

Peripheral Adaptations in High-Performance Handcycling

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## ABSTRACT

### Peripheral Adaptations in High-Performance Handcycling

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This project examined associations between physiological parameters and performance during different efforts in handcycling. Athletes performed a 30-second Wingate, a maximal incremental test and a 20-minute time-trial (TT) on an arm ergometer, during which muscle oxygenation of the biceps brachii (BB), triceps brachii (TB), anterior deltoid (AD), and extensor carpi radialis brevis (ER) was measured using near-infrared spectroscopy (NIRS, Moxy monitors). Oxygen consumption ( $VO_2$ ), cardiac output and power were also evaluated, and grip strength was assessed bilaterally. Pearson and Spearman correlations revealed that  $VO_{2max}$  ( $R = 0.90$  [0.08, 1.91],  $p = 0.040$ ) and BB  $O_2$  extraction ( $R = 0.86$  [0.16, 1.57],  $p = 0.027$ ) were associated to maximal aerobic power (MAP). AD  $O_2$  extraction ( $R = 1.0$ ,  $p < 0.001$ ), reoxygenation of TB ( $R = 0.82$  [-0.15, 0.99],  $p = 0.046$ ), AD ( $R = 0.92$  [0.17, 1.57],  $p = 0.029$ ) and ER ( $R = 0.83$  [-0.13, 0.99],  $p = 0.042$ ), deoxygenation of AD ( $R = -0.90$  [-1.00, 0.17],  $p = 0.037$ ) and ER ( $R = -0.87$  [-1.56, -0.18],  $p = 0.025$ ) were associated to Wingate mean power. Maximal deoxygenation for the muscles average ( $R = -0.88$  [-1.76, 0.01],  $p = 0.049$ ), deoxygenation rate of AD ( $R = -1.00$  [-0.83, 0.63],  $p < 0.001$ ), BB ( $R = -0.95$  [-1.36, 0.03],  $p = 0.036$ ), and ER (-0.90 [-1.00, 0.17],  $p = 0.037$ ), were associated to TT mean power. Reoxygenation rate of AD ( $R = 0.97$  [0.18, 1.51],  $p = 0.032$ ) and BB (-0.99 [-0.98, 0.30],  $p = 0.015$ ) were associated to mean power and TT distance, respectively. Analysis of variance revealed that maximal ER  $O_2$  extraction in the incremental test was 37% higher than in the Wingate, and ER deoxygenation in the Wingate was 88% faster than in the TT. For the physiological variables examined during the TT, most of the time was spent in the highest intensity zone (above 2<sup>nd</sup> lactate threshold) as compared to the lower intensity zones. Results demonstrate the contribution of peripheral adaptations to predict handcycling performance and suggest that upper body muscles oxygenation capacity should be considered in their training regime.

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## **CONTRIBUTION OF AUTHORS**

The article of this thesis (Chapter 2) is entitled “Peripheral Adaptations in High-Performance Handcycling”. I acted as the main author of the article, Myriam Paquette acted as a co-author and Andreas Bergdahl as co-author and last author. The protocol was mainly developed by Myriam Paquette. I was in charge of some of the recruitment of participants, of the tests administration, of the data analysis and interpretation, and of the redaction of the manuscript. Myriam Paquette helped significantly in every step of the process. Andreas Bergdahl helped with the data analysis and redaction.

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## GENERAL INTRODUCTION

Very few studies have evaluated the physiological determinants of handcycling, and most of these studies were performed on small cohorts of able-bodied participants and measured a limited number of physiological parameters. In addition, no study has investigated the association between muscle oxygen extraction capacity and performance in para-cycling. This project is the first to characterize changes in muscle oxygenation and in cardiac output in trained handcyclists and to evaluate their association with performance.

A deeper understanding of the physiological determinants in handcycling would optimize monitoring of athlete's progress and training effects. Portable near-infrared spectroscopy (NIRS) monitors can measure changes in muscle oxygenation and provide information on athlete's peripheral adaptations. Results can also be generalized to other Paralympic sports with efforts generated by upper limbs, such as wheelchair sports, para-canoe, or para-swimming. Therefore, the aim of this thesis was to provide a better understanding of the peripheral cardiovascular adaptations, more precisely the muscle  $O_2$  extraction capacity and its association with performance in trained female and male handcyclists.

The objectives of the study are to provide a physiological profile of well-trained recumbent handcyclists (6 athletes in the H2, H3 or H4 classes, including males and females) and assess the physiological determinants of aerobic, anaerobic, and time-trial (TT) performance. Thus, athletes underwent a 30-second maximal power test (Wingate), a maximal incremental test and a 20-minute TT, on an arm ergometer (Lode Angio). Grip strength was also evaluated with a handheld dynamometer. The physiological variables collected were the changes in muscle oxygenation ( $SmO_2$ , % from baseline) and deoxyhemoglobin concentration ([HHb]) in the biceps brachii (BB), triceps brachii (TB), anterior fibers of the deltoid (AD), and extensor carpi radialis brevis (ER) muscles on the right side of the body with portable NIRS monitors (Moxy). Peak  $VO_2$ , cardiac output, stroke volume, heart rate, (a-v) $O_2$ -difference and blood lactate were also assessed during the tests.

The project examined the interest of using NIRS monitors in the evaluation of handcyclists, which can orientate the choice of training and testing methods and can contribute to the development of norms for the different tests and physiological measures used in handcycling. NIRS could also help in talent identification and selection of athletes. Results can provide insights regarding training protocols and interventions better suited to improve handcycling performance (e.g., heat training, altitude training, ischemic preconditioning, etc.).

## CHAPTER 1: LITERATURE REVIEW

### 1.1 Para-cycling

Para-cycling is the 3<sup>rd</sup> most popular Paralympic sport, a discipline of paratriathlon and an exercise modality for physically or visually impaired individuals<sup>1</sup>. The interest in this athletic discipline increased in the past two decades with a considerable progress in the elite competitiveness around the globe<sup>2,3</sup>. Handcycling, a discipline of para-cycling, was researched extensively in rehabilitation and recreation settings but there is now an emergence of research detailing the physiology of the sport at the elite level, with a few recent studies conducted during competition<sup>3,5</sup>. Rehabilitation and competitive handcycling differ considerably in terms of equipment involved, and in the level of training and performance abilities of the cyclists<sup>3,4</sup>. Thus, previous findings on recreational handcycling are not providing a proper physiological profile of high-performance para-athletes. By definition, elite athletes perform at a national or international level and possess chances of winning World Cup or Paralympic medals. The Canadian government defines high-performance athletes as: “achieving world-class results at the highest level of international competition through fair and ethical means”<sup>6</sup>.

#### 1.1.1 History of para-cycling

Competitive cycling was included for the first time in the Paralympics in the Seoul games in 1988 for athletes practicing tandem only<sup>7</sup>. The sport is now divided in four distinct disciplines with cycling, tricycling and handcycling for physically impaired athletes and tandem for visually impaired athletes. Disciplines are split in several ‘sport classes’ based on the degree of physical impairment with a higher-class number indicating fewer physical restrictions<sup>1</sup>. Para-athletes are generally individuals affected by various injuries, trauma or diseases of the spinal cord or the brain. However, the international Paralympic committee accepts only seven specific physical impairments in para-cycling, and these are, blunted muscle power, athetosis, impaired passive range of motion, hypertonia, limb deficiency, ataxia, or leg length difference (minimally 7 cm)<sup>7</sup>.

The organization of ‘sport classes’ was adopted to minimize the impact of impairment on the outcome of competition, but also for the athlete to rely on physical fitness, training, and athletic talent for performance<sup>8</sup>. Evaluators take into consideration training commitment, biological predisposition and natural talent when classifying an athlete<sup>9</sup>. The present evidence-based classification system was adopted in 2014<sup>9</sup>. In this system, there are five classes in handcycling (H1-5) comprising athletes with restrictions in lower limbs, two in tricycling (T1 and T2) for athletes with coordination and balance impairments, and five in cycling (C1-5) for athletes able to use a regular bike<sup>7</sup>. Tandem cycling includes only one sport class for visually impaired cyclists who race behind a sighted pilot<sup>7</sup>. Cycling and tandem cyclists compete in both track and road events as compared to handcycling and tricycling athletes who race exclusively on the road<sup>7</sup>.

#### 1.1.2 Development of handcycling

Handcycling started developing at the beginning of the 20<sup>th</sup> century but at the time, the focus of the handcycle was on ambulation rather than performance<sup>3</sup>. Research in handcycling gained popularity in the 1960s as it was presented as a useful mode of transportation, 10-15% more mechanically efficient than wheelchairs, with less physical load on the shoulders while allowing to produce higher peak power<sup>3,4,9,10</sup>. The growing interest in handcycling can be attributed to the fact that both able-bodied individuals and people living with a physical impairment can use it as an exercise modality<sup>4</sup>. Currently, handcycles are split in two categories, the attachable unit, suited for daily living and

transportation, and the rigid frame cycle for competition<sup>3</sup>. The competition handcycles are the recumbent and the kneeling configurations (Figure 1.) and these have a special frame with a lower center of gravity, which improves driving properties and allows higher speeds<sup>4</sup>.

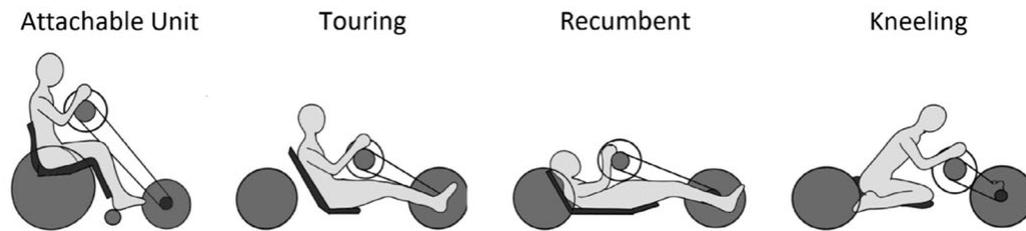


Figure 1. The Different Handcycles. Stephenson et al., 2020

Performance handcycling developed rapidly in the past few decades with its inclusion in the cycling World Championships in 1998 and its recognition as a sport in 1999 by the International Paralympic Committee<sup>2,11</sup>. The discipline only appeared at the 2004 Paralympic games in Athens with the representation of 19 male athletes competing in three classes and across four events<sup>3,11</sup>. Twelve years later in Rio, 65 handcyclists including both men and women contested in 13 events and the sport debuted in paratriathlon, with the inclusion of three classes for both genders, showing a major progress in competitiveness around the world<sup>3</sup>.

At the Paralympics, genders compete separately in the five handcycling classes (H1, H2, H3, H4 or H5) depending on the severity of their impairment<sup>2</sup>. In lower sports classes (H1-2), athletes have upper and lower body functionality restrictions. In contrast, H5 competitors are the least physically restricted athletes with lower level of paraplegia or limb deficiencies. The differences in impairment severity among participants affect largely the cardiorespiratory response to exercise, as well as muscular strength, coordination, and range of motion of the upper limbs and trunk, confirming the importance of athlete classification<sup>3</sup>. The greatest difference between H1 and H2 athletes depends on arm strength, between H2 and H3 athlete on hand strength and between H3 and H4 athlete on trunk strength<sup>9</sup>. Between H4 and H5 athletes, the major difference is based on hip-leg and trunk function, and on the ability to sit on the knees and to maintain this position for extended periods<sup>9</sup>.

H5 athletes only compete in a kneeling position, using both upper body musculature and trunk, thus producing more power to propel the bike than the H1-4 cyclists<sup>2</sup>. H1-4 athletes compete in a recumbent arm power position (Figure 2.) since they do not have sufficient core function to propel the bike on their knees<sup>2</sup>. Studies identified trunk function as the central aspect contributing to sports performance in wheelchair sports classification, since an increased trunk power increases the ability of sprinting<sup>3</sup>. However, due to elevated athlete's frontal surface area, the upright nature of kneeling handcycling is not as aerodynamic and leads to slower race velocities than recumbent handcycling, even with the potential of greater power generation with the use of both arm and trunk muscles<sup>3</sup>.



