order to avoid conflicting with their trainining schedule. However, all sessions were performed at least 48 hours apart, and were all completed within a period of one to two weeks.

3.3.1 Maximal arm crank ergometer graded test

An incremental arm crank ergometer test was performed in order to determine the peak oxygen uptake (Peak $\dot{V}O_2$), VT_1 and maximal aerobic power (MAP) of each participant. All tests were performed on an arm crank ergometer (Lode Angio, Groning, Netherlands), with the participants standing on their feet and with their shoulder joint aligned with the pedal crank axel.

During the test, ventilation and gas exchange data were collected breath-by-breath with a gas analyser (MetaMax 3B, Cortex Biophysik, Leipzig, Germany), after calibration according the manufacturer's instructions. Specifically, the O_2 and CO_2 analyzers were calibrated using ambient air with a gas mixture of known O_2 and CO_2 concentrations. Partial O_2 and CO_2 in ambient air were assumed to be 20,93% and 0,03%, respectively.

The reference gas concentration in O_2 and CO_2 were 16,00% and 5,00%, respectively. The turbine flowmeter was calibrated using a 3-L syringe.

After a warm-up of three minutes with no load applied (0 W), participants started the test with a load of 15 W, followed by 15 W increments every minute, and each participant continued the test until volitional exhaustion was attained. Throughout the test the participants were required to maintain a cadence of 70 ± 5 rpm, as it corresponds to the workload most likely to reduce the static contraction effect associated with a slow cadence, and avoid the early recruitment of type II motor units associated with a faster cadence (Price, Collins, Smith & Goss-Sampson, 2007). All participants were given strong verbal encouragement during the test.

Peak $\dot{V}O_2$ was determined as the highest thirty-second average value attained before participants reached exhaustion.

The VT_1 were estimated from the gas-exchange data, by monotoring the ventilatory equivalents for oxygen ($\dot{V}E/\dot{V}O_2$) and carbon dioxide ($\dot{V}E/\dot{V}CO_2$). The VT_1 was determined by inspection to define the workload at which an increase in $\dot{V}E/\dot{V}O_2$ was observed, with no concomitant increase in $\dot{V}E/VCO_2$ (Reinhard, Müller & Schmülling, 1979).

Throughout the test, the heart rate was monitored (Onrhythm 500, Kalenji, France) and the heart rate value observed in the last stage of exercise was defined as the peak heart rate (Peak HR).

3.3.2 Square-wave transitions

On subsequent days, participants performed two 6 min. constant-workload, heavy intensity (>VT_1 workload) exercise transitions, interspersed with 1-hour rest in between each transition, in order to avoid the effects of prior heavy-intensity exercise on $\dot{V}O_2$ response (Burnley, Doust & Jones, 2006). Each transition was performed at the intensity corresponding to 20 %Δ, determined according to the following equation:

$$W20\%\Delta = [wVT_1 + 0{,}20x\ (w\dot{V}O_2\ Peak - wVT_1)],$$

Where wVT_1 corresponds to the workload that gave rise to the first ventilatory threshold, and $w\dot{V}O_2$ Peak. corresponds to the workload at which peak $\dot{V}O_2$ was elicited.

Each square wave transition was preceded by three minutes where the participants remained stationary while gas exchange data was collected, followed by three minutes of unloaded pedalling (0 W), at a cadence of 70 rpm, in order to determine the baseline oxygen uptake (Baseline $\dot{V}O_2$) and the $\dot{V}O_2$ amplitude associated with the increase in workload.

Fifteen seconds prior to the end of the three-minute baseline period, participants were informed to prepare to transition to the main workload. After the end of the three-minute baseline period, the load was imposed immediately, and participants then performed six minutes of constant-workload arm crank exercise at an intensity of 20% Δ.

Throughout each constant-workload exercise transition, muscle deoxygenation of the long-head of the triceps brachii was monitored using a continuous-wave tissue oximeter (NIMO, Nirox, Brescia, Italy), in order to provide non-invasive estimation of the changes in oxygen saturation of the haemoglobin and myoglobin in the local circulation of the long-head of the triceps brachii. The validity and limitations associated with the measurements obtained via the mentioned oximeter have been reviwed by Rovati and associates (2004).

All NIRS measurements were conducted on the right limb during every testing session. The local skin of each participant's upper arm was initially shaved and cleaned. A probe consisting of a photon emitter and a photon receptor, emitting near-infrared beams with three different wavelenghts (685 nm, 850 nm and 980 nm) was attached to the skin surface of the muscle, and secured with tape in order to minimize movement and prevent loss of near-infrared light signal and constrain the signal emission-recepetion site. The intensity of the incident and detected light signals was continously sampled at a rate of 40 Hz.

To account for the effects of adipose tissue thickness on the NIRS signal, the subcutaneous adipose thickness at the sites where NIRS probes were placed was measured with a caliper (Creative Health, Maryland) and a correction factor was used in the analysis software (Nimo Data Analysis Peak). Because quantitative measurements cannot be made with a

continuous-wave system, which cannot account for path length and light scattering, muscle deoxygenation kinetics were be estimated from the relative changes in [HHb] signal, as it has been shown to less sensitive to relative changes in tissue blood flow (Ferrari, Binzoni, & Quaresima, 1997).

3.3.3 Upper body repeated-sprint test

Participants performed a 6-minute warm-up at 30 W with a cadence of 70 rpm, with three brief sprints during the last 3 minutes of the test, each lasting less than 5-s. The participants were then given 2 minutes of rest before commencing the upper body RSA test in the arm ergometer. The test consisted of four sets of 15-s sprints, with 45-s of passive rest in between. The following figure ilustrates the whole procedure:

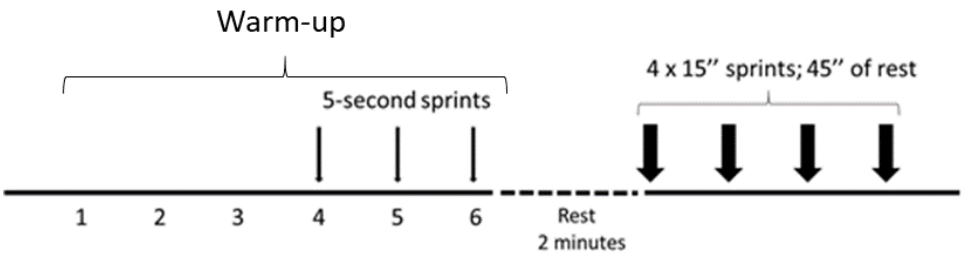

Figure 6. Warm-up and specific repeated sprint test procedures.

Thirty seconds before the start of the sprint test, the participants were asked to grip the ergometer handles. The participants then completed the RSA test. Throughout the whole test, the participants were verbally encouraged to give their maximum effort, and to maintain their work effort until each 15-s work period was completed.

The exercise workload was set at 5% of the body mass of each individual (Tobias et al., 2013). The peak power output (PPO) and mean power output (MPO) attained during each sprint were registered, and the total work performed (Work) during each sprint was calculated as the integral of power generated over the 15-s period.

The 15-s work period was chosen taking into account the data reported by Soriano and associates (Soriano et al., 2019), and was based on the sum of average time it took male

judokas to come to grips (8,4 ±3,1 s), establish a grip and control their opponent (6,1 ±3,5 s) and execute a throw (1,3 ±0,5 s).

In order to analyse the RSA test as a whole, a set of variables were computed, that sought to characterize the overall RSA performance. The decrease in peak power output (↓PPO) was calculated as the difference between the PPO attained in the first sprint and the PPO attained in the fourth sprint; the decrease in mean power output (↓MPO) was computed as the difference between the MPO attained in the first sprint and the MPO attained in the fourth sprint; and the decrease in work performed (↓Work) was calculated as the difference in Work performed in the first sprint and Work performed in the fourth sprint. The percentage decrease in work performed (%↓Work) was calculated as the difference between the Work performed in the first sprint and last sprint, relative to the Work performed in the first sprint. The total work accumulated over the course of the four sprint efforts (ΣWork) was calculated as the sum of the Work performed over the four sprints of the RSA protocol.

3.4 Data analysis

3.4.1 Pulmonary $\dot{V}O_2$ kinetics

The breath-by-breath $\dot{V}O_2$ data from each test was initially examined to exclude errant breaths caused by coughing, swallowing, etc., and those values lying more than 4 standard deviations from the local mean (based on 5 breaths) were deleted. The breath-by-breath data was subsequently linearly interpolated to provide 1-s values. The two repetitions were then time aligned to the start of exercise and jointly averaged to reduce the breath-to-breath noise and enhance the underlying physiological response chracteristics (Barstow, Lamarra, & Whipp, 1987).

$\dot{V}O_2$ kinetics parameters were calculated by an iterative procedure, minimizing the sum of the residuals (squares of the differences between the modeled and the measured $\dot{V}O_2$ values), according to the following bi-exponential equation:

$$\dot{V}O_2\,(t) = \dot{V}O_{2baseline} + A.(1 - e^{-(t-TDp)/\tau p}) + Asc.(1-e^{-(t-TDsc)/\tau sc})$$

where $\dot{V}O_2$ (t) represents the absolute $\dot{V}O_2$ at a given time t, $\dot{V}O_{2baseline}$ represents the mean $\dot{V}O_2$ under unloaded conditions thirty second prior to the work transition; A, TDp, and τp represent the amplitude, time delay, and time constant of the phase II of the increase in $\dot{V}O_2$ after the onset of exercise, and Asc, TDsc, and τsc represent the amplitude of the SC, time delay before the onset of, and time constant of the SC phase of $\dot{V}O_2$ kinetics, respectively (Whipp et al., 1981). The end-exercise $\dot{V}O_2$ was defined as the mean $\dot{V}O_2$ value obtained in the last thirty seconds of the six-minute constant-workload transition.

The first 20-s of $\dot{V}O_2$ data were excluded from the analysis to remove the influence of the cardiodynamic phase on the subsequent response (Whipp, Ward, Lamarra, Davis & Wasserman, 1982).

3.4.2 Muscle deoxygenation kinetics

The [HHb] data was normalized to resting values. The [HHb] response was characterized by a timed-delay (TD) at the onset of exercise, followed by an expontential increase (DeLorey et al., 2003). The TD was defined as the time between the onset of exercise and the time at which a first increase in the [HHb] signal was observed (McKay et al., 2009), which was determined by visual inspection. The [HHb] kinetics was characterized according to a monoexponential model, with an initial TD until the end of the exercise period:

$$[HHb]\ (t) = [HHb]_{baseline} + A[HH_b].(1 - e^{-(t-TD[HHb])/\tau[HHb]})$$

Where [HHb] (t) represents the [HHb] at a given time (t), $[HHb]_{baseline}$ represents the 60-s average [HHb] prior to the gripping the handles, and A[HHb] and τ[HHb] correspond to the amplitude and time constant of the exponential phase of [HHb] kinetics, respectively. The exponential-like phase of the [HHb] kinetics was also characterized by an "effective" time constant (τ′), which corresponded to the sum of TD and τ (McKay et al., 2009).

3.4.3 Statistical analysis

The statistical tests and analysis used in this study were conducted with the Statistical Package for Social Sciences (SPSS Inc., Chicago, IL, USA). Descriptive statistics was applied to characterize the groups. All variables were checked for normality, using the Shapiro-Wilk test.

In order to determine if there were any statistically significant differences between the $\dot{V}O_2$ kinetics variables, local [HHb] kinetics parameters and upper body RSA performance variables between both groups, a Levene test was conducted, to determine the homogenity of variance of the distribution of scores, followed by a t-test for independent samples. If either the assumption of normality or equality of variance could not be assumed, a Mann-Whitney test was performed as a non-parametric alternative.

In order to determine if there were any significant differences between groups in MPO and Work performed in each sprint, a mixed ANOVA for repeated measures was conducted to compare the effects of repeating each sprint on the MPO attained and the Work performed.

To determine the relationship between pulmonary $\dot{V}O_2$ and [HHb] kinetics parameters and upper-body RSA variables, a Pearson's product correlation coeficient test was performed.

In order to determine if a linear relationship could be established between a variable of interest and other independent variables, a regression analysis was performed, using Peak $\dot{V}O_2$, MAP, VT_1 _$\dot{V}O_2$, $A_{phase\ II}$, $\tau_{phase\ II}$, the effective slow component amplitude (A'_{SC}), A_{HHb}, τ'_{HHb}, Max. A_{HHb} 1, Max. A_{HHb} 2, Max. A_{HHb} 3 and Max. A_{HHb} 4 (maximal A[HHb] attained in each sprint) as independent variables, and Σ Work as the dependent variable.

The collected data regarding each individual variable was analysed as a whole, considering all participants as a single heteregoneous group, and was analyzed separately by groups, in order to analyse the significance and meaningfulness of the correlations in each individual group.

The effect size for the differences between groups was calculated based on the quotient between the difference between mean values and the pooled SD. The threshold values for

Cohen effect size (ES, *d*) statistics were > 0,2 (small), > 0,6 (moderate), and > 1,2 (large) (Hopkins, Marshall, Batterham & Hanin, 2009)

Statistical significance was accepted at $p < 0,05$ for all statistical tests performed.

Chapter IV – Results

4.1 Sample characteristics

An independent samples t-test was peformed to compare the differences between mean values for body mass, height, age and tricipital skinfold thickness (4,5 ± 0,8 mm JT vs. 6,6 ± 1,8 mm UT). No significant differences were found between groups for each variable.

4.2. Peak $\dot{V}O_2$, maximal aerobic power and gas exchange thresholds

The variables associated with the incremental test are displayed in table 2.

Table 2. Peak physiological responses upper body ergometer incremental test.

Variables	UT	JT
Peak $\dot{V}O_2$ (ml.kg^{-1}.min^{-1})	32,3 ± 5,1	40,4 ± 3,7 *
MAP (W)	101,4 ± 13,8	149,3 ± 17,4 *
VT_1 W (W)	42,9 ± 20,2	69,4 ± 11,2 *
Rel. VT_1 W (%)	40,9 ± 5,6	46,5 ± 5,6 *
VT_1 _$\dot{V}O_2$ (ml.kg^{-1}.min^{-1})	11,9 ± 1,1	16,9 ± 1,3
20% Δ W (W)	57,1 ± 18,5	86,5 ± 11,7 *
Peak HR (beats/min)	172,4 ± 11,4	177,0 ± 6,4

Peak $\dot{V}O_2$, peak oxygen consumption attained in the incremenetal test; MAP, maximal aerobic power; VT_1_W, workload at the onset of the first ventilatory threshold; Rel. VT_1_WL, workload corresponding to the onset of the first ventilatory threshold relative to the maximal aerobic power; VT_1 _$\dot{V}O_2$, oxygen consumption rate at the onset of the first ventilatory threshold; 20% ΔW, Workload corresponding to 20% of the difference between the maximal aerobic power and the workload correspondent to the first ventilatory threshold; Peak HR, Peak heart rate achieved during the incremental test.

A significantly higher Peak $\dot{V}O_2$ (p = 0,004), workload at VT_1 (VT_1 W) (p = 0,007) and 20%Δ workload (p = 0,003) were observed in the JT group. JT displayed a significantly higher value for the distribution of ranks of MAP compared to the UT group (p = 0,001). No significant differences were found for peak heart rate (peak HR), workload at VT_1

relative to MAP (Rel. VT_1 W), $\dot{V}O_2$ at workload corresponding to VT_1 (VT_1_$\dot{V}O_2$) and % of 20%Δ workload (% Δ W) relative to MAP.

4.3. Pulmonary $\dot{V}O_2$ kinetics

The ̇VO2 kinetics variables derived from fitting the data from the square-wave transitions according to a biexponential model are displayed in table 3.

Table 3. Oxygen uptake kinetics variables derived from the average of the $\dot{V}O_2$ response to two heavy intensity exercise transitions performed with arm crank ergometer.

Variables	UT	JT
$\dot{V}O_{2\ baseline}$ (ml.kg^{-1}.min^{-1})	9,4 ± 1,2	9,1 ± 1,1
A $_{phase\ II}$ (ml.kg^{-1}.min^{-1})	11,5 ± 5,5	15,1 ± 2,8
TD $_{phase\ II}$ (s)	11,4 ± 9,2	10,5 ± 8,0
τ $_{phase\ II}$ (s)	61,6 ± 8,2	47,5 ± 13,4 *
A_{SC} (ml.kg^{-1}.min^{-1})	2,9 ± 0,7	7,0 ± 4,6
A'$_{SC}$ (ml.kg^{-1}.min^{-1})	2,6 ± 0,7	4,9 ± 3,4
TD_{SC} (s)	204,4 ± 74,9	175,6 ± 49,0
τ_{SC} (s)	53,6 ± 26,5	102,2 ± 52,1 *
EE $\dot{V}O_2$ (ml.kg^{-1}.min^{-1})	23,9 ± 6,1	28,4 ± 3,4
A'$_{SC}$/EE $\dot{V}O_2$	0,1 ± 0,1	0,2 ± 0,1
Model R	0,91 ± 0,06	0,97 ± 0,02 *
Sum of residuals	533,2 ± 222,9	369,0 ± 185,6

$\dot{V}O_2$ baseline, baseline oxygen consumption rate; A $_{phase\ II}$, Amplitude of the primary phase of ̇VO2 kinetics; τ $_{phase\ II}$, Time constant of the primary phase of $\dot{V}O_2$ kinetics; $TD_{phase\ II}$, Time delay of the primary phase of $\dot{V}O_2$ kinetics; Asc, Amplitude of the slow component phase of $\dot{V}O_2$ kinetics; A'sc, Effective amplitude of the slow component; TD_{SC}, Time delay of the slow component phase of $\dot{V}O_2$ kinetics; τ_{sc}, Time constant of the slow component phase of $\dot{V}O_2$ kinetics; EE $\dot{V}O_2$, oxygen consumption rate observed at the end of the

square-wave transitions; A'SC/ EE $\dot{V}O_2$, Effective amplitude of the slow component of $\dot{V}O_2$ kinetics relative to the oxygen consumption rate observed at the end of the square-wave transitions; Model R, Fit of the observed data relative to the modelled data; Sum of residuals, Discrepancy in a data set that is not explained by the model.

JT displayed significantly lower $\tau_{\text{phase II}}$ (p = 0,016) and significantly higher τ_{SC}. There were significant differences in the distribution of ranks of model fit for $\dot{V}O_2$ kinetics exponential function (p = 0,034) between groups, with JT displaying a significantly higher value for the model fit. A medium effect size (d = 0,95) was observed for the A $\dot{V}O_{2SC}$/EE $\dot{V}O_2$.

The $\dot{V}O_2$ response of two representative participants from each training group are illustrated in figure 7.

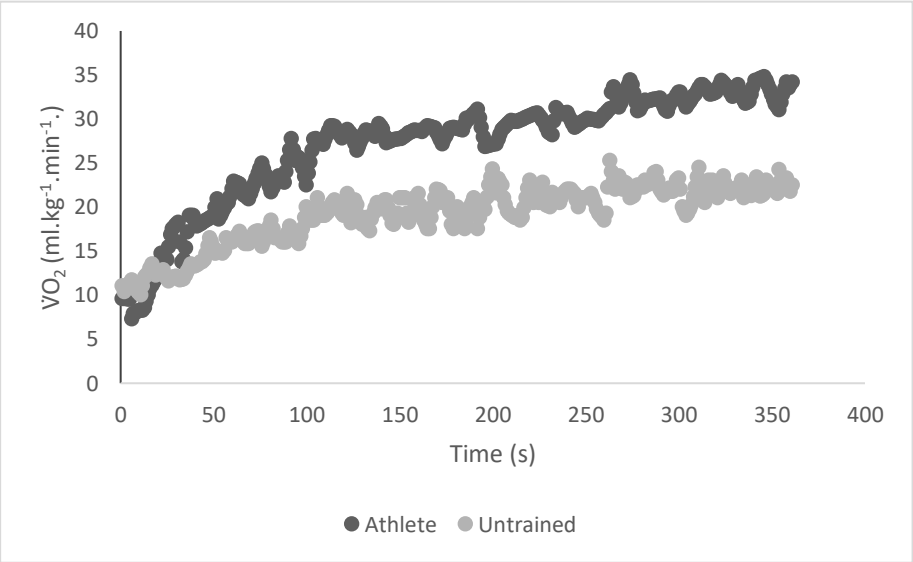

Figure 7. Pulmonary oxygen uptake response to a heavy-intensity, constant workload transition in a representative JT and UT participant (20% Δ). The JT participant is represented in dark grey and the UT particiant in light grey.

4.4 Muscle deoxygenation parameters and NIRS-derived [HHb] kinetics

The normalized parameters of response of [HHb] are presented in table 4.

Table 4. [HHb] kinetics during a heavy intensity exercise transition performed with upper body ergometer.

Variables	UT	JT
τ' [HHb] (s)	36,6 ± 17,1	36,2 ± 10,7
A [HHb] (A.U.)	18,5 ± 13,8	35,4 ± 15,9 *
R squared	0,8 ± 0,2	0,9 ± 0,1

τ'[HHb], Effective time constant, the sum of time delay and time constant of [HHb] kinetics; A[HHb], Amplitude of response of haemoglobin/myoglobin deoxygenation; R squared, Agreement of the model relative to the observed data.

T-tests revealed significant differences between groups (35,4 ± 15,9 JT vs. 18,5 ± 13,8 UT) in the amplitude of response of [HHb] (p = 0,049), with JT displaying a larger A[HHb]. No significant differences were found for any other variables. The effect size for τ' [HHb] was equal to d = 0,03, which is considered a small effect size. The observed effect size for A [HHb] was d = 1,14, which is considered a medium effect.