Figure 2. Team Canada H3 Athlete. Retrieved from Paralympic.ca, 2022

### 1.1.3 Competitive handcycling

At the Paralympic games, handcyclists take part in two outdoor events including the individual TT with a time interval between competitors and the mass start road race<sup>9</sup>. Handcycling individual TTs vary from 10-30 km in length for the women's categories and 12-35 km for the men's categories, lasting 20-40 minutes, and road races range from 40 to 70 km in the women's categories and 45-80 km in the men's categories over a duration of 60-150 minutes with events often scheduled on consecutive days<sup>5,9</sup>. Velocities during handcycling TTs average 24-45 km h<sup>-1</sup> with peaks of 55 km h<sup>-1</sup><sup>3</sup>. At the Rio Paralympics, the highest handcycling average velocity of 43.4 km h<sup>-1</sup> was recorded on a male H4 in the 20 km TT (see Table 1 for more results)<sup>3</sup>. Race terrains and tactics are similar to able bodied cycling, with drafting behind opponents only permitted in the road races to reduce the overall energy costs by 25 to 40 %<sup>2</sup>. Some other popular pacing strategies involve short bursts of accelerations to pass an opponent and the use of negative splits, meaning the second half of the race is performed faster than the first<sup>2</sup>. Climbs cannot represent more than 25% of the total length of the course and the maximal inclination permitted is 15%<sup>9</sup>.

Average margins of 0.5% were measured between the final TT times of the first and second finishers at the Rio Paralympics, representing differences ranging between 3 and 76 seconds between the two positions, and in road races, there was a difference of split seconds between medalists, due to sprint finishes (see Table 1.)<sup>3</sup>. Two Paralympics earlier in Beijing, shorter 12.7 km TTs were won at 25-37 km h<sup>-1</sup> with gaps of 3.1% between finisher's total course times<sup>3</sup>. At the most recent Paralympics in Tokyo, TTs were won by split seconds in the men H2 and men H3 sports classes, as well as road races in the men H3 and H5 (Table 2.)<sup>77</sup>. For the women's H1-H4 road race, there was a margin of 6 seconds between gold and silver in Tokyo 2020 (Table 2.)<sup>77</sup>. These race results demonstrate a rapid progress in elite handcycling competitiveness, which coincides with the emergent research avenues and validates the importance of conducting more studies to optimize the sport's development.

Classification	Distance (km)	Gold		Silver	Bronze	Fourth
		Velocity (km h <sup>-1</sup> )	Time (h:min:s)	Time behind (min:s)		
Time trial						
MH2	20	37.4	00:32:07	0:06	1:33	3:57
MH3	20	42.4	00:28:19	1:06	1:07	1:08
MH4	20	43.4	00:27:39	0:10	1:03	1:16
MH5	20	41.9	00:28:37	0:03	0:15	0:26
WH1-H3	20	35.5	00:33:45	0:13	0:36	1:00
WH4-H5	20	38.0	00:31:36	0:40	1:27	1:38
Road race						
MH2	45	35.8	01:15:23	>0:01	7:49	7:51
MH3	60	38.6	01:33:17	>0:01	>0:01	>0:01
MH4	60	40.5	01:28:48	0:03	0:06	>0:07
MH5	60	36.8	01:37:49	>0:01	>0:01	0:02
WH1-H4	45	35.8	01:15:56	0:02	>0:03	0:05
WH5	45	27.8	01:37:07	0:02	>0:03	>0:03

Note: M: men; W: women; MH1 classification not included in the 2016 Paralympic program.

Table 1. Handcycling TT and Road Race Results at the 2016 Rio Paralympics. Stephenson et al., 2020  
 Depicted ">" signs should be "<"

Classification	Distance (km)	Gold		Silver	Bronze	Fourth
		Velocity (km h <sup>-1</sup> )	Time (h:min:s)	Time behind (min:s)		
Time trial						
MH1	22	30.1	00:43:49	1:55	3:12	9:07
MH2	22	42.1	00:31:23	< 0:01	1:18	4:54
MH3	22	30.2	00:43:39	< 0:02	< 0:10	0:10
MH4	22	35.2	00:37:28	1:02	2:20	2:41
MH5	22	34.6	00:38:12	1:03	1:24	3:09
WH1-H3	22	40.3	00:32:46	0:44	1:04	2:50
WH4-H5	22	28.9	00:45:40	1:46	3:05	3:35
Road race						
MH1-H2	45	24.6	01:49:36	4:07	5:00	33:32
MH3	60	23.3	02:34:35	< 0:01	< 0:01	0:29
MH4	60	26.6	02:15:13	5:43	7:25	8:41
MH5	60	24.9	02:24:30	< 0:01	0:10	0:27
WH1-H4	22.5	24	00:56:15	0:06	0:09	1:32
WH5	45	18.8	02:23:39	3:11	4:32	23:14

Table 2. Handcycling TT and Road Race Results at the 2020 Tokyo Paralympics. Retrieved from paralympic.org 2022

Without any surprise, most studies found a considerable involvement of the aerobic energy system in handcycling events<sup>11</sup>. However, the anaerobic energy system is necessary to generate power output in climbs, surges, and sprints in races of various lengths<sup>2,15</sup>. Previous literature identified maximal aerobic power (MAP) as the maximal amount of work produced by the aerobic system over a given short bout (~ 5 minutes), generally during the last stage of incremental tests to exhaustion and these values vary from 89-150 W, 240-250 W or 220 W in trained H1-H2, H3-H4 and H5 handcyclists, respectively<sup>3</sup>. These values of MAP found by several studies vary in the protocols used, especially in the stage duration and workload increments, making the comparisons between studies difficult<sup>3</sup>. Twenty-one studies reported a peak oxygen consumption ( $\text{VO}_2\text{peak}$ ) of  $> 3.0 \text{ L min}^{-1}$  or  $> 40.0 \text{ ml kg}^{-1} \text{ min}^{-1}$  in H3-5 athletes<sup>3</sup>. These values of  $\text{VO}_2\text{peak}$  are attributed to 5-6 years of handcycling experience with  $6 \pm 2$  training sessions per week and representing  $223 \pm 57 \text{ km/week}$ <sup>3</sup>. For the H1-2 categories,  $\text{VO}_2\text{peak}$  of  $1.1 \pm 0.4$  to  $2.0 \pm 0.4 \text{ L min}^{-1}$  were found with higher values representing greater training experience<sup>3</sup>. Lower  $\text{VO}_2\text{peaks}$  observed in the H1-2 groups as compared to the H3-5 athletes represent the activation of less muscle mass due to greater impairment<sup>3</sup>. For the H5 athletes, trunk muscles propelling the bike in kneeling handcycling increase oxygen demand on the body as compared to recumbent arm propulsion<sup>3</sup>. In addition to  $\text{VO}_2\text{peak}$ , studies in handcycling using trained participants have reported peak heart rate, peak blood lactate content and peak respiratory exchange ratios, as being  $> 180 \text{ beats min}^{-1}$ ,  $10 \text{ mmol L}^{-1}$ , and 1.20 respectively<sup>3</sup>. In Stephenson's et al. review article (2020) detailing the physiology of handcycling, only 51/450 athletes included in these studies were female, demonstrating a lack of information regarding female handcyclists<sup>3</sup>.

## 1.2 Cardiovascular Physiology

### 1.2.1 $\text{VO}_2\text{max}$ and its limitations

To understand the physiological determinants involved in competitive handcycling, different physiological terms should be defined. Maximal oxygen consumption ( $\text{VO}_2\text{max}$ ) is the most widely used variable to assess cardiorespiratory fitness and is the highest rate at which oxygen is taken up by the lungs and utilized by the body during exercise<sup>16,17,39</sup>. At rest,  $\text{VO}_2$  is around  $3\text{-}5 \text{ ml kg}^{-1} \text{ min}^{-1}$  and only a small portion of  $\text{O}_2$  is consumed within the skeletal muscles<sup>71</sup>. In contrast, during maximal exercise, the pulmonary  $\text{VO}_2$  can reach values as high as  $90 \text{ ml kg}^{-1} \text{ min}^{-1}$  in the highly trained<sup>71</sup>. Determined by the Fick principle,  $\text{VO}_2$  is the product of cardiac output (Q) and arterio-venous oxygen difference ( $a\text{-vO}_2 \text{ diff}$ ) (Figure 3). Endurance training results in high  $\text{VO}_2\text{max}$  due to an enhanced pumping capacity of the heart with superior Q, stroke volume (SV), and capacity of the muscles to extract  $\text{O}_2$  ( $a\text{-vO}_2 \text{ diff}$ )<sup>30,39</sup>. Trained hearts relax quickly and fill with a large end-diastolic volume (EDV), representing the amount of blood in the ventricles before systole, to accommodate high blood flow and  $\text{O}_2$  transport during exercise<sup>30</sup>. Recent evidence has shown that some amount of  $\text{VO}_2\text{max}$  is heritable, but length, duration, type, intensity, and age of initiation of training, as well as gender, body weight and health are all contributing factors as well<sup>30, 71</sup>.

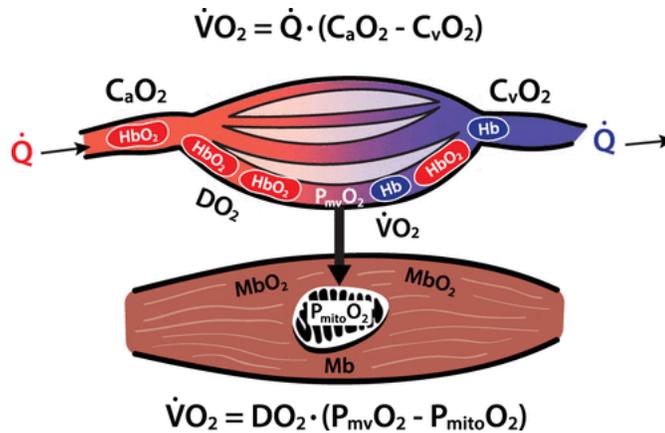


Figure 3. O<sub>2</sub> Delivery and O<sub>2</sub> Diffusion from Capillary to Muscle Fiber Mitochondria (Fick's Law): CaO<sub>2</sub>, arterial concentration of O<sub>2</sub>; CvO<sub>2</sub>, venous content of O<sub>2</sub> draining the muscle/limb; DO<sub>2</sub>, diffusivity of O<sub>2</sub>; Mb, myoglobin; PmitoO<sub>2</sub>, mitochondrial partial pressure of O<sub>2</sub>; PmvO<sub>2</sub>, microvascular partial pressure of O<sub>2</sub>; Q̇, blood flow; V̇O<sub>2</sub>, oxygen uptake. Barstow, 2018

The Scientific community suggests that VO<sub>2max</sub> is limited by different mechanisms related to the rate at which O<sub>2</sub> can be supplied by the heart, lungs, and blood (central mechanisms) and extracted by the skeletal muscles (peripheral mechanisms) during strenuous exercise (Figure 4.). These mechanisms will be presented in this section.