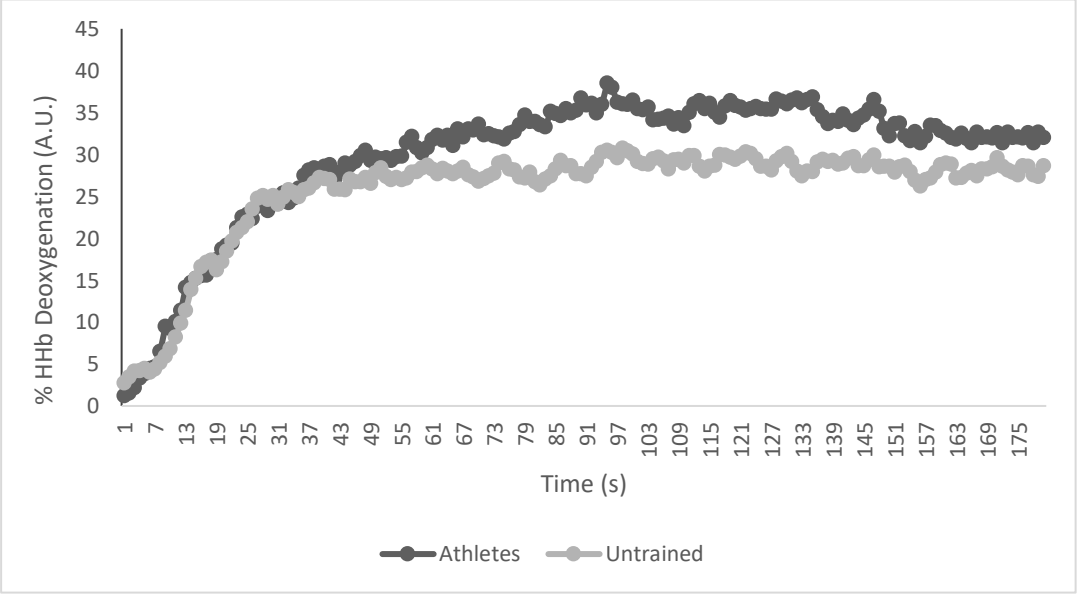

Figure 8. Kinetics of [HHb] response to a heavy-intensity, constant workload transition in a representative JT and UT participant. The JT is represented in dark grey and UT participant in light grey.

4.5 Upper-body repeated sprint performance

The results of a mixed ANOVA for repeated measures revealed that there were significant differences in the MPO attained across repetitions, (F (3,39) = 10,905, $p < 0,001$), and significant differences between groups in the MPO attained across sprints (F (1,13) = 23,469, $p < 0,001$), with JT attaining a higher MPO during each sprint. No significant interaction was observed between MPO and training status (JT or UT). Bonferroni multiple comparisons tests revealed a significant difference in the MPO attained in the third ($p = 0,002$) and fourth ($p = 0,006$) sprints relative to the MPO attained in the first sprint.

Figure 9 presents the mean MPO attained by each group over the course of each single sprint.

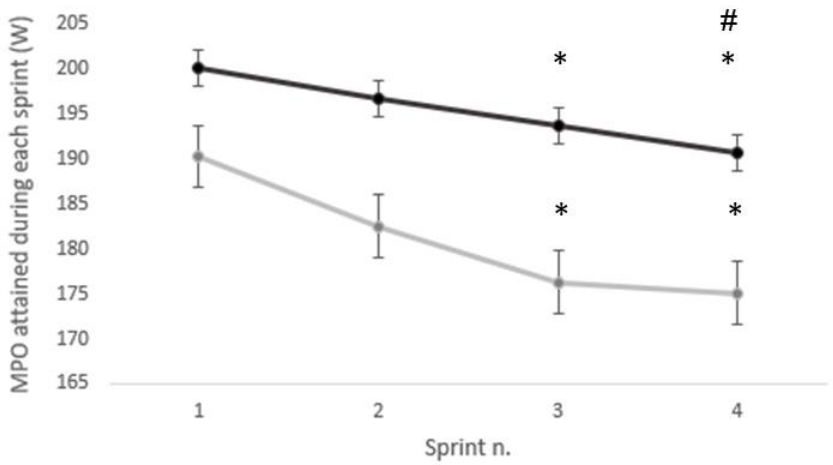

Figure 9. Work performed over the course of each single sprint across the RSA test in each group. The JT is represented in dark grey and UT participant in light grey. Values are means ± SD. *Significantly different from sprint 1; # Significantly different from UT.

There were significant differences in the Work performed across repetitions, (F (3,39) = 11,547, $p < 0,001$), and significant differences between groups in the Work performed in each sprint (F (1,13) = 19,635, $p < 0,001$), with JT performing more work throughout each sprint. No significant interaction was observed between Work and training status. Bonferroni multiple comparisons tests revealed a significant difference in the Work performed in the third ($p = 0,004$) and fourth ($p = 0,005$) sprints relative to the Work performed in the first sprint.

Figure 10 presents a prespective of the mean Work performed by each group over the course of each single sprint.

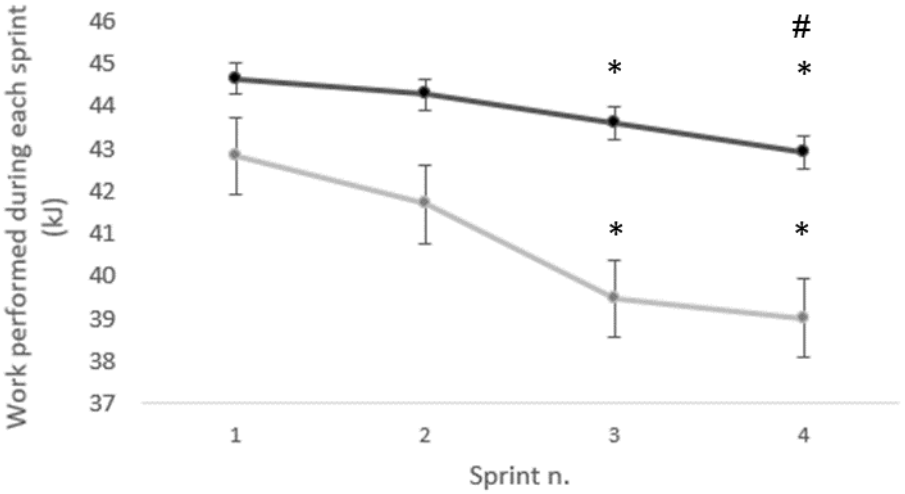

Figure 10. Work performed over the course of each single sprint across the RSA test in each group. The JT is represented in dark grey and UT participant in light grey. Values are means ± SD. *Significantly different from sprint 1; # Significantly different from UT.

Differences in key performance variables observed between the first and fourth upper body sprints in both groups of participants were computed into several variables. The mean values (± SD) for each value are presented in table 5.

Table 5. Differences in key performance variables observed in first vs. fourth upper body ergometer sprint test in both groups of participants.

Variables	UT	JT
↓PPO (W)	42,6 ± 8,8	22,1 ± 14,4 *
↓MPO (W)	14,2 ± 12,0	9,4 ± 9,6
↓ W (kJ)	3,2 ± 2,7	2,2 ± 2,1
% ↓ W	7,4 ± 6,2	4,6 ± 4,8

↓PPO Decrease in peak power output or difference in peak power outpt; ↓MPO Decrease in mean power output or difference in mean power output; ↓ W, decrease in work performed; % ↓ W, Difference in work performed in the last sprint and the first sprint, relative to the work performed in the first sprint.

There were significant differences in the decrease in PPO (↓PPO) between the first and fourth sprint (p = 0,006), with the JT group displaying a lower ↓PPO between the first and last sprint.

A large effect size was observed for the ↓PPO (d = 1,72), a small effect size was observed for the decrease in MPO (↓MPO) (d = 0,44), for the decrease in Work performed (↓Work) (d = 0,42) and for the % ↓Work (d = 0,50). No significant differences were found for any other variables.

Figure 11 presents the data regarding the accumulated work over the course of the four sprints (ΣWork).

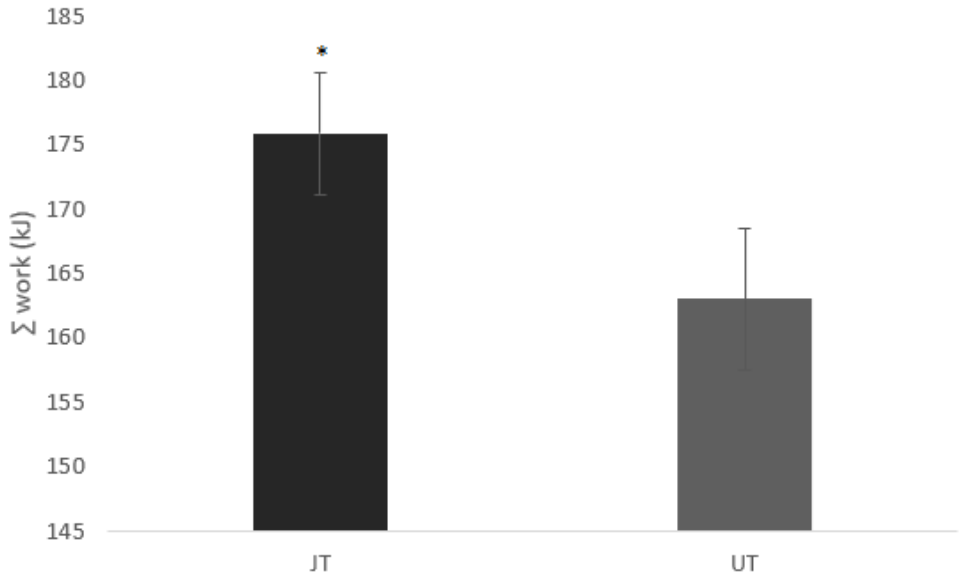

Figure 11. Accumulated work over the course of the four sprints for each group. Values are means ± SD.

Significant differences were observed between groups in the ΣWork over the course of the four sprints (p < 0,001), and a larger mean value of Σ Work was observed in the JT group (175,8 ± 4,8 kJ JT vs. 163,0 ± 5,5 kJ UT). A large effect size was observed for the ΣWork of the four sprints in both groups (d = 2,51).

Table 6 presents the values pertaining to the normalized maximal [HHb] achieved during the course of each 15-s sprint.

Table 6. Maximal A[HHb] during the upper body repeated-sprint test.

Variables	UT	JT
Max. [HHb] Sprint 1 (A.U.)	41,5 ± 11,1	57,3 ± 22,9
Max. [HHb] Sprint 2 (A.U.)	38,5 ± 11,3	59,3 ± 25,7
Max. [HHb] Sprint 3 (A.U.)	36,0 ± 10,4	61,0 ± 31,0
Max. [HHb] Sprint 4 (A.U.)	37,4 ± 12,7	59,9 ± 27,4

Max. [HHb] Sprint 1, maximal amplitude of haemoglobin/myoglobin deoxygenation attained in the first sprint; Max. [HHb] Sprint 2, maximal amplitude of haemoglobin/myoglobin deoxygenation attained in the second sprint; Max. [HHb] Sprint 3, maximal amplitude of haemoglobin/myoglobin deoxygenation attained in the third sprint; Max. [HHb] Sprint 4, maximal amplitude of haemoglobin/myoglobin deoxygenation attained in the fourth sprint.

No statistically significant differences were observed between groups for any of the variables presented. However, a medium effect size was observed for the maximal [HHb] amplitude achieved in the first (*d* = 0,88), second (*d* = 1,05), third (*d* = 1,08) and fourth (*d* = 1,12) sprints.

4.6. Correlation between pulmonary $\dot{V}O_2$ kinetics and upper body repeated-sprint performance

Table 7 presents the values of Pearson's r for the correlation between $\tau_{\text{phase II}}$ and the ↓PPO, ↓MPO, ↓Work performed between the first and last sprint, and the ΣWork over the course of the four sprints.

Table 7. Calculated Pearson's r for the relationship $\tau_{phase\ II}$ of $\dot{V}O_2$ kinetics and the differences in key performance variables attained between the first and fourth sprint, and ΣWork.

Variables	Combined	UT	JT
↓PPO	0,30	-0,04	-0,15
↓ MPO (W)	-0,10	-0,51	-0,15
↓ Work (kJ)	-0,10	-0,51	-0,15
% ↓ Work	-0,10	-0,51	-0,15
ΣWork (kJ)	-0,39	0,30	0,02

↓PPO Decrease in peak power output or difference in peak power outpt; ↓MPO Decrease in mean power output or difference in mean power output; ↓ W, decrease in work performed; % ↓ W, percentage decrease in work performed relative to the total work performed in the first sprint; ΣWork Sum of the total work performed in each of the four sprints.

No significant correlations were found between any of the variables and magnitude of $\tau_{phase\ II}$ when we analyse the sample as a single heteregoneous group or when we consider each group separately.

Table 8 presents the values of Pearson's r for the correlation between the effective amplitude of SC (A'_{SC}) and variables pertaining to the difference in performance between the first and last (fourth) upper body sprints.

Table 8. Calculated Pearson's r for the relationship between differences in key performance variables between the first and fourth sprint and ΣWork, and the effective amplitude of SC ($A'\dot{V}O_{2SC}$) of pulmonary $\dot{V}O_2$ kinetics.

Variables	Combined	UT	JT
↓ PPO (W)	0,10	-0,33	0,27
↓ MPO (W)	-0,12	-0,27	0,22
↓Work (kJ)	-0,12	-0,27	0,22
% ↓ Work	-0,11	-0,26	0,21
ΣWork (kJ)	-0,40	-0,44	0,02

↓PPO Decrease in peak power output or difference in peak power outpt; ↓MPO Decrease in mean power output or difference in mean power output; ↓ W, decrease in work performed; % ↓ W, percentage decrease in work performed relative to the total work performed in the first sprint; ΣWork Sum of the total work performed in each of the four sprints.

No significant correlations were found between any of the variables and A'_{SC} when we analyse the whole sample as a single heterogenous group, or when we analyse each group separately.

4.7 [HHb] kinetics and upper body repeated-sprint performance

Table 9 presents the values of Pearson's r for the correlation between τ' [HHb] and the ↓PPO, ↓MPO, ↓Work performed between the first and last sprint, and the ΣWork over the course of the four sprints.

Table 9. Calculated Pearson's r for the relationship τ' [HHb] and the differences in key performance variables attained between the first and fourth sprint and ΣWork.

Variables	Combined	UT	JT
↓PPO (W)	0,20	-0,13	0,64
↓MPO (W)	0,10	0,11	0,02
↓Work (kJ)	0,10	0,11	0,02
% ↓Work	0,10	0,10	0,03
ΣWork (kJ)	-0,14	- 0,31	- 0,10

↓PPO Decrease in peak power output or difference in peak power outpt; ↓MPO Decrease in mean power output or difference in mean power output; ↓ W, decrease in work performed; % ↓ W, percentage decrease in work performed relative to the total work performed in the first sprint; ΣWork Sum of the total work performed in each of the four sprints.

No significant correlations were found between any of the variables and magnitude of τ' [HHb] when we analyse the sample as a single heteregoneous group or when each group is analysed separately.

4.8 Relationship between $\tau_{phase\ II}$, A'$\dot{V}O_{SC}$ and A[HHb] and individual variables of upper body repeated sprint performance

A Pearson product-moment correlation coefficient was computed to assess the relationship between the $\tau_{phase\ II}$ and the PPO, MPO and Work performed during each sprint. No correlation was found between the $\tau_{phase\ II}$ and any of the individual sprint variables, both when we consider the sample as a single heteregoneous group, and when we analyse each group separately.

A Pearson product-moment correlation coefficient was computed to assess the relationship between the A'$\dot{V}O_{2SC}$ and the PPO, MPO and Work performed during each sprint. No correlation was found between the A'$\dot{V}O_{2SC}$ and any of the individual sprint variables, when we analyse the sample as a single group, or when each group is analysed separately.

A Pearson product-moment correlation coefficient was computed to assess the relationship between the A[HHb] and the PPO, MPO and Work performed during each sprint. Significant positive correlations were found between the A[HHb] and the PPO (r = 0,56; *p* = 0,035) attained in the first sprint and the PPO (r = 0,71; *p* = 0,003) attained in the fourth sprint when we consider the sample as a single heterogenous group. When we analyse each group separately, no correlations were observed between the A[HHb] and individual sprint variables.

4.9 Relationship between $\tau_{phase\ II}$, A'$\dot{V}O_{SC}$ and A[HHb] and upper body repeated sprint performance

A Pearson product-moment correlation coefficient was computed to assess the correlation between the $\tau_{phase\ II}$, A'$\dot{V}O_{2SC}$ and A[HHb] and the variables pertaining to the difference in performance between the first and last sprint as well as the ΣWork across all sprints. A significant correlation was found between the A[HHb] and ΣWork (r = 0,63; *p* = 0,012). No other significant correlation was found, either when we analyse the sample as a single heteregoneous group, or when we analyse each group separately.

4.10 Relationship between MAP, Peak $\dot{V}O_2$ and VT_1_$\dot{V}O_2$ and upper body repeated sprint performance

Table 10 presents the Pearson's r for the correlation between the MAP attained in the incremental exercise test and variables pertaining to the difference in performance between the first and last sprints and the ΣWork.

Table 10. Calculated Pearson's r for the relationship between differences in key performance variables between the first and fourth sprint and ΣWork, and MAP attained in the incremental test.

Variables	Combined	UT	JT
↓PPO (W)	-0,74*	-0,44	-0,45
↓MPO (W)	-0,18	-0,45	0,43
↓Work (kJ)	-0,18	-0,45	0,44
% ↓Work	-0,22	-0,48	0,42
ΣWork (kJ)	0,83*	0,83*	0,21

↓PPO Decrease in peak power output or difference in peak power outpt; ↓MPO Decrease in mean power output or difference in mean power output; ↓ W, decrease in work performed; % ↓ W, percentage decrease in work performed relative to the total work performed in the first sprint; ΣWork Sum of the total work performed in each of the four sprints.

A strong negative correlation was found between the MAP and the ↓PPO between the first and last sprint (r = -0,74, p = 0,002) and a strong positive correlation was found between the MAP and the ΣWork throughout the four sprints (r = 0,83, p < 0,001).

When each group was inspected individually, no significant correlations were found between the MAP attained in the incremental test and the sprint performance variables in the JT group, whilst a strong positive correlation could be observed for the relationship

between the MAP and the ΣWork throughout the four sprints (r = 0,83; *p* = 0,022) in the UT group.

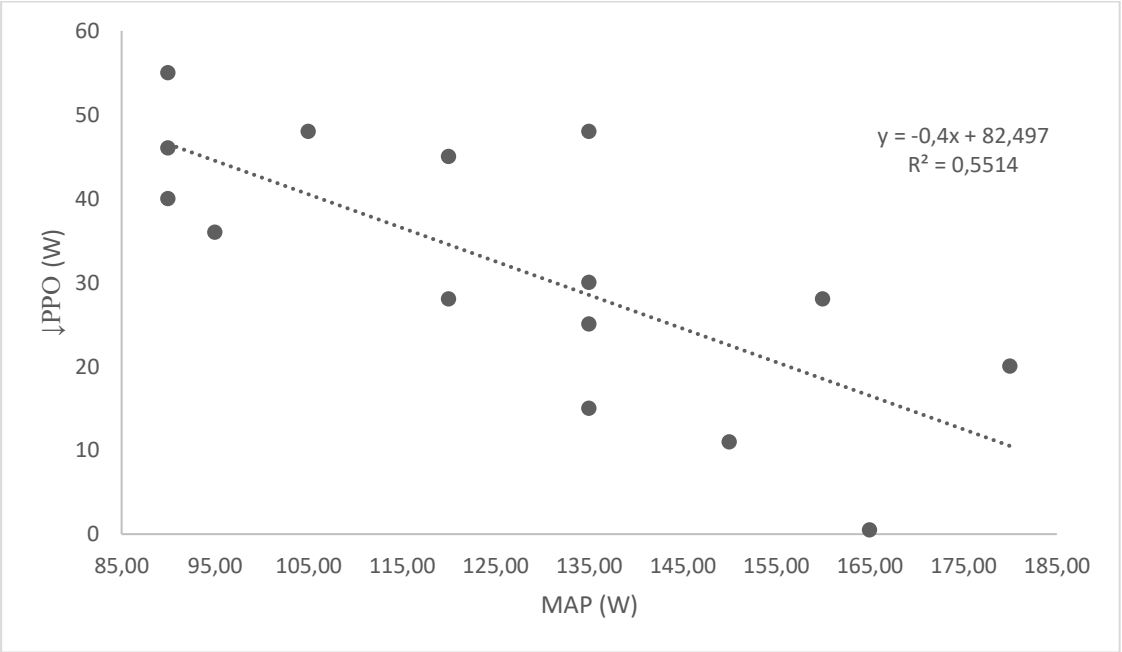

Figure 12. The relationship between decrement in peak power output (W) and maximal aerobic power (MAP) attained in the incremental test.

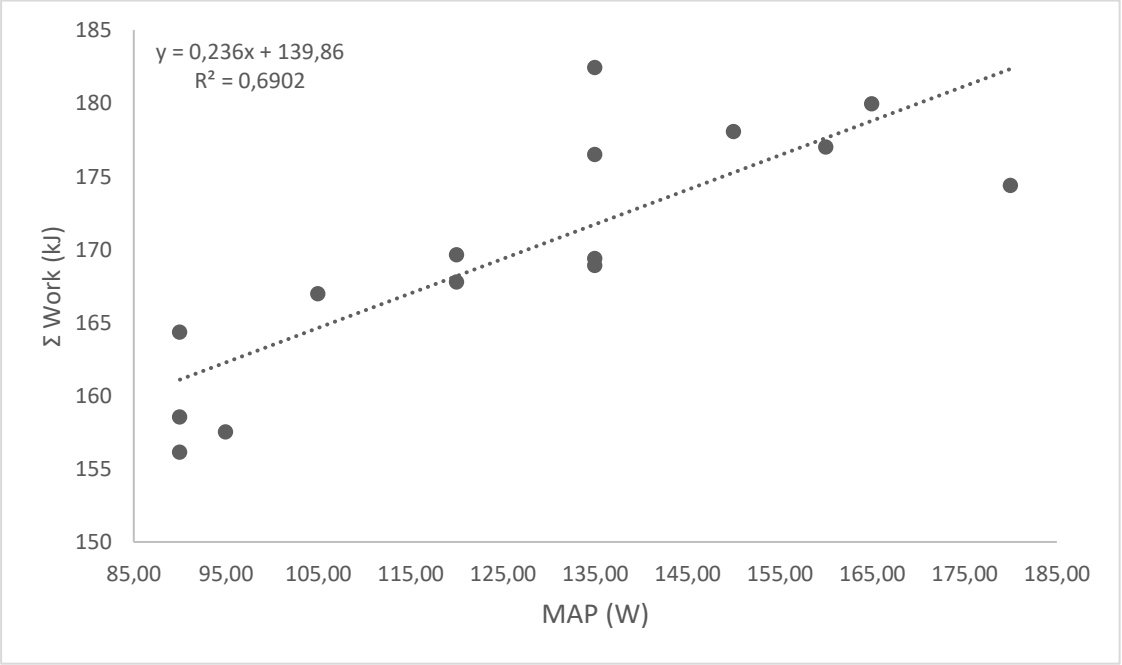

Figure 13. The relationship between accumulated work (kJ) and maximal aerobic power (MAP) attained in the incremental test.