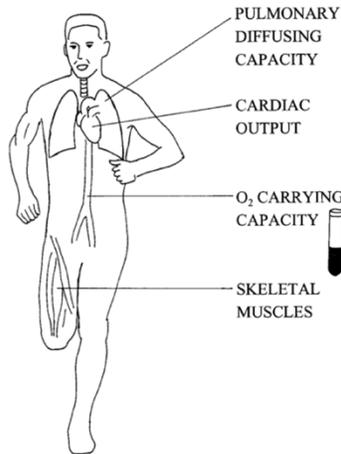


Figure 4. Limitations of VO<sub>2max</sub>. Basset et al., 2000

### 1.2.2 Central adaptations of VO<sub>2max</sub>

#### 1. Pulmonary diffusion capacity

It is generally accepted that O<sub>2</sub> transport during exercise at sea level is adequate to meet the metabolic demands of the body<sup>46</sup>. However, under certain circumstances, such as in altitude training or in extremely high Q<sub>max</sub>, it appears that the pulmonary system may limit VO<sub>2max</sub>, where arterial O<sub>2</sub> desaturation impairs diffusion capacity of O<sub>2</sub> into the blood during periods of maximal exercise<sup>16,46,71</sup>. In addition, this phenomenon can be seen at very high exercise intensities, where

the increase in cardiac output in trained participants leads to a decrease in transit time of red blood cells in the pulmonary capillary (alveolar/capillary gas equilibrium impaired), which leads to exercise induced arterial hypoxemia, as the red blood cells do not have enough time to re-saturate completely.<sup>16,71</sup>

## 2. Cardiac output

Q is the volume of blood the heart pumps per minute and depends on heart rate, contractility, pumping capacity, preload, and afterload and is the product of heart rate (HR) and SV<sup>18</sup>. Q is a major determinant of VO<sub>2</sub>max, accounting for 70-85% of its limitation in systemic O<sub>2</sub> delivery<sup>16,19,71</sup>. Trained individuals have a higher maximal Q than the untrained (40 vs. 25 L·min<sup>-1</sup>) with superior heart contractility<sup>16,39</sup>. Differences in Q accounting for disparities in VO<sub>2</sub>max in sedentary men and women are usually due to individual variances in SV, the most influential factor affecting Q, since maximal HR is not considerably variable between individuals ( $Q = SV \times HR$ )<sup>16,19</sup>.

## 3. Stroke volume

SV is the amount of blood being pumped by the ventricles at each heart contraction. The high SV reported in endurance athletes is attributed to a large EDV and in turn, this is due to greater cardiac chambers compliance, distensible pericardium, and increase in plasma volume<sup>30</sup>. A strong relationship exists between heart size and SV as well as heart size and VO<sub>2</sub>max<sup>17</sup>. Preload is the increase in distension of the ventricle and its myocardial cells that will increase the force of contraction of the heart, thus increasing SV<sup>18</sup>. In turn, afterload refers to the resistance against which ventricles must eject blood and this parameter depends on the arterial blood pressure and on the vascular tone, so that increasing afterload will reduce SV<sup>18</sup>. Elite endurance athletes, and especially those working upright against gravity (e.g., runners) must also have a rapid diastolic relaxation with vigorous suction<sup>30</sup>. Suction occurs from the left atrium across the mitral valve into the apex of the left ventricle to maximize venous return<sup>30</sup>.

## 4. Heart rate

HR is the simplest physiological determinant since faster heart rate generally represents more blood being pumped over a certain period, however in highly elevated heart rates, ventricular filling can be insufficient, and SV can be diminished<sup>18</sup>. HR is not a dominant physiological determinant of VO<sub>2</sub>max, considering there are very little, or no training effects associated to maximal HR<sup>16</sup>. Endurance trained subjects have lower resting HR and lower HR at a fixed submaximal work rate, correlated to an increased SV and an enlarged heart with higher pumping abilities<sup>16,17</sup>.

## 5. Oxygen carrying capacity of the blood

Oxygen carrying capacity can be a strong limitation of VO<sub>2</sub>max in cases of anemia or when large quantities of blood are lost, where O<sub>2</sub> transport to the working muscles is altered by changes in hemoglobin content<sup>16,47</sup>. Hemoglobin is a protein found in red blood cells containing iron, a mineral with O<sub>2</sub> transport properties. With a reduction in blood volume, accompanied by a reduction in red blood cells count, we see a reduction in hemoglobin mass (amount of total circulating hemoglobin), which in turn reduces VO<sub>2</sub>max considerably, due to the decrease in oxygen transport capacity<sup>47</sup>. On the other hand, an increase in total hemoglobin mass improves VO<sub>2</sub>max and endurance performance<sup>34</sup>.

### 1.2.3 Peripheral adaptations of VO<sub>2</sub>max: skeletal muscle characteristics

The (a-v)O<sub>2</sub> diff represents the difference in oxygen content between the arterial blood and the venous blood. When blood circulates in the body, (a-v)O<sub>2</sub> diff indicates the quantity of oxygen used

by the capillaries. At rest and during exercise, available oxygen is extracted from the blood perfusing the active muscles<sup>16</sup>. During maximal exercise, the arterial O<sub>2</sub> content is approximately 200 mL O<sub>2</sub>·L<sup>-1</sup> of blood and the venous O<sub>2</sub> content is as low as 20-30 mL O<sub>2</sub>·L<sup>-1</sup> of blood since the working muscles consume oxygen to function<sup>16</sup>. The (a-v)O<sub>2</sub> diff is determined by the muscles' ability to extract O<sub>2</sub> and by the arterial O<sub>2</sub> content, set by the hemoglobin content and by the O<sub>2</sub> saturation of hemoglobin (HbO<sub>2</sub>)<sup>71</sup>. Hemoglobin content adapts considerably to training<sup>71</sup>. Peak (a-v)O<sub>2</sub> diff is only slightly higher in athletes as compared to healthy non-athlete individuals, confirming that the major factor contributing to a large VO<sub>2</sub>max in the athletic population is a large Q, through an increase in SV<sup>30</sup>. With exercise modalities activating large muscle masses (e.g., whole-body exercises or leg exercises), muscle O<sub>2</sub> extraction capacity is described "in excess" as compared to the muscle O<sub>2</sub> extraction at VO<sub>2</sub>max (the muscle could extract a lot more O<sub>2</sub>, but in this case extraction is limited since the muscle delivery is restricted, as blood needs to be supplied to the whole-body), suggesting it is not an important VO<sub>2</sub>max determinant in these types of exercises<sup>16</sup>. However, these hemodynamic changes could be different in handcycling, where smaller upper body muscles are involved in the propulsion.

The mitochondria are the organelles in the muscle cells where O<sub>2</sub> is utilized during the final stages of the electron transport chain and are responsible for adenosine triphosphate (energy) production and distribution<sup>49</sup>. A linear relationship exists between VO<sub>2</sub>max and muscle mitochondrial content<sup>48</sup>. VO<sub>2</sub>max is determined by skeletal muscles mitochondrial O<sub>2</sub> uptake through a peripheral O<sub>2</sub> gradient (O<sub>2</sub> diffusion capacity between the muscle capillary's erythrocytes and the mitochondria), mitochondrial enzymes and oxidative activity<sup>16,17,20</sup>.

#### Peripheral adaptations changes with exercise

The increase in mitochondrial volume in response to endurance training elicits metabolic effects such as muscles oxidizing fat at a higher rate (sparing muscle glycogen and blood glucose) and a decrease in lactate production during exercise, partly due to morphological changes in electron transport chains<sup>16,49,50</sup>. In addition, elevated muscle mitochondrial density may promote a greater extraction of O<sub>2</sub> from the blood by the muscles, thus promoting a slight increase in VO<sub>2</sub>max<sup>16</sup>. Bassett and colleagues (2000) suggested that an increase in mitochondrial content will not improve VO<sub>2</sub>max but will rather increase endurance performance, especially in athletes with similar VO<sub>2</sub>max, but in whom large disparities in mitochondrial enzymes content and oxidative capacity can be seen<sup>16</sup>. Moreover, aerobic capacity and the activity of skeletal muscle oxidative enzymes varies with the level of fitness<sup>52</sup>. Gollnick et al. observed a doubling of vastus lateralis succinate dehydrogenase activity (SDH) after 5 months of endurance training, confirming that oxidative citric acid cycle and respiratory chain enzymes significantly augment with training<sup>53,31</sup>. Other studies have shown that submaximal training improves the oxidative capacity of the slow twitch (oxidative) fibers, shifting towards a greater utilization of fat, while high intensity interval training improves oxidation and SDH level of the fast twitch (glycolytic) fibers<sup>31,52</sup>. An augmentation in oxidative enzymes in the exercising muscles are found in both slow and fast twitch fibers, thus enhancing oxidation of free fatty acids in all fiber types<sup>31,51</sup>. In addition to SDH, citrate enzyme levels rise in the exercising muscles, inhibiting phosphofructokinase, which slows down glycolysis and lactate formation, thus enhancing aerobic performance<sup>31,51</sup>.

In highly trained endurance subjects, there is superior quantity of capillaries per muscle fiber, and this physiological change is closely associated to VO<sub>2</sub>max, and adapts considerably to training<sup>31</sup>. Over time, aerobic exercise generates high blood flow in isolated muscles of the body through the

development of capillary density (capillaries per mm<sup>2</sup>) to prolong the mean transit time (MTT), which largely promotes gas exchanges by reducing the diffusion distance from blood to muscle<sup>16,17,50</sup>. This physiological change increases oxygen delivery to the active muscles and maintains oxygen extraction and (a-v)O<sub>2</sub> diff at high work rates<sup>16,50</sup>. In contrast, when detraining occurs, muscle capillary diameter and density may decrease, shortening the MTT, thus limiting gas and substrates exchanges and increasing glycolytic energy demand and lactate production<sup>50</sup>. Large proportion of type I fibers in active muscles is generally associated with a reduction in diffusion distance (large MTT), facilitating free fatty acids, blood glucose and O<sub>2</sub> utilization.<sup>31</sup> Since arm muscles have a greater proportion of type II fibers, a smaller capillary-to-fiber ratio, and elevated intramuscular pressure that can exceed perfusion pressure, blood and oxygen delivery and substrate exchanges may be limited, re-emphasizing the importance of peripheral development<sup>61</sup>.

A study examined the impacts of one-legged cycling by making participants exercise only one leg while testing VO<sub>2</sub>max locally in each leg<sup>16</sup>. Authors found a 23% increase in VO<sub>2</sub>max in the exercising leg as compared to a 7% increase in VO<sub>2</sub>max in the control leg over time, confirming a large contribution of peripheral skeletal muscle adaptations<sup>16</sup>. In 1985, Saltin et al. examined what happens when only small muscle masses (unilateral knee extension) are used during an effort and found that VO<sub>2</sub>max was limited by a greater proportion of blood flow (Q) vehiculated to the isolated exercising thigh as compared to a smaller Q vehiculated to the same isolated exercising thigh when two-legged exercise was performed (bilateral knee extension)<sup>16,17</sup>.

Researchers reported 2-3 times higher O<sub>2</sub> uptake in the quadriceps during unilateral vs. bilateral knee extension<sup>16,17</sup>. The results of these studies suggest that whole-body training at sea level (e.g., running, cycling, or swimming) is primarily limited by Q, by the convective O<sub>2</sub> delivery of capillary blood flow, which depends on muscle blood flow, arterial O<sub>2</sub> saturation and hemoglobin content<sup>17</sup>. The blood flow perfusing the muscles is less than during 1-limb exercise, and lower O<sub>2</sub> extraction is seen and could be explained by a shorten MTT in muscle capillaries and an increase in resistance<sup>17</sup>. Then, Q is not the main factor limiting VO<sub>2</sub>max in efforts recruiting small, isolated muscle groups and performance and VO<sub>2</sub>max limitations in efforts recruiting small muscle masses are primarily peripheral<sup>16,60,71</sup>. Thus, peripheral cardiovascular adaptations to training may be more important in handcycling than the central VO<sub>2</sub> adaptations, and we should find a way to measure this assumption.

#### **1.2.4 Lactate threshold and endurance**

During submaximal exercise, O<sub>2</sub> delivery to the muscles is closely related to the mitochondrial O<sub>2</sub> demand<sup>16</sup>. If the intensity surpasses LT, glycolysis is speeding up due to an increase in cellular charge to bring VO<sub>2</sub> to higher levels, depleting the remaining carbohydrates so that lactate is accumulating too quickly for its removal to be effective<sup>16</sup>. Studies have shown that trained individuals deplete their muscle glycogen stores less rapidly than the untrained, increasing their resistance to fatigue and endurance performance<sup>51</sup>.

LT is determined during an incremental test where blood lactate samples are generally taken at every stage (every few minutes). LT is designated at the intensity at which the blood lactate content rises sharply above baseline, illustrating the shifting point from aerobic to anaerobic energy turnover and predicting endurance performance<sup>16,54</sup>. Lactate elevation is associated with the recruitment of fast-twitch muscle fibers, elevation of plasma epinephrine, reduction in liver blood flow, and varies according to mitochondrial content of the muscles, measured by mitochondrial enzyme activity and training level<sup>16,54</sup>.

## 1.3 Physiological determinants of handcycling performance

### 1.3.1 Role of the aerobic energy system in handcycling

Most studies in handcycling confirmed the important role of the aerobic energy system for performance. In fact, studies identified the aerobic metabolism as the primary energy system in the handcycling marathon (42.195 km) up to the ultralong handcycling event (540 km)<sup>11</sup>, while shorter Paralympic TT distances (10-35 km) were associated both to the aerobic and anaerobic energy systems<sup>3</sup>.

The main determinants of performance in elite cycling were extensively researched and well described as  $\text{VO}_2\text{max}$ , endurance, economy of motion (amount of energy spent per unit of velocity) and anaerobic capacity<sup>19</sup>. In para-cycling, most studies were conducted with sedentary, minimally trained, or able-bodied participants in rehabilitation or recreation settings. Only a few studies explored the role of physiological determinants of trained handcyclists.  $\text{VO}_2\text{max}$ , ventilatory and lactate thresholds and MAP were the physiological determinants found to be better associated to handcycling performance in these studies<sup>5,11</sup>. De Groot et al. (2014) and Fisher et al. (2015) established that a handcycling mountain climbing TT and a road 22-km TT, were strongly dependent on  $\text{VO}_2\text{max}$  and on MAP during an incremental test, respectively<sup>8,20</sup>.

Even though several studies have shown high correlations between  $\text{VO}_2\text{max}$  and handcycling performances ( $r \cong 0.9$ ), these were conducted with sedentary participants or lower-level athletes ( $\text{VO}_2\text{max}$  of 1.1 to 2.3 L min<sup>-1</sup>)<sup>3,11,42</sup>. In contrast, recent work by Stone et al. (2020) conducted with highly trained handcyclists showed no significant correlation between a 16 km TT performance and  $\text{VO}_2\text{max}$ <sup>3</sup>. This paradigm is well understood in highly trained able-bodied athletes possessing high  $\text{VO}_2\text{max}$ , in whom endurance is measured as the fractional utilisation of  $\text{VO}_2\text{max}$  (% of  $\text{VO}_2\text{max}$  that can be sustained for the duration of the race) and is a stronger predictor of performance as compared to  $\text{VO}_2\text{max}$ <sup>3,21</sup>. Endurance is closely linked to the  $\text{VO}_2$  at the lactate threshold (LT) and is a trainable parameter over time<sup>16</sup>.

Fisher et al. (2015) established that ventilatory and lactate thresholds and economy of motion were associated to performance on a 22-km handcycling TT<sup>3</sup>. In another project by Stephenson et al. (2020) conducted with paratriathletes (ambulatory) and handcyclists (non-ambulatory), a 20-km TT bike performance was strongly associated to the relative power (W/kg) at the lactate threshold in handcyclists but rather better correlated to maximal aerobic power in paratriathletes<sup>44</sup>. These results indicate some differences in the physiological determinants of performance between lower and upper body cycling, suggesting that findings from studies performed with Olympic cyclists cannot be generalized to handcyclists and vice versa, thus, reinforcing the differences in the physiology of lower and upper body sports.

### **1.3.2 Role of the anaerobic energy system in handcycling**

Anaerobic (glycolytic) activity is defined as being exhaustive, lasting less than 90 seconds and conducted with smaller quantities of oxygen<sup>70</sup>. The two major energy systems involved in the anaerobic metabolism are the adenosine-triphosphate-phosphocreatine or anaerobic alactic system, lasting 3 to 15 seconds during maximal effort and the anaerobic glycolysis or anaerobic lactic system which is involved for the remaining of the maximal effort<sup>70</sup>.

Anaerobic capacity is an important parameter in high intensity endurance events, predominantly for the start and finish of races but also for bursts of accelerations to pass opponents, although there is a small contribution of the anaerobic energy system in long events<sup>68</sup>. Surprisingly, very few studies investigated the anaerobic capacity of highly trained handcyclists, even though races at the elite level are generally won with a sprint finish. Stone et al. (2019) reported maximum PO values of  $337 \pm 59$  W in elite handcyclists<sup>14</sup>. In another study, Van der Woude et al. (1997) assessed the anaerobic capacity of wheelchair athletes specializing in various sports and concluded that anaerobic work capacity appeared to be strongly influenced by functionality (related to the amount of active muscle mass), hours of training and gender<sup>69</sup>.

The 30-second all-out Wingate Anaerobic Test measures the muscle's ability to use these two systems. The Wingate typically measures lower or upper body peak power (muscular strength and speed), anaerobic capacity, and the reduction of power (fatigue index) on a cycle ergometer<sup>70</sup>. The pedal resistance is usually set at a certain percentage of the athlete's body weight<sup>70</sup>. PO is measured by the number of revolutions achieved over the 30 seconds, peak power is the maximal PO achieved over  $\sim 5$  seconds (usually the first 5 seconds), anaerobic capacity is the average power over the 30 seconds, and the difference in power between the highest power and lowest power (generally the last 5 seconds) is known as the fatigue index (FI)<sup>70</sup>. Thus, aerobic (incremental  $\text{VO}_2\text{max}$  test and time-trial effort), and anaerobic testing such as an upper body Wingate should be assessed to provide a complete physiological profile of highly trained handcyclists and to understand the contribution of the cardiovascular (central and peripheral adaptations), respiratory, and neuromuscular systems to performance.

## **1.4 Physiological characteristics of athletes with a spinal cord injury**

Disparities exist in heart size and function among athletes who had a spinal cord injury (SCI) and able-bodied athletes. Endurance trained individuals possess enlarged hearts, high hemoglobin mass (Hb mass) and high total blood volume, but also an elevated stroke volume partly due to the enlargement of heart cavities<sup>34</sup>. Despite similar training loads to able-bodied athletes, heart hypertrophy is not consistently observed in athletes with a SCI, attributable to a reduced venous return due to venous blood pooling and smaller vasculature (reduced diameters of large vessels), but also to a reduced stroke volume and diminished active muscle mass caused by arm-exercises<sup>34,62</sup>. In addition, observations of lower HRmax are often seen in athletes with a SCI due to autonomous neural system fibers denervation further limiting  $\dot{Q}$ <sup>34,61</sup>. Houtman and colleagues (2000) found in sedentary males with a T4 and above lesion SCI, a lower total blood volume and a lower Hb mass with samples taken in the brachiocephalic vein, associated to paralysis and sedentary lifestyle, reducing the overall amount of muscle mass<sup>55</sup>. Authors also associated the lower total blood volume and Hb mass to the impairment of the sympathetic nervous system, which affects blood volume regulation<sup>55</sup>. These physiological changes could be associated to lower  $\text{VO}_2\text{max}$  as compared to sedentary able-bodied men.

Additionally, Schumacher and colleagues (2009) recruited endurance athletes with a SCI, untrained participants with a SCI and able-bodied endurance athletes, and assessed total Hb mass with the carbon monoxide rebreathing method, and cardiac dimensions with echocardiography, allowing the calculation of cardiac volume<sup>34</sup>. Participants also performed an incremental exercise test to measure  $VO_2$ peak<sup>34</sup>. The main finding of Schumacher's study was that total Hb mass adapts to chronic endurance training in trained participants with a SCI (total Hb mass per kg was 30% higher in athletes with a SCI than in untrained people with a SCI), while cardiac volume did not<sup>34</sup>. Overall,  $VO_2$ max and hemoglobin mass are largely diminished in participants with a SCI, however, they can adapt to prolonged endurance training, as suggested by Houtman and co-workers (2000) who trained sedentary men with a SCI with electrically stimulated leg cycling for 6 weeks and found a drastic increase in total blood volume and Hb mass relative to body mass following the exercise intervention<sup>34,55</sup>. However, trained participants with a SCI can only reach the Hb mass levels of untrained able-bodied individuals but not the levels of trained able-bodied participants<sup>34</sup>.

Hopman and colleagues (1998) suggested that peripheral changes induced by arm exercise, determining maximal oxygen utilization, are the main limiting factors of  $VO_2$ peak in individuals with a SCI<sup>61</sup>. The reduced amount of active muscle mass in SCI would probably limit the oxygen demand and consumption during maximal arm exercise<sup>61</sup>. Skeletal muscle oxidative capacity may also be lower since it is associated to the amount of active muscle mass and to the mitochondrial oxidative capacity<sup>61</sup>. In handcycling, smaller upper body structures are responsible for the propulsion, and the physiological patterns of the sport are different than in able-bodied cycling, which involves larger muscle groups<sup>62</sup>.

Moreover, LT is dependent on the amount of muscle mass involved in the exercise activity<sup>63</sup>. There is some evidence that paraplegia leads to lower  $VO_2$  peak and lower LT, with higher submaximal exercise blood lactate in paraplegic individuals than in able-bodied subjects<sup>63</sup>. However, there is no significant difference in absolute  $VO_2$  at the LT between untrained able-bodied individuals and untrained individuals with paraplegia<sup>64</sup>. Overall, arm exercise involves different peripheral cardiovascular mechanisms than whole-body training, therefore, it would be of great interest to investigate in depth the peripheral cardiovascular changes in athletes with a SCI.

### **1.5 Muscle recruitment in handcycling**

Recumbent handcycling muscle electromyography (EMG) has been conducted in few studies which generally used able-bodied athletes or sedentary participants. A study conducted with an able-bodied participant inexperienced in handcycling revealed EMG activation of several primary mover muscles defined as being active for at least 5% of each entire arm cycle<sup>12</sup>. These muscles were the biceps brachii, the anterior and posterior fibers of the deltoids, the pectoralis major fibers, the triceps brachii and the upper fibers of the trapezius (Figure 6)<sup>12</sup>. Another EMG project by Quittmann and colleagues (2019) subjected 12 able-bodied male triathletes to an incremental handcycling test and found the latissimus dorsi muscle to be supporting the handcycling propulsion phase of the crank cycle, thus acting as a stabilizer in this group of participants. Overall, Quittmann and his team (2019) found an increased activity for the biceps brachii, posterior and anterior fibers of the deltoids, triceps brachii, extensor carpi radialis and rectus abdominis muscles with an increase in workload but in contrast, muscle activation was not altered by workload for the pectoralis major and for the flexor carpi radialis muscles (Figure 5)<sup>13</sup>. Further research including trained handcyclists with EMG analysis is required to confirm the muscles involved in the sport.

Alike leg cycling, handcycling kinematics revealed continuous force application throughout the propulsion cycle, which consists of a whole revolution<sup>12</sup>. One study characterized the upper body kinematics of handcycling during sports specific intensities in a group of competitive and recreational recumbent handcyclists<sup>14</sup>. Stone and colleagues (2019) found that competitive handcyclists flex their thorax, extend their shoulder and posterior tilt their scapulae significantly more than recreational handcyclists<sup>14</sup>. These differences in scapular motions only occurred at lower intensities (50%  $PO_{peak}$ ), but shoulder extension and thorax flexion also occurred during higher intensity efforts (70%  $PO_{peak}$ )<sup>14</sup>. These kinematic disparities between recreational and competitive handcycling athletes could be attributed to muscle recruitment optimization and force generation by the arm due to technical training adaptations, re-emphasizing that it is difficult to compare findings on recreational handcyclist with highly trained athletes<sup>14</sup>.