Table 11 presents the values of Pearson's r for the correlation between peak $\dot{V}O_2$ attained in the incremental exercise test and variables pertaining to the difference in performance between the first and last sprint and the ΣWork throughout all sprints.

Table 11. Calculated Pearson's r for the relationship between differences in key performance variables between the first and fourth sprint and ΣWork, and peak $\dot{V}O_2$ attained in the incremental test.

Variables	Combined	UT	JT
↓PPO (W)	-0,81*	-0,81*	-0,61
↓MPO (W)	-0,50	-0,80*	0,03
↓Work (kJ)	-0,48	-0,80*	0,03
% ↓Work	-0,51	-0,82	0,01
ΣWork (kJ)	0,70*	0,49	0,11

↓PPO Decrease in peak power output or difference in peak power outpt; ↓MPO Decrease in mean power output or difference in mean power output; ↓ W, decrease in work performed; % ↓ W, percentage decrease in work performed relative to the total work performed in the first sprint; ΣWork Sum of the total work performed in each of the four sprints.

A strong negative correlation was found between the peak $\dot{V}O_2$ and the ↓PPO between the first and last sprint ($r = -0,81$, $p < 0,001$) and a strong positive correlation was found between the peak $\dot{V}O_2$ and the ΣWork throughout all sprints ($r = 0,70$, $p = 0,004$). When each group is analysed separately, no significant correlation exists between the peak $\dot{V}O_2$ attained and the sprint performance variables in the JT group. Significantly strong negative correlations were observed between the peak $\dot{V}O_2$ attained in the incremental test and the ↓PPO ($r = -0,83$; $p = 0,022$), the ↓MPO ($r = -0,80$; $p = 0,030$) and the ↓Work ($r = -0,80$; $p = 0,030$) in the UT group.

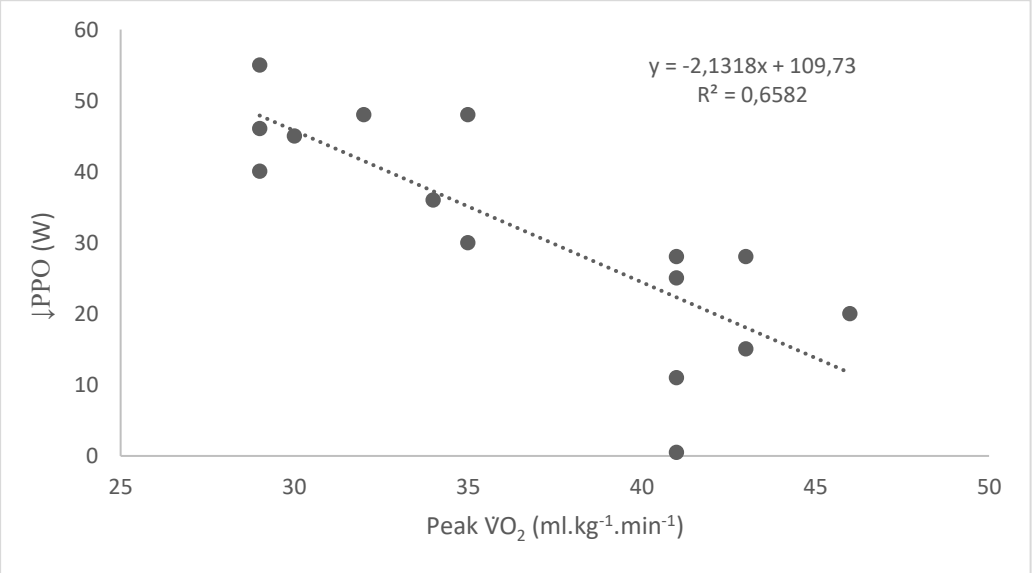

Figure 14. The relationship between decrement in peak power output (W) and peak oxygen uptake ($ml.kg^{-1}.min^{-1}$) attained in the incremental test.

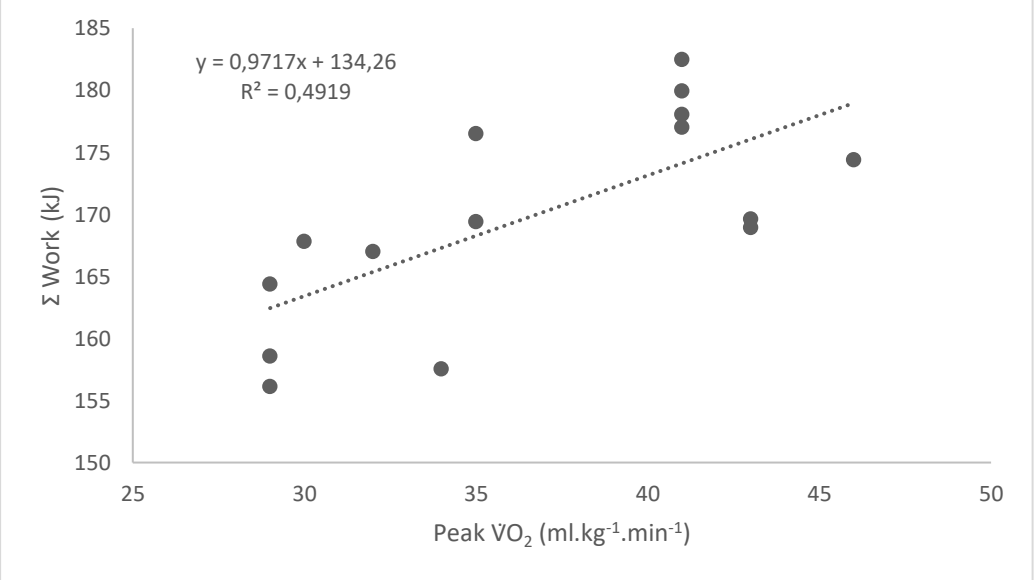

Figure 15. The relationship between accumulated work (kJ) and peak oxygen uptake ($ml.kg^{-1}.min^{-1}$) attained in the incremental test.

Table 12 presents the values of Pearson's r for the correlation between the $\dot{V}O_2$ at the first ventilatory threshold ($VT_1\_\dot{V}O_2$) observed during the incremental exercise test and variables pertaining to the difference in performance between the first and last sprint and the ΣWork throughout all sprints.

Table 12. Calculated Pearson's r for the relationship between differences in key performance variables between the first and fourth sprint and ΣWork, and the measured $\dot{V}O_2$ upon attainement of VT_1 ($\dot{V}O_2$_VT_1) during the incremental test.

Variables	Combined	UT	JT
↓PPO (W)	-0,59*	-0,47	-0,41
↓MPO (W)	-0,06	0,11	0,11
↓Work (kJ)	-0,06	0,11	0,11
% ↓Work	-0,10	0,10	0,10
ΣWork (kJ)	0,61*	0,03	0,42

↓PPO Decrease in peak power output or difference in peak power outpt; ↓MPO Decrease in mean power output or difference in mean power output; ↓ W, decrease in work performed; % ↓ W, percentage decrease in work performed relative to the total work performed in the first sprint; ΣWork Sum of the total work performed in each of the four sprints.

When we consider the sample as a single heteregoneous group, a significant negative correlation exists between VT_1_$\dot{V}O_2$ and the ↓PPO between the first and last sprint (r = -0,59; p = 0,021), and a significant positive correlation exists between VT_1_$\dot{V}O_2$ and the ΣWork across all sprints (r = 0,61; p = 0,015) for the heterogeneous group. When each group is analysed separately, no significant correlations exist between the VT_1_$\dot{V}O_2$ and the sprint performance variables in both groups.

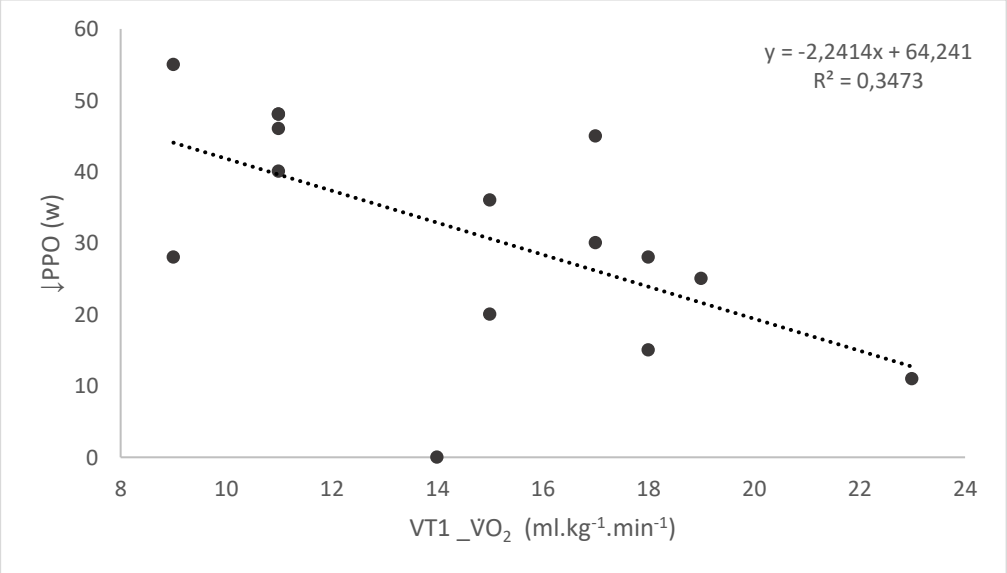

Figure 16. The relationship between decrement in peak power output (W) and oxygen consumption measured upon the attainement of the first ventilatory threshold (VT_1_$\dot{V}O_2$, in $ml.kg^{-1}.min^{-1}$) during the incremental test.

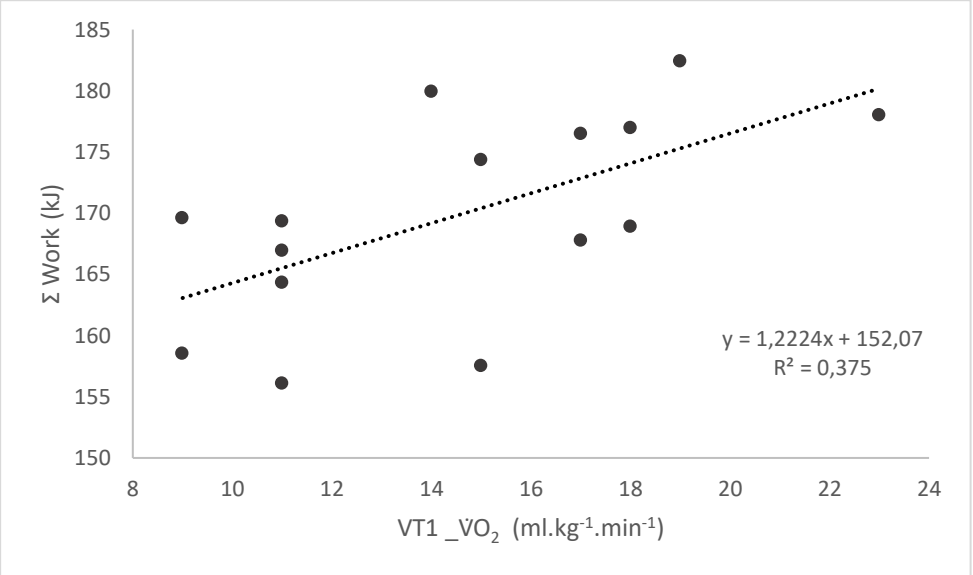

Figure 17. The relationship between accumulated work (kJ) across sprints and oxygen consumption measured upon the attainement of the first ventilatory threshold (VT_1_$\dot{V}O_2$, in $ml.kg^{-1}.min^{-1}$) during the incremental test.

4.11 Predictors of accumulated work over the course of the upper body repeated-sprint test

A linear regression was calculated to predict the ΣWork over the four sprints based on the maximal A[HHb] achieved in the fourth sprint and peak $\dot{V}O_2$. A significant regression equation was found (F (2,12) = 12,737; $p < 0,001$), with an r^2 of 0,68. The participants' predicted ΣWork was equal to 132,91 + 0,8 (Peak $\dot{V}O_2$) + 0,155 (Max. A [HHb] sprint 4) kJ. The participants' accumulated work increased 0,8 kJ for each mL of O_2 consumed per kg of body mass and 0,155 kJ for each arbitrary unit of [HHb] attained in the fourth sprint.

When each group was analysed separately, no variables were found to be predictors of the ΣWork across the four sprints.

Chapter V – Discussion

Discussion

The present study sought to determine if an association could be established between pulmonary $\dot{V}O_2$ kinetics, [HHb] kinetics, and other parameters of aerobic fitness and upper body RSA performance. This was the first study to date which sought to determine if a relationship existed between parameters of aerobic fitness and upper body RSA performance, both is a heterogeneous sample consisting of trained and untrained individuals, and more specifically, in a group of trained judo athletes. The main conclusions to be derived from the current study are the following: 1) There seem to be no significant correlations between pulmonary $\dot{V}O_2$ kinetics – namely, the $\tau_{\text{phase II}}$ - and upper body RSA performance variables; 2) There seems to be no relationship between [HHb] kinetics – namely, τ'[HHb] - and upper body RSA performance variables; 3) There seems to be a strong positive association between MAP, peak $\dot{V}O_2$ and the ΣWork over the course of four 15-s sprints interceded with 45-s of recovery in a heterogeneous group of individuals; 4) No significant association seems to exist between peak $\dot{V}O_2$, MAP, and VT_1 and upper body RSA parameters in a group of trained judo athletes.

The observed results refute our hypothesis that proposed that $\dot{V}O_2$ and [HHb] kinetics, particularly the $\tau_{\text{phase II}}$ and τ'[HHb], would be significantly associated with upper body RSA performance parameters. However, some parameters of aerobic fitness, namely MAP and peak $\dot{V}O_2$, seem to be related to a greater ability to sustain PPO over the course of the four maximal upper body repeated sprints, and to the ΣWork over the course of four sprints, in a heterogeneous group of individuals of variable fitness level.

5.1 Analysis of upper-body repeated sprint performance

The results of the present study reveal a trend for the decrease in MPO and Work performed over the course of four repeated sprints, particularly following the second sprint, and that this trend was similar in both groups, even though the JT group attained higher absolute MPO values during each sprint. These observations likely demonstrate that participants performed at their maximal ability during each sprint.

Gaitanos and associates (1993) observed similar trends for the decrease in MPO over the course of ten 6-s repeated sprints, interceded with 30-s of recovery, performed in a bicycle ergometer. Similar results were also observed by Tobias and associates (2013) during the performance of an upper body ergometer repeated sprint protocol, although it involved longer work periods of 30-s and a longer recovery time of 4 minutes between each sprint. Although these protocols are quite different from the one employed in the present study, they confirm a general trend that can be observed during the performance of maximal intermittent activity with incomplete recovery.

Edge and associates (2006) compared bicycle ergometer repeated sprint performance (5 x 6-s sprints, 30-s rest) in a group of team-sport, endurance-trained and untrained young females. The team-sport athlete group completed significantly more relative total and absolute total work than either the endurance-trained and untrained groups during the repeated-sprint performance test. Despite the different variables analysed, and the major differences in protocols that were used, the authors reported similar findings to those found in the present study, where a similar pattern was observed for the decrease in Work performed, but JT group performed significantly more Work over the course of the upper body RSA test.

5.2 Pulmonary $\dot{V}O_2$ Kinetics and upper-body repeated sprint performance

It has been proposed that $\dot{V}O_2$ kinetics influences high-intensity exercise performance, as expressed by the Critical Power (Jones, Vanhatalo, Burnley, Morton, & Poole, 2010; Murgatroyd et al., 2019; Poole, 2009). Several authors propose that faster $\dot{V}O_2$ kinetics, as expressed by a shorter $\tau_{\text{phase II}}$, are associated with the ability to support a given workload without tapping into O_2 deficit-related metabolic processes (Jones & Burnley, 2009) and that faster $\dot{V}O_2$ kinetics are related with faster [PCr] recovery kinetics following exercise (Chilibeck et al., 1998), two potential aspects that may determine exercise tolerance during high-intensity exercise.

The present study indicates that there are significant differences between JT and UT participants in regards to upper body $\dot{V}O_2$ kinetics parameters. No previous study has reported upper body $\dot{V}O_2$ kinetics parameters in a group of JT. The $\tau_{\text{phase II}}$ values observed

in this group of athletes is similar to that found by Koppo and associates (2002) in a group of physically active males ($\tau_{\text{phase II}} = 48 \pm 12$-s). The values for the $\dot{V}O_2$ kinetics parameters for the group of UT participants observed in this study is similar to that reported by Schneider and associates (2002) in a group of untrained participants ($\tau_{\text{phase II}} = 66{,}4 \pm 3{,}0$-s). Both mentioned studies analysed the $\dot{V}O_2$ kinetics response across the same exercise-intensity range that was used in the present study. By comparison, the $\tau_{\text{phase II}}$ values observed by Invernizzi and associates (2008) in a group of specifically upper body trained participants (elite competitive swimmers; $\tau_{\text{phase II}} = 34{,}3 \pm 8{,}5$-s), determined in an arm crank ergometer test, were much shorter than those which were observed in the JT group.

It is possible that judo-specific training may have induced sufficient training adaptations that resulted in a faster $\dot{V}O_2$-on kinetics response to exercise that is faster relative to untrained participants. However, given that judo-specific drills involve a different skeletal muscle function regimen, where isometric muscular actions of the upper body are emphasized, and muscle actions are performed in an intermittent way, the physiological adaptations that occur may involve very different mechanisms than those which are associated with the performance of high-volume, continuous exercise-training of moderate-heavy exercise intensity, typical of swimming. This may also explain the similar results observed in the upper body $\dot{V}O_2$ kinetics in the JT group compared to a group of physically active males (Koppo et al., 2002).

McNeil and associates (2015) have observed that performing isometric dorsiflexions at 100% of the maximal voluntary contraction (MVC) resulted in significant decreases in NIRS-derived tissue oxygenation compared to performing isometric dorsiflexions at 30% of MVC. Even though these authors reported no significant changes in tibial artery mean blood flow during the course of 60-s of sustained contraction, they suggested that the capillary mean blood-flow might have been severely compromised over the course of the sustained exercise, and that this may have compromised tissue oxygenation dynamics throughout the 100 % MVC exercise periods (Sjøgaard, Savard & Juel, 1988).

Given that over the course of a judo training and competition drills athletes are likely to be exposed so similar conditions, muscle oxygen uptake dynamics might be compromised, and in turn, this may influence the type of physiological adaptations that take place.

Several studies have observed that different training programs, performed at different training intensities have the potential to induce adaptations compatible with shorter $\tau_{phaseII}$ of $\dot{V}O_2$ kinetics. Studies have observed improvements in $\dot{V}O_2$ kinetics with training protocols ranging from low-intensity work at 60% $\dot{V}O_2$ max. (Berger, Tolfrey, Williams & Jones, 2006) to sprint-interval training performed at supramaximal intensities for periods of 30-s, interceded with 4 minute recovery periods (Bailey, Wilkerson, DiMenna & Jones, 2009). Nevertheless, these observations have been reported for studies involving dynamic, running or cycling exercise, which involve a different set of muscle groups and muscle action regimen compared to judo-specific training. As it has already been noted, judo-specific modalities seem to rely more on upper-body musculature (Franchini et al., 2011). Given that upper-body exercise has been associated with different hemodynamic (Calbet et al., 2015) and metabolic responses (Pendergast, 1989; Steinacker, 1996, pp.219-226), physiological adaptations may vary considerably compared to other forms of exercise. In fact, Franchini and associates (2004) reported values of maximal aerobic power of the upper body in a group of trained judo athletes (114,7 ± 26 W) which are lower than those observed in a group of trained swimmers (181,25 ± 11,25 W) (Invernizzi, Caporaso, Longo, Scurati & Alberti, 2008), lending support to the notion that judo-specific modalities may not influence aerobic performance variables to a great extent, compared to moderate-heavy exercise of continuous nature.

The results observed in the current study indicate that there is no significant association between pulmonary $\dot{V}O_2$ kinetics variables and upper body RSA performance, namely between $\tau_{phase\ II}$ and the ↓PPO, ↓MPO and ΣWork over the course of the four sprints, either when we consider the sample of participants as as a single heteregoneous group, or when we analyse each group separetely. These results contradict one of the main hypotheses of the present study, in which we proposed that pulmonary $\dot{V}O_2$ kinetics variables, namely a shorter $\tau_{phase\ II}$, would be associated with improved RSA performance variables, namely a higher ΣWork and smaller decreases in PPO and MPO.

Dupont and associates (2005) have previously reported a significant positive association between $\tau_{phase\ II}$ and relative decrease in speed and total work performed over the course of 15 x 40-meter sprints, interceded with 25-s of active recovery at 50% of maximal aerobic speed. Rampini and associates (2009) also found a significant ($r = 0,62$; $p < 0,05$)

association between $\tau_{phase\ II}$ and the relative decrease in sprint speed over the course of six 40-m (20 m run-and-back) shuttle sprints separated by 20-s of passive recovery.

In contrast, Buchheit (2012) found no strong correlations between RSA ability (RSA) performance and $\tau_{phase\ II}$ ($r = 0,28$; $p < 0,05$), reporting that stepwise multiple regression analyses showed mean repeated-sprint time, best sprint time and incremental test speed (MAS) were the only significant predictors of RSA performance.

Christensen and associates (2010) in turn, found that even though $\tau_{phase\ II}$ was not associated with better RSA performance in a group of soccer players, changes in $\tau_{phase\ II}$ over the course of a speed-endurance training period and an inactivity period were associated with changes in repeated sprint performance over ten 20-m sprints interspersed by 15-s of active recovery, indicating a link between improvements in $\dot{V}O_2$ kinetics and increased repeated sprint performance.