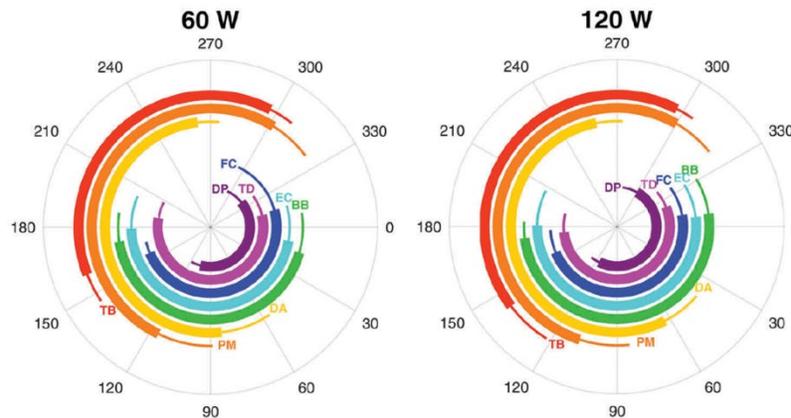


Figure 5. Muscular Activity Above Threshold with Respect to Crank Angle at 60 W and 120 W in 12 able-bodied male competitive triathletes. Thick lines represent muscular activity above 30%. Thin lines represent the standard deviation addition of the onsets and offsets. BB = M. biceps brachii, Caput breve; DA = M. deltoideus, Pars clavicularis; DP = M. deltoideus, Pars spinalis; EC = M. extensor carpi ulnaris; FC = M. flexor carpi radialis; PM = M. pectoralis major, Pars sternalis; TB = M. triceps brachii, Caput laterale; TD = M. trapezius, Pars descendens. Quittmann et al., 2019

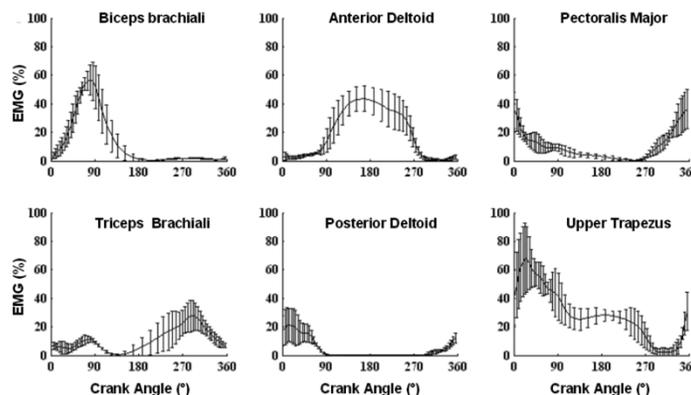


Figure 6. EMG Pattern Normalized by Maximal Voluntary Contraction for Six Muscles Recorded in terms of Crank Angle (0-360°) in an Able-Body Subject, Faupin et al., 2010

## 1.6 Handcycling and muscle oxygenation

### 1.6.1 Muscle oxygenation

Near-infrared spectroscopy (NIRS) gained popularity in the 1980s and had been used since to assess muscle oxygenation changes during exercise<sup>22,23,58</sup>. NIRS can be used to understand if the dynamic balance between O<sub>2</sub> demand and supply and the muscle O<sub>2</sub> extraction are the main determinants of performance in trained handcyclists. NIRS can be used with portable and non-invasive monitors during different exercise interventions to measure SmO<sub>2</sub><sup>23,58</sup>. The signal represents the weighted average of the oxygen saturation of the heme group of hemoglobin (Hb) mostly in the muscle tissues around the capillaries, since these small vessels contribute to > 90% of the muscle total blood volume during exercise, but also of the heme group of myoglobin in muscle fibers<sup>58</sup>. NIRS measurements exclude large vessels (arteries and veins) since their hemoglobin concentration is too high and light interacting in these regions is absorbed<sup>59</sup>. In contrast, capillaries are less optically dense areas, so that the NIRS photons can be refracted into the detector of the monitor<sup>59</sup>.

Different NIRS techniques based on specific illumination types are characterized by:

1. Continuous-wave modality, which measures attenuation of constant light in the tissues;
2. Frequency-domain model which measures the attenuation and the shifting phase of the emerging light;
3. Time-domain modality collects shape of the pulses of light after propagation in the tissues<sup>23</sup>.

The most widely used oximetry technique is the constant-wave spatially resolved spectroscopy modality, since it is transportable and low-cost, whereas frequency and time-domain technologies are more expensive and complex<sup>23</sup>. NIRS temporal resolution ranges from 2 Hz (Moxy monitor) up to 100 Hz and can sample changes in SmO<sub>2</sub> for 2-4 seconds exercise bouts<sup>23</sup>. One unique advantage of NIRS is that it can provide acceptable signal-to-noise ratios during exercise when movement artefacts are limited<sup>58</sup>. However, at high exercise intensities, more frequent muscle contractions can cause movement artefacts and tissue ischemia, leading to greater variations in measurements<sup>32</sup>.

### 1.6.2 NIRS and the Moxy monitors

The Moxy monitor became popular recently since the small and lightweight device is easily transportable on the field and do not require any wiring<sup>32</sup>. Moxy monitors are continuous-wave devices that have data storage and telemetric capability<sup>32</sup>. The reliability of these devices is similar to traditional NIRS instruments<sup>32</sup>. Moxy monitors sequentially send light waves at and just beyond the red end of the visible spectrum, at 630 nm (clearly visible to human eye) to 850 nm (barely visible to human eye) from four light emitting diodes into the muscle tissue beneath its application and record the amount of scattered (returned) light to the tissues at two detectors positioned 12.5 and 25 mm from the light source<sup>32</sup>. NIRS light travels through skin, fat, and muscle tissue without being completely absorbed<sup>59</sup>. NIRS monitors use different methods to measure or correct for the scattering effect. The penetration depth of the light received at each detector is half the distance between the light source and the detector and the scattered light is processed via an algorithm (a tissue light propagation model and the modified Beer-Lambert law) to determine the amount of light absorbed at wavelengths associated to oxy- and deoxyhemoglobin<sup>32</sup>. NIRS light is absorbed differently by hemoglobin and myoglobin molecules that have oxygen bound to them making it possible to quantify the percentage of hemoglobin and myoglobin molecules that are carrying oxygen<sup>59</sup>.



Figure 7. The Moxy Monitor. Fortiori Design LLC., 2015

### 1.6.3 Modified Beer-Lambert Law

The approximate tissue concentration of oxy- and deoxy-hemoglobin are estimated by measuring the optical pathlength of NIRS photons crossing the tissue<sup>28</sup>. The mechanism relies on the relationship between absorption and chromophore concentration in the modified Beer-Lambert Law for the scattering media:  $A = \epsilon [c] LB + G$ , where  $A$  is the absorption of light expressed as optical density,  $\epsilon$  the extinction coefficient of the chromophore,  $[c]$  the chromophore concentration,  $L$  the distance between the point of light entry and exit (optode separation),  $B$  the pathlength from scatter in the tissue and  $G$  a parameter associated to tissue and optode geometry<sup>28</sup>.

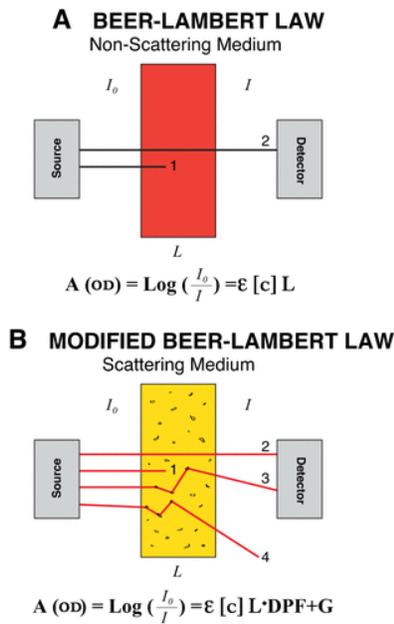


Figure 8. Beer-Lambert Law for a Non-Scattering Solution and Modified Beer-Lambert Law to Account for Scattering in Biological Tissues. 4, Loss of photons from field of view due to scattering, but not absorption ( $G$  in Modified Beer-Lambert equation);  $I_0$ , source light intensity;  $I$ , detected light;  $L$ , source-detector distance. 1, loss of photons due to absorption; 2, photons that are not absorbed and travel a non-scattering path length (source-detector distance  $L$ ) to be recorded by detector; 3, photons that are not absorbed but are scattered on their way to the detector, increasing their path length by  $L \cdot \text{DPF}$ . Barstow, 2018

### 1.6.4 Variables obtained with NIRS

Different NIRS methods measure the Hb saturation of the muscle tissue ( $\text{SmO}_2$ , %), where  $\text{SmO}_2$  accurately quantifies the oxygenation changes in the muscle reflecting the dynamic balance between oxygen supply and oxygen consumption<sup>23</sup>. The formula to calculate  $\text{SmO}_2$  is  $[(\text{oxygenated hemoglobin} + \text{oxygenated myoglobin}) / (\text{total amount of hemoglobin} + \text{myoglobin})] \times 100$

100]<sup>59</sup>. Deoxyhemoglobin concentration ([HHb]), represents muscle O<sub>2</sub> extraction. Only the frequency and time-domain NIRS methods provide accurate concentrations of oxyhemoglobin ([HbO<sub>2</sub>]), [HHb] and a derived parameter, total hemoglobin ([THb]), where [THb] = [HbO<sub>2</sub>] + [HHb]<sup>23</sup>. The parameters [HbO<sub>2</sub>] and oxymyoglobin ([MbO<sub>2</sub>]) reflect the tissue O<sub>2</sub> delivery, while the [HHb] and deoxymyoglobin ([HMb]) parameters reflect the tissue O<sub>2</sub> utilization<sup>72</sup>. Reliable muscle oximeters can provide specific measures such as muscle oxygenation level, deoxygenation rate and reoxygenation rate following a training intervention<sup>58</sup>. The desaturation curve at the beginning of exercise is represented by a negative slope during the muscle contraction phase at the onset of exercise, whereas a more negative (faster) SmO<sub>2</sub> slope would be attributed to an acceleration in muscle O<sub>2</sub> demand and energy consumption, that is greater than the acceleration of the O<sub>2</sub> supply<sup>23</sup>.

Parameter	Abbreviation	Function
Muscle oxygen saturation (%)	SmO <sub>2</sub>	Balance between O <sub>2</sub> supply and consumption
Deoxyhemoglobin + deoxymyoglobin concentration, AU	[HHb] + [HMb]	Tissue O <sub>2</sub> extraction
Oxyhemoglobin + oxymyoglobin concentration, AU	[HbO <sub>2</sub> ] + [MbO <sub>2</sub> ]	Tissue O <sub>2</sub> delivery
Total hemoglobin, AU	[THb]	Local blood volume

Table 3. Overview of the Various Parameters Provided by NIRS Technology. Definition: AU, Arbitrary Units.

### 1.6.5 The effect of training on NIRS parameters

Paquette et al. (2018) investigated muscle oxygen saturation and its correlation to performance in highly trained canoe-kayakers using NIRS (Moxy monitors). Authors were interested to know if the peripheral cardiovascular marker SmO<sub>2</sub> is better correlated to performance than VO<sub>2</sub>max in short and long-distance canoe-kayak events, a sport predominantly involving the upper body alike handcycling<sup>27</sup>. In fact, authors found that SmO<sub>2</sub> parameters are better associated to performance than VO<sub>2</sub>max in competitive canoe-kayakers<sup>27</sup>. This important finding could also be true for handcyclists using primarily isolated upper body muscles for propulsion, in which peripheral adaptations of VO<sub>2</sub> may predominate over the central VO<sub>2</sub> adaptations to predict performance.