In the present study, a stepwise multiple regression analysis showed that both peak $\dot{V}O_2$ and maximal [HHb] achieved in the fourth sprint were significant predictors of the ΣWork over the course of the four sprints. This may indicate that aerobic performance parameters may play an important role in the ability to maintain a constant performance over the course of several upper body high-intensity efforts, which may be relevant during a judo match.

The studies mentioned above (Buchheit, 2012; Christensen et al., 2010) envolved running activities, utilizing a different set of muscle groups, in different set of participants, exposed to very different training regimens compared to the participants observed in the present study. However, they allow us to make some assertions regarding the results observed in the present study. The protocol used in the study by Dupont and associates involved significantly more volume, and involved active recovery periods, which may have biased the contribution of the aerobic energy system to the total work performed, and therefore the degree of association between RSA performance and $\tau_{phase\ II}$, while Buchheit, using a set of lower volume RSA protocols (10 × 30 m; 6 × 2 × 15 m; 6 × 16 m; 6 × 16 m; 20 × 15 m; 6 × 25 m) did not find such correlations. It is probable that an association may only be found between $\tau_{phase\ II}$ and RSA performance envolving a high volume of repeated-sprint activity, given the increased contribution of the aerobic system as the number of sprints increases (Gaitanos et al., 1993). It is possible to assume that an association between $\tau_{phase\ II}$ and RSA performance may have been found for the present study if a protocol

envolving a higher volume of sprints had been used. In turn, it may be possible that $\dot{V}O_2$ kinetics of the upper body musculature play a more important role in judo contests that drag over a longer period of time.

Given that in the present study the total sprint time was higher than in the total sprint times reported by Dupont and associates, we expected to find an association between % decrease in PPO and $\dot{V}O_2$ kinetics parameters. Such association could be expected given the link that has been established between oxidative metabolism and recovery of muscle PCr stores (Harris et al., 1976; Haseler et al., 1999). Given that upper-body musculature is composed of a greater % of fast-twitch muscle fibers, and that isolated fast-twitch muscle fibers have been reported to have slower oxygen consumption dynamics (0,53 fast twitch vs. 0,77 slow twitch umol $O_2.kg^{-1}.min^{-1}$) (Kushmerick et al., 1992) and slower phosphocreatine recovery rates (92 ± 4.6% slow twitch vs. 66,3 ± 7.6% fast twitch, relative to resting values, expressed in $mmol.kg^{-1}$ of dry tissue, 60-s after the work period) (Casey et al., 1996; Karatzaferi et al., 2001; Tesch et al., 1989), this may explain why no association was found between $\dot{V}O_2$ kinetics and upper-body RSA performance.

It has been shown that there are differences in the fibre type composition and functional properties between the upper and lower body musculature, with the lower body displaying a higher proportion of type I fibers and higher mitochondrial content (Essén, Jansson, Henriksson, Taylor & Saltin, 1975; Gollnick et al., 1972; Johnson, Polgar, Weightman & Appleton, 1973), whilst the upper body musculature displays a greater propotion of type II fibers and lower mitochondrial content. It is possible that because of these biochemical and functional differences, there may be a greater decrease in intracellular energy charge (([ATP] + 0,5[ATP])/([ATP] + [ADP] + [AMP])) in the muscle fibers of the upper body over the course of high-intensity upper body activity. Given that [AMP] is one of the main intermediates which activates glycolysis (Dzeja & Terzic, 2009), it is possible that glycolysis may play a greater role in ATP resynthesis during high-intensity upper body exercise, which would also help explain the inability to establish a correlation between $\tau_{\text{phase II}}$ and upper body RSA variables in the present study.

5.3 $\dot{V}O_{2\ SC}$ and upper-body repeated-sprint performance

Berger and Jones (2007) observed that the A$\dot{V}O_{2SC}$, expressed as the difference in $\dot{V}O_2$ from the third minute of exercise and the sixth minute of exercise, was significantly greater in a group of sprint-trained athletes compared to endurance-trained athletes. The authors observed a significant association between the relative amplitude of $\dot{V}O_{2SC}$ and the PPO, MPO, and fatigue index (FI) attained in the 30-s bicycle ergometer Wingate anaerobic test. This $\dot{V}O_{2SC}$ reflects an increased O_2 cost of force production (Poole et al., 1991; Rossiter, Ward, Howe, et al., 2002), with subsequent implications for exercise tolerance (Poole, Barstow, Gaesser, Willis, & Whipp, 1994). This $\dot{V}O_{2SC}$ has been attributed to an increased recruitment of type II muscle fibres (Barstow et al., 1996; Krustrup, Söderlund, Mohr, & Bangsbo, 2004; Pringle et al., 2003).

The upper-body musculature has been shown to have a higher proportion of type II muscle fibres relative to lower-body musculature (Elder, Bradbury & Roberts 1982), and wrestling athletes have been shown to have a larger Type II fiber area in upper body musculature compared to normal participants (Mandroukas et al., 2010). Given that an elevated proportion of type II muscle fibres is associated with a greater, and more rapid, generation of muscle power (Bar-Or et al., 1980), we expected to find an association between the A'$\dot{V}O_{2SC}$ and key parameters of the upper-body RSA test. However, in the present study no association was found between the A'$\dot{V}O_{2SC}$ and upper body RSA variables.

In the present study, a large effect size was observed for A'$\dot{V}O_{2SC}$/EE $\dot{V}O_2$ between groups, with the JT group displaying higher values. Even though the $\dot{V}O_{2SC}$ has often been linked to a decline in muscular efficiency, and supposedly to a reduction in exercise tolerance, associated with the recruitment of type II muscle fibres over the course of exercise, it has also been associated with perturbations in metabolic status of the already-recruited muscle fibres (Korzeniewski & Zoladz, 2015). It is possible that the meaningfully higher, but not significant, A'$\dot{V}O_{2SC}$/EE $\dot{V}O_2$ observed in the JT group may be associated with a greater ability to tolerate metabolic perturbations within the working skeletal muscle, and that an association between the A'$\dot{V}O_{2SC}$ and RSA performance variables may have been found if the protocol had involved more repetitions and/or shorter work: rest ratio.

5.4 [HHb] Kinetics and upper-body repeated sprint performance

The results of the present study revealed significant differences in [HHb] amplitude of response between JT and UT participants, with the JT group displaying a greater amplitude of response. Even though our results demonstrate that there were no significant differences between groups in the τ' [HHb], implying that both groups demonstrated a similar rate of oxygen extraction to match muscle O_2 demands following the onset of exercise, the greater A [HHb] observed in the JT group (d = 1,14) may be associated with a greater muscle oxygen extraction following the onset of exercise (Bailey et al., 2009; Krustrup, Hellsten, & Bangsbo, 2004), given that participants performed the exercise transitions at the same relative workload. McNarry and associates (2010) found similar results, albeit in a very different population. They observed that trained female pubertal swimmers displayed significantly higher [HHb] amplitude compared to untrained pubertal girls when performing square-wave transitions at the same relative intensity.

Moreover, even though no significant differences were found in the maximal A[HHb] between both groups, a medium-large effect size between groups was observed for each sprint of the upper-body RSA test, indicating that there may exist important practical differences between groups in [HHb] amplitude achieved over the course of each sprint, with JT attaining greater values. It is possible that this greater [HHb] amplitude of response may contribute to the increased RSA observed in the JT group.

The greater O_2 extraction ability, expressed in terms of A[HHb] may be associated with an increased mitochondrial content in skeletal muscle fibers that takes place following training (Glancy, Barstow & Willis, 2008; Zoladz, Korzeniewski & Grassi, 2006). Judo training involves several types of muscle action/contraction regimens and training regimens, and all these seem to have the potential to increase skeletal muscle mitochondrial density (Howald, Hoppeler, Claasen, Mathieu & Straub, 1985; Ingjer, 1979; Saltin & Gollnick, 2011). Increased triceps brachii mitochondrial content as been observed in upper body trained athletes compared to untrained control participants (Berg et al., 2019).

Bailey and associates (2009) observed that supramaximal interval training, consisting of 30-s all out bicycle ergometer sprints followed by 4 min. recovery periods, performed three

times per week over two weeks, resulted in no changes in τ'[HHb] determined for moderate intensity exercise transitions. However, the authors reported that the high-intensity training intervention resulted in significant decreases in τ'[HHb] (12 ± 3-s pre. vs. 9 ± 3-s post.) determined for severe-intensity exercise transitions. Given that Judo-specific training demands high-intensity efforts interceded with short recovery periods, and has been shown to result in significant increases in upper body anaerobic peak power measured in a wingate test (Franchini et al., 2016), it is possible that judo training may lead to faster muscle O_2 extraction in the context of high-intensity exercise.

The observations made in the present study suggest that there is no significant association between [HHb] kinetics variables and upper body RSA performance. No meaningful association could be established between τ' [HHb] and a ↓PPO, ↓MPO and ΣWork over the course of the four sprints, either when we consider the sample of participants as a single heteregoneous group, or when we analyse each group separately, which also refutes our hypothesis for the present study, that [HHb] kinetics, as a proxy of muscle O_2 extraction, would be associated with improved RSA performance variables.

5.5 MAP, Peak $\dot{V}O_2$ and $\dot{V}O_2$ at VT_1 and upper body repeated sprint performance

The results observed in the present study indicate that there is a strong negative correlation between the MAP attained in the incremental upper-body ergometer test and the ↓PPO between the first and fourth sprints for the whole group of participants. Given that MAP is associated with training status (Poole, 1990), these results reveal that participants which are "aerobically" trained to a greater extent display an increased ability to resist a decrease in power output over the course of high-intensity exercise. This may be due to adaptations in skeletal muscle such as increased concentration of aerobic enzymes, increased mitochondrial volume and increased myoglobin concentration, which seem to play an important role in the ability to produce ATP aerobically (Costill, Daniels, Fink, Krahenbuhl & Saltin, 1976; Gollnick et al., 1972; Holloszy & Coyle, 1984).

Interestingly, this association was not observed for the JT group. It may be that the MAP of the individuals of the JT group was too similar (low range or spread of values) for any meaningful association to be established. It may also be that there is a certain fitness

threshold for which this association is valid, and that above this fitness threshold other variables are more important in determining upper-body RSA performance.

However, for UT participants, the strong positive correlation observed between the MAP and the ΣWork throughout the four sprints may be due to the lower training status in this group of individuals.

In the present study, a strong negative correlation was found between the peak $\dot{V}O_2$ and the ↓PPO between the first and last sprint and the ΣWork throughout all sprints. Similar findings have been reported by other authors (Bogdanis et al., 1996; Gaitanos et al., 1993).

The importance of peak $\dot{V}O_2$ or $\dot{V}O_2$ max. to RSA performance seems to be two-fold: 1) Across multiple sprints, aerobic ATP provision progressively increases such that aerobic metabolism may contribute as much as 40% of the total energy supply during the final repetitions of a repeated-sprint protocol (McGawley & Bishop, 2015); 2) Enhanced oxygen delivery to muscles post-exercise potentially accelerates the rate of PCr resynthesis, an oxygen-dependent process (Colliander, Dudley, & Tesch, 1988; Harris et al., 1976), facilitating a faster recovery from high-intensity exercise.