Moreover, Shibuya and colleagues (2004) measured muscle oxygenation of the vastus lateralis in 9 males performing an incremental cycling test with a NIRS instrument (mmmBOM-L1TR), which uses three laser diodes and calculates relative tissue levels of oxy-, deoxy-, and total hemoglobin according to the modified Beer-Lambert Law. Muscle oxygenation during the incremental cycling test was expressed as a percentage of the maximal range observed during an arterial occlusion as the lower reference point. Authors found an important decrease in muscle oxygenation with increasing cycling intensity, indicating more muscle O<sub>2</sub> utilization than muscle O<sub>2</sub> supply, due to a greater muscle O<sub>2</sub> extraction<sup>24</sup>. The authors also found a significant relationship between thigh muscle oxygen extraction ability (muscle oxygenation at exhaustion) and maximal oxygen consumption (VO<sub>2</sub>max) during cycling<sup>24</sup>. Thus, it remains to be confirmed if muscle oxygen extraction capacity might be a strong contributing factor to performance in handcycling as well, where smaller muscles are involved in the propulsion.

## CHAPTER 2: MANUSCRIPT

### Peripheral Adaptations in High-Performance Handcycling

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**Introduction:** The main physiological determinants of handcycling were described in the literature as  $\text{VO}_2\text{max}$ , ventilatory and lactate thresholds and maximal aerobic power (MAP). It remains to be confirmed if handcycling performance is well associated to other peripheral adaptations as measured by muscle oxygenation changes. **Purpose:** To develop a physiological profile of well-trained handcyclists while assessing the associations between physiological parameters (central and peripheral), and peak and mean power output during a 30-second maximal sprint test and a maximal incremental test and time trial (TT) performance. **Methodology:** Six athletes including three women and 3 men competing in recumbent handcycling classes (H2-4) participated in a Wingate, a maximal incremental test and a 20-minute TT with variations in load, on an arm ergometer, where changes in muscle oxygenation of the biceps brachii (BB), the triceps brachii (TB), the anterior fibers of the deltoid (AD), and the extensor carpi radialis brevis (ER) were measured using near-infrared spectroscopy (NIRS) monitors. Maximal muscle  $\text{O}_2$  extraction as maximal deoxyhemoglobin content ([HHb]), rate of muscle deoxygenation (beginning of effort) and reoxygenation (end of effort), and maximal muscle desaturation as the ability to sustain low oxygenation ( $\text{SmO}_2$  minimal) were measured with NIRS. Oxygen consumption ( $\text{VO}_2$ ), cardiac output (Q), stroke volume (SV), heart rate (HR), blood lactate concentration, and power output, were measured. Grip strength was also assessed bilaterally using a handheld dynamometer. **Results:** High  $\text{VO}_2\text{max}$  ( $R = 0.90$  [0.08, 1.91],  $p = 0.040$ ) and maximal  $\text{O}_2$  extraction, especially of BB ( $R = 0.86$  [0.16, 1.57],  $p = 0.027$ ) were related to a high maximal aerobic power (MAP) during the incremental test. Maximal  $\text{O}_2$  extraction, especially of AD ( $R = 1.0$ ,  $p < 0.001$ ), rapid reoxygenation rate especially of TB ( $R = 0.82$  [-0.15, 0.99],  $p = 0.046$ ), AD ( $R = 0.92$  [0.17, 1.57],  $p = 0.029$ ) and ER ( $R = 0.83$  [-0.13, 0.99],  $p = 0.042$ ), rapid deoxygenation rate, especially of AD ( $R = -0.90$  [-1.00, 0.17],  $p = 0.037$ ) and ER ( $R = -0.87$  [-1.56, -0.18],  $p = 0.025$ ) were related to a higher mean power in the Wingate. The ability of muscles to sustain low levels of oxygenation ( $R = -0.88$  [-1.76, 0.01],  $p = 0.049$ ), rapid deoxygenation rate especially of the AD ( $R = -1.00$  [-0.83, 0.63],  $p < 0.001$ ), BB ( $R = -0.95$  [-1.36, 0.03],  $p = 0.036$ ), and ER (-0.90 [-1.00, 0.17],  $p = 0.037$ ), were related to a higher mean power in the TT, rapid reoxygenation rate especially of the AD ( $R = 0.97$  [0.18, 1.51],  $p = 0.032$ ) and of the BB (-0.99 [-0.98, 0.30],  $p = 0.015$ ) were related to a higher mean power (% MAP) and to a greater distance in the TT, respectively. Maximal ER  $\text{O}_2$  extraction in the incremental test was 37% higher than in the Wingate, and ER deoxygenation rate in the Wingate was 88% faster than in the TT. For the 3 physiological variables examined (heart rate,  $\text{VO}_2$  and  $\text{SmO}_2$ ), more time was spent in the highest intensity zone (above 2<sup>nd</sup> lactate threshold) as compared to the time spent in the lower intensity zones during the TT, demonstrating major contribution of the anaerobic energy system. **Conclusions:** Results demonstrate the contribution of peripheral adaptations to predict handcycling performance. Consequently, these should be considered in training plans of elite handcyclists.

**Keywords:** muscle oxygen saturation, peripheral adaptations, aerobic fitness, handcycling

## Introduction

At the Paralympics, handcycling athletes compete in two outdoor events including the individual TT and the road race. TTs vary from 10-30 km for women and 12-35 km for men (Muchaxo et al., 2020), lasting 20-40 minutes (Stone et al., 2020). Criteriums range from 37-70 km for women and 45-80 km for men (Muchaxo et al., 2020) over a duration of 60-150 minutes (Stone et al., 2020). Studies identified the contribution of the aerobic metabolism as the primary energy system in the handcycling marathon (42.195 km) and unsurprisingly in ultralong handcycling events (up to 540 km), while shorter Paralympic TT distances (10-22 km) are associated to both the aerobic and the anaerobic energy systems contribution (Stephenson, 2020 and Fisher, Figuierdo & Ardigò, 2015). The anaerobic contribution generates power output in climbs, surges, and sprint finishes (Nevin, 2017). Most studies in handcycling were conducted on sedentary participants, making it difficult to generalize the findings to elite athletes. Only a few studies explored the role of physiological determinants of well-trained handcyclists.  $VO_2$ max, ventilatory and lactate thresholds and MAP are the best determinants of handcycling performance according to these studies (Fisher, Figuierdo & Ardigò, 2015 and Stone et al., 2015). No study investigated in detail the peripheral cardiovascular adaptations in well trained handcyclists as measured by muscle oxygenation. Moreover, there is very little information published regarding the fitness of female handcycling athletes.

Since handcyclists predominantly use smaller isolated upper body muscles for propulsion and generate lower  $VO_2$ max and stroke volume as compared to able-body athletes, it remains to be confirmed if peripheral oxygen extraction is well developed in this group of athletes (Schumacher et al., 2009). In whole-body endurance sports where large muscle masses are activated (e.g., running and leg cycling), it is well known that cardiac output (Q) is the main cardiovascular limitation to  $VO_2$ max (Basset, 2000). Sports recruiting small muscles masses may present unique physiological determinants due to an increased blood flow to the isolated muscular areas involved in the exercise and an increase in  $O_2$  diffusion from the blood to the muscle mitochondria, resulting in a greater muscle  $O_2$  extraction capacity (Basset, 2000). Arm muscles also display larger cross-sectional area of type II muscle fibers and lower oxidative enzyme activity (Mygind, 1995). Thus, peripheral cardiovascular adaptations to training may be more important in handcycling than the central determinants of  $VO_2$ , as supported by Paquette, Bieuzen & Billaut (2018), demonstrating that muscle oxygenation is the best predictor of sprint canoe-kayak performance, a sport predominantly using the upper-body for propulsion.

Recumbent handcycling muscle electromyography (EMG) has been conducted in few studies. A study with an able-bodied participant inexperienced in handcycling revealed EMG activation of several primary mover muscles defined as being active for at least 5% of each entire arm cycle (Faupin et al., 2010). These muscles were the biceps brachii, the anterior and posterior fibers of the deltoids, the pectoralis major fibers, the triceps brachii and the upper fibers of the trapezius (Faupin et al., 2010). Another EMG project by Quittmann and colleagues (2019) subjected 12 able-bodied male triathletes to an incremental handcycling test and found an increased activity for the biceps brachii, posterior and anterior fibers of the deltoids, triceps brachii, extensor carpi radialis and rectus abdominis muscles with an increase in workload (Quittmann et al., 2019). Further research including trained handcyclists with EMG and NIRS analysis is required to confirm the muscles involved in the sport. NIRS monitors gained popularity in the past decades due to their affordability, portability, and ability to measure muscle oxygenation changes during exercise.

Alike leg cycling, handcycling kinematics revealed continuous force application throughout the

propulsion cycle, which consists of a whole revolution (Faupin et al., 2010). One study characterized the upper body kinematics of handcycling during sports specific intensities in a group of competitive and recreational recumbent handcyclists (Stone et al., 2019). Stone and colleagues (2019) found that competitive handcyclists flex their thorax, extend their shoulder and posterior tilt their scapulae significantly more than recreational handcyclists (Stone et al., 2019). These differences in scapular motions only occurred at lower intensities (50%  $PO_{peak}$ ), but shoulder extension and thorax flexion also occurred during higher intensity efforts (70%  $PO_{peak}$ ) (Stone et al., 2019). These kinematic disparities between recreational and competitive handcycling athletes could be attributed to muscle recruitment optimization and force generation by the arm due to technical training adaptations, re-emphasizing that it is difficult to compare findings on recreational handcyclist with highly trained athletes (Stone et al., 2019).

The objectives of this study were (1) to develop a physiological profile of well-trained recumbent handcyclists (men and women) competing in H2-4 classes (2) to assess and compare changes in muscle oxygenation between different efforts and muscles (3) to examine associations between peripheral adaptations (muscle saturation and maximal  $O_2$  extraction) and handcycling performance (peak and mean anaerobic power, MAP and TT performance) and (4) to assess the physiological response during a handcycling TT. In a sport predominantly using smaller upper body muscles like handcycling, it is hypothesized that peripheral physiological determinants (muscle oxygen extraction capacity) will be better correlated to performance than the central physiological determinants ( $VO_2max$  and cardiac output).

## Methods

### Participants

Six athletes participated in this study, of which 3 were men (2 competing in the H3 handcycling class and 1 competing in the H2 handcycling class) and 3 were women (all competing in the H4 handcycling class). Five athletes compete at the international level (4 for the Canadian National Team and one for the Nederland National Team) and one female athlete compete at the provincial level. Participants were  $33.5 \pm 2.6$  years of age (range 31–38 y old) and weighed  $64.3 \pm 13.0$  kg (Men:  $74.4 \pm 8.7$ , Women:  $54.1 \pm 6.0$ ). The study was approved by the Concordia University ethics committee and every participant provided written informed consent.

### Experimental Design

During the nine months following the 2021 Tokyo Paralympics, handcyclists performed two anaerobic tests, a maximal incremental test, and a TT to examine the relations between power output and TT performance with various physiological variables in laboratory. A hand grip test, a 30-second maximal arm power test (Wingate) followed by a 30-minute break and by a maximal incremental test were conducted on the first day and the 20-minute TT was performed on the consecutive day.

### Questionnaires

Participants filled a PAR-Q+ to assess the risks of engaging in strenuous physical activity. Participants were asked to provide a questionnaire with their name, age, gender, height, physical

impairment history, handcycling class, level of experience in handcycling and in endurance sports, injury history, participation in other sports, participation in provincial, national, or international handcycling competitions, weekly training volume and medications taken. Athletes filled a food record upon arrival in the lab on both days.

### **Anthropometry**

Participants had their weight measured with an adapted scale for wheelchair users (Seca, Hamburg, Germany) and girths of the right upper arm and right upper forearm measured according to the ISAK protocol (Norton, 2018).

### **Ergometer Testing**

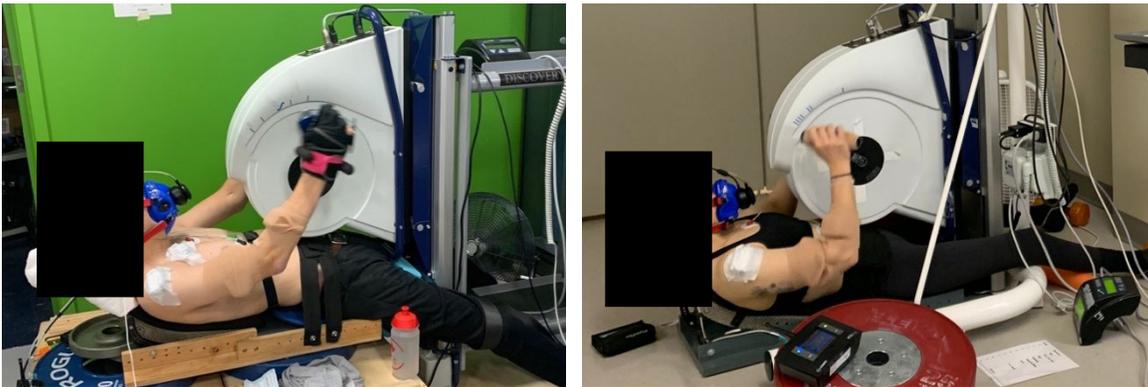


Figure 1. Man athlete (on the left) and Figure 2. Woman athlete (on the right) setup with the Lode Angio arm ergometer during the study.

Athletes were placed and setup according to comfort in a racing recumbent handcycling wooden chair built and used by Cycling Canada for laboratory testing, which mimics the racing position (Figures 1 and 2). Athletes performed all tests with an arm ergometer (LODE Angio 917900, Groningen, Netherlands).

### **Grip Strength**

On the first test day, participants had their grip strength assessed bilaterally with a hydraulic handheld dynamometer (Tekdynamics, Irving, New York) and performed two trials on each side without warm-up. A first trial on both sides was conducted, followed by a very short rest, and by the second trial on both sides. All scores were rounded to the nearest 0.5 kg and the best score was chosen on both sides.

### **Wingate Test**

Athletes performed a maximal synchronous arm cranking power test, the 30-second Wingate. Athletes warmed-up for a light 5-min of cranking at 50 W, followed by a pre-programmed 2-minute protocol on the Lode Angio software which included two to four short sprints of 6 seconds. The torque factor (Nm/kg) was set between 0.3 and 0.6 depending on the handcyclist's class and gender. Athletes were instructed to reach peak power as soon as possible at the beginning of the test, and to maintain power as high as possible until the end of the test. Loud encouragements

were provided to insure a maximal effort. The ergometer was calibrated before each test according to the manufacturer's recommendations and crank length was adjusted for each participant. Mean power over the 30-second test was assessed, peak power represented the highest 1-second power achieved in the test, and fatigue index was the difference in power between the highest 1-second and the lowest 1-second achieved during the test.

### **Maximal Incremental Test**

All participants performed a continuous  $\text{VO}_2\text{max}$  test consisting of five or six 3-minute stages of increasing intensity on the Lode Angio arm ergometer (synchronous cranking). Participants received cadence feedback during the test as they were asked to maintain a speed above 70 RPM for the full duration of the test. Expired air was continuously recorded using a portable gas analyzer (K5, COSMED, Rome, Italy) for five athletes and the Vmax Encore metabolic cart (CareFusion Corp, San Diego, CA) for one athlete due to technical difficulties with the K5. Maximal aerobic power (MAP) was the average PO generated during the last 3-minute of the test. Cardiac output (Q) and stroke volume (SV) were evaluated during the test using thoracic electrical bioimpedance (Physioflow; Manatec Biomedical, Poissy, France). Arterio-venous  $\text{O}_2$  difference ( $[\text{a-v}]\text{O}_2$  diff [mL/dL]) was calculated using the following equation:  $(\text{a-v})\text{O}_2 \text{ diff} = \text{VO}_2 (\text{L}/\text{min})/\text{Q} (\text{L}/\text{min}) \times 100$ .  $\text{VO}_2\text{max}$ , maximal Q ( $\text{Qmax}$ ), maximal SV ( $\text{SVmax}$ ), and maximal  $(\text{a-v})\text{O}_2$  diff were defined as the highest values achieved over a 30-second period during the test. Maximal respiratory exchange ratio ( $\text{RERmax}$ ) was the average of RER from the 30-second period when  $\text{VO}_2\text{max}$  occurred.

### **Time Trial**

On the second testing day, 5 participants performed a progressive 15-minute warm-up and completed a 20-minute TT (synchronous arm cranking) with self-selected speed (cadence) but fixed variations in loads using the Lode Angio arm ergometer, which replicated the Tokyo 2020 TT course. Expired air was continuously recorded using a portable gas analyzer (K5, COSMED, Rome, Italy). Q and SV were also evaluated during the test using thoracic electrical bioimpedance (Physioflow; Manatec Biomedical, Poissy, France). Different intensity zones were analyzed during the TT. Zone 1 consists of efforts below the first lactate threshold, zone 2 intensities are between the first and second lactate thresholds, and zone 3 intensities are above the second lactate threshold. For  $\text{SmO}_2$ , zones and thresholds were calculated with data from the incremental test, by plotting power at each stage (average of the last 30 seconds of every stage in the incremental test) with  $\text{SmO}_2$  at each stage (average of the last 30 seconds of every stage in the incremental test), the equation of the function was calculated, and MAP at the lactate thresholds was used in the equation.

Segment	Time (minute)	Slope (%)
S1	0 to 2	-0.9
S2	2 to 3	-4.5
S3	3 to 5	2.5
S4	5 to 5.5	-4.1
S5	5.5 to 9	3.3
S6	9 to 11	1.2
S7	11 to 12.5	-4.5
S8	12.5 to 14	4.2
S9	14 to 15	-0.1
S10	15 to 18	9.5
S11	18 to 19	-0.4
S12	19 to 20	2.1

Table 1. Description of the 20-Minute Handcycling TT Course

### Muscle Oxygenation

For all ergometer tests, NIRS monitors (Moxy monitors; Fortiori Design, Minnesota, USA) were used to assess changes in muscle oxygenation. The Moxy monitor utilizes four wavelengths of near-infrared light (680, 720, 760, and 800 nm), with source detector spacing of 12.5 and 25.0 mm (McManus et al., 2018). Skinfold thicknesses of four highly active muscle groups in handcycling, supported by handcycling electromyography studies (Faupin et al., 2010 and Quittmann et al., 2019), were measured to the nearest millimetre on the right side of the body, using calipers (Harpenden, Holtain Ltd, United Kingdom). The triceps brachii (TB) was pinched at the medial aspect approximately 6 to 8 cm from the elbow epicondyle (Kounalakis et al., 2008). The biceps brachia (BB) was pinched at the middle of the muscle belly, 8-12 cm above the elbow fold (Paquette, Bieuzen & Billaut, 2018). The anterior deltoid (AD) was pinched at one finger width distal and anterior to the acromion, in the direction of the line between the acromion and the thumb (SENIAM). The extensor carpi radialis brevis (ER) was pinched 4 cm distal to a point half-way between the supracondylar ridge of the humerus and the inner band of the elbow (Crenshaw et al., 2012). These landmarks were identified and were used to place the Moxy monitors later on. Skinfold thickness at each site was measured to ensure it is < than ~60% of the distance between the emitter and the detector of the Moxy monitor (maximum of 15 mm) so that the photons penetrate the skin, and the absorbency of infrared light is optimal (Feldmann, Schmitz & Erlacher, 2019). Power output (PO) was recorded and the PerfPRO software (Studio 2021) was used to retrieve live SmO<sub>2</sub> data.

The Moxy monitors were placed on four active muscles of the right side of the body, parallel to the muscle fibers orientation. They were attached and secured with a double-sided adhesive disk and tape and covered by bandage to reduce the intrusion of extraneous light. The raw SmO<sub>2</sub> signal, representing the balance between O<sub>2</sub> delivery and O<sub>2</sub> consumption by the muscle, and total hemoglobin content ([THb]), an indicator of local blood volume, were treated using a smooth spline filter on the R software to reduce the noise generated by movement (Paquette, Bieuzen & Billaut, 2021). The deoxyhemoglobin concentration ([HHb]), representing muscle O<sub>2</sub> extraction, was computed from SmO<sub>2</sub> and [THb] using the following equation:  $[HHb]=[THb]-SmO_2 \times [THb]$  (Ferrari,

Muthalib & Quaresima, 2011). Baseline  $SmO_2$  and [THb] were computed as a 2-minute average when athletes sat still on the ergometer before the beginning of the tests.  $SmO_{2min}$  depicts the peak deoxygenation (peak deoxy) and was calculated for every muscle in each test.  $SmO_{2min}$  represents the lowest 5-second average  $SmO_2$  reached during the test (the nadir), expressed in  $\Delta SmO_{2min}$  which is in change from baseline  $SmO_2$  ( $SmO_{2min} - SmO_{2baseline}$ ).  $\Delta[HHb]_{max}$  is in change from baseline values; and represents the highest 5-second average in each test.  $SmO_{2mean}$  and [THb] $_{mean}$  was the average of the test, also expressed in change from the baseline ( $\Delta SmO_{2avg}$  and  $\Delta[THb]_{avg}$ ).  $SmO_{2mean}$  represents the average of the last stage for the incremental test. [THb] $_{mean}$  represents the average of the last stage for the incremental test.  $SmO_2$  mean and [THb] mean was not presented for the Wingate since the test is too short (30 seconds) to represent a steady state. A linear model was used to assess rapid changes in  $SmO_2$  where  $SmO_2$  vs. time values were modeled from the start to the 12<sup>th</sup> second of exercise for the Wingate and the TT (Paquette, Bieuzen & Billaut, 2021). The linear portion of the slope of the relationship represented an index of deoxygenation rate (deoxy rate), and reoxygenation rate (reoxy rate) represented the slope of the linear part of the  $SmO_2$  vs. time curve in the 30-second following the end of the exercise for each test (Paquette, Bieuzen & Billaut, 2021). Deoxy rate was not calculated for the incremental test due to the low intensity of the first stage.

### **Blood Lactate**

Blood was collected on the earlobe using a portable lactometer (The Edge Handheld Lactate Analyser, Woodley Equipment Company LTD, Lancashire, United Kingdom). Asepsis was performed with 70% ethylic solution on the distal portion of the earlobe before collection and puncture was conducted with disposable lancets. Blood lactate was collected prior to the incremental test to make sure the concentration was < 2 mmol/L prior to the start, at every stage during the incremental test, at the minute 1 and 3 following the incremental test and at the minute 1 and 3 following the TT. Rating of perceived exertion (RPE) on a Borg scale from 0 to 10 was collected at the end of the incremental test and at the end of the TT.

All devices were calibrated according to the manufacturer guidelines before every test.

### **Statistical Analysis**

Normality tests were performed to determine if the data was normally distributed. Means and standard deviations were calculated for performance and physiological parameters. Analysis of variance and Kruskal-Wallis tests for variables not normally distributed were performed to examine if there was a significant difference between groups (among the four muscles and among the three tests). Partial eta squared and epsilon squared effect sizes (ES) and 90% confidence intervals were computed to better emphasize the magnitude of the significant group difference when it existed. ES of 0.01 to 0.05 indicated a small effect, 0.06 to 0.13 a medium effect and 0.14 or greater indicated a large effect (Seo et al., 2018). Bonferroni and Scheffe post-hoc tests were conducted to determine which groups differed when a difference existed. Pearson correlations with 95% confidence limits were performed between physiological parameters and power or TT distance. Pearson and Spearman correlation coefficients > 0.6, > 0.7 and > 0.9 were considered moderate, large, and very large respectively (Akoglu, 2018). Stepwise multiple regressions were computed to determine the best combination of physiological variables to predict power output and TT performance. All statistical analysis were conducted on SPSS (version 28.0).