Bishop and associates observed a significant negative association ($r = - 0,50$; $p < 0,05$) between $\dot{V}O_2$ max. and % Work decrement over the course of 5 x 6-s sprints (Bishop & Edge, 2006) in a group of female basketball athletes. The authors proposed that athletes with greater $\dot{V}O_2$ max. would be able to achieve a higher oxygen consumption rate throughout each sprint, reducing the contribution of substrate-level phosphorylation to ATP resynthesis, and therefore allowing more work to be done over the course of the sprint protocol (McGawley & Bishop, 2015). This may also be associated with a higher cardiac output ($\dot{Q}$) and subsequent increase in muscle blood flow, which may aid post-exercise recovery (Bangsbo & Hellsten, 1998). Interestingly, this association was not observed in the JT group in the present study.

$\dot{V}O_2$ max. is thought to be mainly limited by maximal $\dot{Q}$, especially in the context of exercise involving large muscle groups (Helgerud et al., 2007; Spurway & Ekblom, 2011). In non-specifically trained individuals, upper-body $\dot{V}O_2$ max. is generally 70% of that attained in the context of whole-body/lower-body exercise (Seals, & Mullin, 1982), and therefore, cardiac output is not thought to limit $\dot{V}O_2$ max. in such type of exercise. High-

intensity training has been shown to increase upper-body skeletal muscle capillary density (Zinner et al., 2016), and while it may be that in untrained individuals there is a perfusion limitation relative to mitochondrial volume which may limit V̇O2 max. (Terjung, Zarzeczny, & Yang, 2002) given that the upper-body musculature has been shown to extract less oxygen relative to lower-body musculature due to differences in mean blood transit time, diffusing area, and larger diffusing distance (Calbet et al., 2005), such relationship might not hold true for the trained individuals.

Decreases in arterial oxygen saturation have been associated with a decrement in mechanical work of 23,5% over the course of 20 x 5-s bicycle sprints interceded with 25-s of rest (Billaut & Smith, 2010), which was also linked to reductions in muscle integrated electromyogram response. The authors proposed a link between arterial oxygen saturation and motor unit activity, which may also help explain why a significant negative correlation between Peak $\dot{V}O_2$. and decrease in ΣWork over the course of the four sprints was observed in the group of UT participants in the present study.

The present study found a strong negative correlation between VT_1_$\dot{V}O_2$ and the ↓PPO between the first and fourth sprint and a strong positive correlation between the ΣWork throughout all sprints, when the whole sample is analysed as a single heterogeneous group, but not when each group is analysed separately.

The measured oxygen consumption observed during the attainement of peak $\dot{V}O_2$ is akin to the concept of fractional utilization of $\dot{V}O_2$ max., and is associated with a greater capacity to utilize a large percentage of $\dot{V}O_2$ max. over the course of an high-intensity activity (Costill, Thomason & Roberts, 1973). However, given the short duration of each sprint, it is possible that other variables of aerobic fitness may be more important in determining RSA performance.

The VT_1 has also been recognized as an important physiological variable associated with aerobic fitness (Anderson & Rhodes, 1989), which seems to be associated with the lactate threshold (Tanaka et al., 1983), although this association has been disputed (Brooks, 1985; Walsh & Banister, 1988).

Anderson and Rhodes (1989) suggest that even though different mechanisms may mediate the occurrence of both the ventilatory and lactate thresholds, and that they may not be coincident, they seem to be clearly related to thresholds in exercise tolerance. Ivy and

associates observed a significant strong positive correlation between the ability to oxidize pyruvate and the velocity at lactate threshold (r = 0,83; p<0,05) and a strong positive association between the percent of slow twitch fibers and the velocity at lactate threshold (Ivy, Whiters, Van Handel, Elger & Costill, 1980), establishing a strong link between oxidative capacity and the lactate/ventilatory threshold.

Other authors have also reported strong correlations between variables related to muscular oxidative capacity and high-intensity exercise performance. Bogdanis and associates (1996) observed a strong positive relationship (r = 0,84) between the percentage of $\dot{V}O_2$ max corresponding to 4 mmol/L of blood lactate concentration and the resynthesis of PCr following a 30-s maximal bicycle ergometer sprint, establishing a link between aerobic fitness or muscle oxidative capacity and PCr resynthesis, which seems crucial to RSA performance (Gaitanos et al., 1993). Lowery and associates (2018) have recently demonstrated a significant moderate negative relationship between workload at VT_1 attained in a ice-hockey-specific incremental test and the decrease in speed over half of a course during eight maximal ice-skating bouts of 25-s of duration interceded with 90-s of passive recovery (r = - 0,55; p < 0,001).

It is possible that some association may have been found between VT_1_$\dot{V}O_2$ and key RSA performance variables if each group included individuals with more contrasting performance levels. Moreover, given that in the present study a different type of exercise modality was used, involving the upper body musculature, it is also possible that the relationship between [PCr] recovery and $\dot{V}O_2$_VT_1 (or lactate threshold) may not be observable.

Overall, it seems that peak $\dot{V}O_2$ and MAP are associated with improved upper body RSA in a group of individuals with different training status, but not in a group of JT.

5.6 Predictive variables of Σ Work over the course of the upper body RSA test

NIRS-derived [HHb] signal has been considered to reflect the ratio between muscle $\dot{V}O_2$ relative to muscle $\dot{Q}O_2$, and therefore has been considered an index of muscle O_2 extraction (DeLorey et al., 2003; Grassi et al., 2003b). Bishop and associates (2004) observed a

significant positive correlation (r = 0,54) between the $\dot{V}O_2$ at the lactate threshold, an indicator of muscle oxidative capacity, and the total work performed over the course of five 6-s cycle sprints in a group of untrained female participants (Bishop et al., 2004). In the present study, the maximal A[HHb] achieved in the fourth sprint was found to be a predictor of ΣWork when the sample of participants is analysed as a single heterogenous group. However, when we analyse each group separetly, maximal A[HHb] achieved in the fourth sprint was not found to be a predictor of ΣWork.

Despite the methodological differences between the present study and the study undertaken by Bishop and associates (2004), the observed relationship between these indicators of muscle oxidative capacity and total work performed seem to indicate that muscle oxidative capacity may be an important predictor of total work performed over the course of an RSA task.

Aguiar and associates (2016) reported a significant negative association (r = -0,58) between $\dot{V}O_2$ max. and the decrease in performance over the course of 10 x 35 m sprints, interspersed with 20-s of recovery in an heterogenous group composed of endurance runners, sprinters and healthy individuals (Aguiar et al., 2016). These results seem to emphasize the relationship between $\dot{V}O_2$ max. and the ability to maintain a high power output over the course of several RSA efforts. Even though the present study used a very different exercise protocol, using different activity and recovery periods, as well as a different exercise type/modality, peak $\dot{V}O_2$ was found to be a significant predictor of ΣWork over the course of the RSA protocol.

However, given that neither A[HHb] achieved in the fourth sprint, nor peak $\dot{V}O_2$ were found to be predictors of ΣWork in the JT group, it is likely that other parameters may be stronger predictors of RSA performance in a group of trained individuals, in particular, trained judo athletes.

5.7 Limitations

The current study presents several limitations, which may limit the degree to which we can generalize the observations that were made. The athletes that participated in the present study were mostly national-level athletes, although it included some international level

athletes. It is possible that different associations may have been found if the study had included judo athletes with a higher training status, and therefore, further conclusions may have been drawn regarding the physiological and fitness parameters which may be associated with upper body RSA performance in this group of athletes.

The upper-body RSA protocol used in the present study was defined based on the observations reported by Soriano and associates (2019), where the sum of time to kumi kata (8,4-s), kumi kata (6,1-s) and throwing time (1,3-s) corresponded to approximately 15-s, the duration chosen for the working periods. The recovery period of 45-s was chosen so as not to introduce a bias in the aerobic contribution towards exercise performance. However, it is recognized that these chosen times may not correctly match the actual work:rest periods observed in a judo match, and that the number of sprints may have been insufficient to match the number of sequences of attacks observed in an actual judo match. Castarlenas and Planas (1997) observed that in an average judo match, up to 8 standing sequences occurred. Therefore, it is possible that different associations may have been found if a different protocol had been used.

5.8 Future research

Future research should seek to elucidate which factors may contribute to upper-body RSA performance in judo athletes, particularly aspects related to anaerobic capacity, such as maximal accumulated oxygen deficit or 30-s wingate-derived performance variables. It would also be interesting to study such aspects in a heterogeneous population in order to highlight what factors, in a general manner, contribute to upper-body RSA performance, not only because of its importance to several sports and daily activities, but in order to further highlight several aspects regarding cardiorespiratory response, metabolic control and energetics during upper-body exercise.

Future research should include a larger sample of athletes, and athletes of different training status, in order to highlight which physiological parameters might be related to improved upper-body RSA performance in judo athletes of different training background, and therefore, which factors may be associated with the ability to maintain high activity levels over the course of a judo match in athletes of different level.

It is important that future research focuses on using different repeated-sprint protocols using more repetitions and possibly a different work: rest period in order to reveal which factors may be associated with improved RSA ability under different match conditions.

One crucial aspect that should be the focus of future research is the association between the physiological variables studied in the present study, namely $\dot{V}O_2$ and [HHb] kinetics, and performance in the actual match and/or specific fitness tests of judo performance.

Chapter VI – Conclusions

The results of the present study revealed that $\dot{V}O_2$ kinetics and [HHb] kinetics are not associated with increased RSA performance. The results observed in the current study indicate that there is no significant association between $\dot{V}O_2$ and [HHb] kinetics variables and upper body RSA performance, namely between $\tau_{\text{phase II}}$ and τ' [HHb], indicators of the speed of $\dot{V}O_2$ and [HHb] response, and the decrease in PPO, MPO and ΣWork over the course of the four sprints, either when we consider the sample of individuals as a single heteregoneous group, or when we analyse each group separately. However, a strong negative correlation was found between the MAP and peak $\dot{V}O_2$ and the ↓PPO between the first and last sprint and a strong positive correlation was found between the MAP and the ΣWork throughout the four sprints when we consider both groups as single heterogeneous group. Significant strong negative correlations were also observed between the peak $\dot{V}O_2$ and the ↓PPO, the ↓MPO and the ↓Work performed in the group of UT participants.

These results suggest that aerobic fitness variables may play an important role in upper body RSA performance. The importance of upper body aerobic fitness, as expressed by peak $\dot{V}O_2$ and MAP, may be important for upper body RSA performance given that as high intensity efforts are repeated over time, performance seems to become more reliant on aerobic ATP provision and that enhanced oxygen delivery to muscles may contribute to post-exercise recovery, facilitating the maintenance of power output over time.

However, no correlations were found between peak $\dot{V}O_2$ or MAP and upper body RSA performance variables in the group of judo athletes, which may suggest that aerobic fitness may only be a limiting factor if individuals do not have an adequate (aerobic) training status, and that other physiological and fitness variables may play a more important role in upper body RSA performance or upper body high-intensity efforts typical of judo once individuals attain a certain fitness level/training status.

In fact, a large effect size was observed for the $\dot{V}O_{2SC}$ amplitude relative to end-of-exercise $\dot{V}O_2$ between groups, with JT displaying significantly higher value, which may be associated with a greater ability to tolerate metabolic stress within the working skeletal muscle compared to untrained individuals, reinforcing the notion that other

physiological factors may play a more important role compared to aerobic fitness variables in determining upper body RSA once athletes attain a certain training status.

Chapter VII – References

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