## Results

### Study Participants

Table 2 presents characteristics of the 6 participants included in this study. The VO<sub>2</sub> measurements are presented for 5 athletes due to technical issues. As a result of poor signal quality on some recordings, data from the Physioflow device are only presented for 3 athletes. The 6 athletes performed the Wingate and the incremental test, but the TT was performed by 5 athletes.

Variable	Athlete 1	Athlete 2	Athlete 3	Athlete 4	Athlete 5	Athlete 6
Athlete Code	MH2	MH3.1	MH3.2	WH4.1	WH4.2	WH4.3
Gender, F or M	M	M	M	F	F	F
Age, years	38	35	31	32	33	32
Weight, kg	66.5	73.0	83.8	53.3	48.6	60.5
Handcycling class	H2	H3	H3	H4	H4	H4
Competition level	International	International	International	International	International	Provincial
Handcycling experience, years	10	7	10	7	5	5
Training, hours/week	11.0	11.5	9.5	19.5	15.0	9.5
VO <sub>2</sub> max, L/min	2.4	2.9	—	2.3	1.8	1.5
VO <sub>2</sub> max, mL/kg/min	36.0	39.7	—	42.7	36.3	25.5
RERmax	1.1	1.2	1.1	1.1	1.2	1.3
RRmax, breaths/min	49.0	48.0	49.0	60.0	58.0	60.0
Lactate max, mmol/L	11.8	14.0	11.2	11.4	18.8	12.8
VEmax, L/min	89.4	104.9	—	103.6	93.3	82.4
MAP, W	147	205	212	142	140	90
MAP, W/kg	2.2	2.8	2.5	2.7	2.9	1.1
SVmax, mL/beat	—	125.1	164.4	—	109.1	—
Qmax, L/min	—	23.6	28.5	—	17.2	—
Max (a-v)O <sub>2</sub> diff, mL/dL	—	12.3	—	—	10.2	—
Max heart rate, beats/min	183	195	177	178	175	192
20-min TT distance, km	9.6	12.9	—	13.9	13.5	6.8
20-min TT mean P, W	120	161	—	113	108	68
20-min TT mean P, W/kg	1.8	2.2	—	2.1	2.2	1.1
20-min TT, mean P, %MAP	82	79	—	80	77	76
Wingate peak P, W	274.6	571.7	421.6	237.8	233.3	223.3
Wingate peak P, W/kg	4.1	7.8	4.2	4.9	3.9	5.0
Wingate mean P, W	214.2	420.9	193.3	192.7	190.3	338.6
Wingate mean P, W/kg	3.2	5.8	3.6	4.0	3.1	4.0
Fatigue Index, %	41.4	47.6	24.2	31.2	30.9	10.1
Right prehension force, kg	25.0	114.0	78.0	54.0	60.5	17.5
Left prehension force, kg	2.0	108.0	85.0	57.5	50.0	27.5
Right upper arm girth, cm	29.9	37.9	36.0	26.8	27.3	14.4
Right forearm girth, cm	25.6	31.0	28.0	23.3	22.8	16.6

Table 2. Characteristics of the Study Participants. Abbreviations: F, female; M, male; (a-v)O<sub>2</sub> diff, arteriovenous O<sub>2</sub> difference; Q, cardiac output; RER, respiratory exchange ratio; RR, respiratory rate; SV, stroke volume; P, power; TT, time trial; VO<sub>2</sub>max, maximal oxygen uptake; grip strength is the sum of 2 trials on each side; arm girth is the average of two trial

## Muscle Oxygenation Changes

Table 3 describes muscle oxygenation response to the Wingate, incremental test, and TT. The skinfold thickness of triceps brachii was  $10.3 \pm 3.8$  mm, biceps brachii was  $5.8 \pm 3.0$  mm, anterior deltoid was  $9.5 \pm 3.9$  mm and extensor carpi radialis brevis was  $5.7 \pm 1.6$  mm. For one athlete, the AD muscle was excluded from all analysis since the skinfold thickness exceeded the acceptable limit ( $> 15$  mm). Some NIRS data were lost due to excessive noise and signal.

Variable	Wingate (n = 6)	Incremental (n = 6)	Time-trial (n = 5)
$\Delta\text{SmO}_2\text{min, \%}$			
TB	-35 (12) n = 5	-51 (14) n = 5	-46 (7) n = 4
AD	-41 (13) n = 5	-43 (13) n = 5	-48 (11) n = 4
BB	-39 (10) n = 5	-48 (14)	-56 (6) n = 4
ER	-34 (14)	-48 (13)	-40 (12)
$\Delta\text{SmO}_2\text{mean, \%}$			
TB	—	-46 (13) n = 5	-31 (10) n = 4
AD	—	-37 (13) n = 5	-33 (7) n = 4
BB	—	-43 (15)	-35 (4) n = 4
ER	—	-41 (13)	-26 (10)
$\Delta[\text{THb}]_{\text{mean, AU}}$			
TB	—	-0.1 (0.4) n = 5	-0.1 (0.2) <sub>c,d</sub> n = 4
AD	—	0.2 (0.3) n = 5	-0.1 (0.1) <sub>c</sub> n = 4
BB	—	0.1 (0.5)	0.1 (0.1) n = 4
ER	—	0.3 (0.4)	0.1 (0.1)
$\Delta[\text{HHb}]_{\text{max, AU}}$			
TB	5.5 (2.1) n = 5	7.2 (6.4) n = 5	5.6 (1.1) n = 4
AD	5.2 (2) n = 5	7.7 (5.8) n = 5	5.8 (1.2) n = 4
BB	5.0 (1.5) n = 5	6.3 (2)	7.2 (1) n = 4
ER	3.4 (2.3) <sub>a</sub>	6.7 (1.6)	5.4 (1.7)
Deoxy rate, %/sec			
TB	-1.7 (1.5) n = 5	—	0.6 (0.8) n = 4
AD	-2.9 (3.1) n = 5	—	-0.3 (0.3) n = 4
BB	-2.6 (1.9) n = 5	—	-0.4 (0.4) n = 4
ER	-2.5 (2.2) <sub>b</sub>	—	-0.3 (0.3)
Reoxy rate, %/sec			
TB	0.9 (1.4)	0.5 (0.6) n = 5	0.3 (0.3) n = 4
AD	0.6 (0.6) n = 5	0.4 (0.5) n = 5	0.3 (0.2) n = 4
BB	0.8 (0.6) n = 5	0.6 (0.5)	0.4 (0.5) n = 4
ER	0.9 (1.5)	0.6 (0.7)	0.3 (0.5)

Table 3. Muscle Oxygenation Response across the Tests in Handcyclists, Mean (SD). Abbreviations: AU, arbitrary units;  $\Delta\text{SmO}_2\text{min}$ ,  $\text{SmO}_2$  minimal in change from baseline;  $\Delta\text{SmO}_2\text{avg}$ ,  $\text{SmO}_2$  mean in change from baseline;  $\Delta[\text{THb}]_{\text{avg}}$ , total hemoglobin mean in change from baseline;  $\Delta[\text{HHb}]_{\text{max}}$ , maximal deoxyhemoglobin in change from baseline; deoxy rate, deoxygenation rate; reoxy rate, reoxygenation rate. a Different from incremental ( $P < 0.05$ ). b Different from TT ( $P < 0.05$ ). c Different from BB ( $P < 0.05$ ). d Different from ER ( $P < 0.05$ ).

Peak deoxy, average muscle  $\text{O}_2$  saturation, reoxy rate, deoxy rate and maximal  $\text{O}_2$  extraction did not differ statistically among the muscles in the same test. TT average total hemoglobin significantly differed between the muscles ( $H(3) = 10.614$ , ES: 0.598,  $p = 0.014$ ). TT average total hemoglobin of the TB was lower than the BB (-200%,  $p = 0.005$ ) and ER (-308%,  $p = 0.039$ ), and TT total average hemoglobin of the AD was lower than the BB (-140%,  $p = 0.016$ ), the other muscles did not differ statistically.

For each muscle, peak deoxy, average muscle O<sub>2</sub> saturation and reoxy rate did not differ statistically across the tests. Wingate maximal O<sub>2</sub> extraction of the ER was lower than in the incremental test, (-37%, ES: 0.392 [0.0245, 0.5668], p = 0.031); Maximal O<sub>2</sub> extraction did not differ statistically across the tests for TB, AD and BB. Deoxy rate of the ER was faster in the Wingate than in the TT, (-88% (*H*(1) = 5.633, ES: 0.563, p = 0.018), although deoxy rate did not differ across the tests for TB, AD and BB. Average total hemoglobin did not differ statistically across the tests.

Figure 2 displays the changes in SmO<sub>2</sub>min (expressed in ΔSmO<sub>2</sub>min) across the tests and across the muscles in every athlete and Figure 3 displays the changes in maximal O<sub>2</sub> extraction (expressed in Δ[HHb]max) across the tests and across the muscles in every athlete.

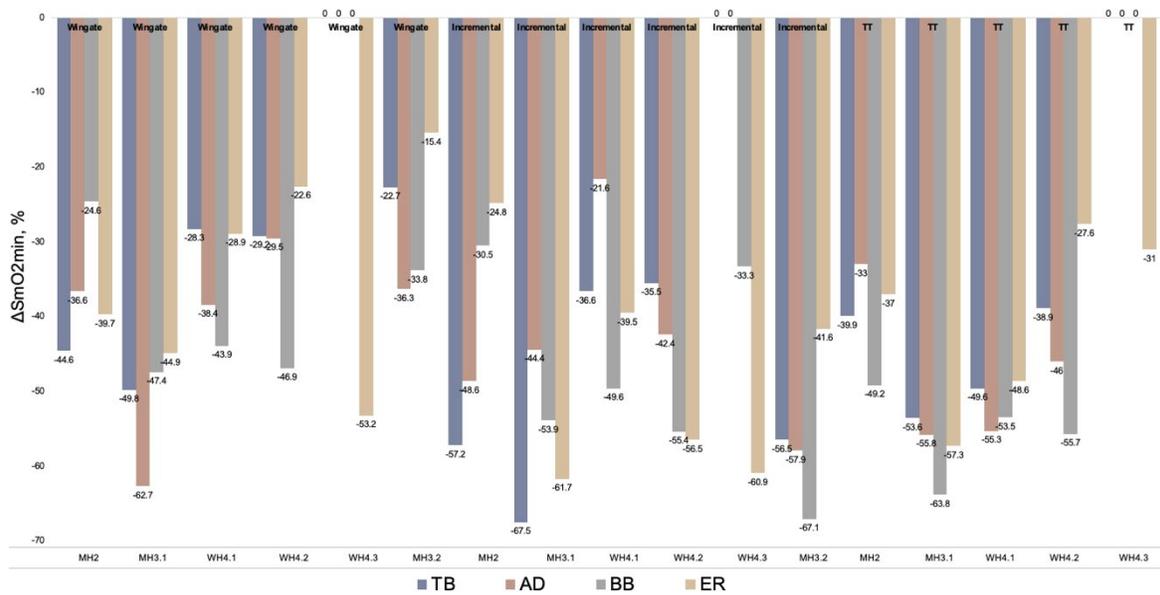


Figure 2. Changes in Maximal Deoxygenation Level (SmO<sub>2</sub>min) across Tests and Muscles in Handcyclists

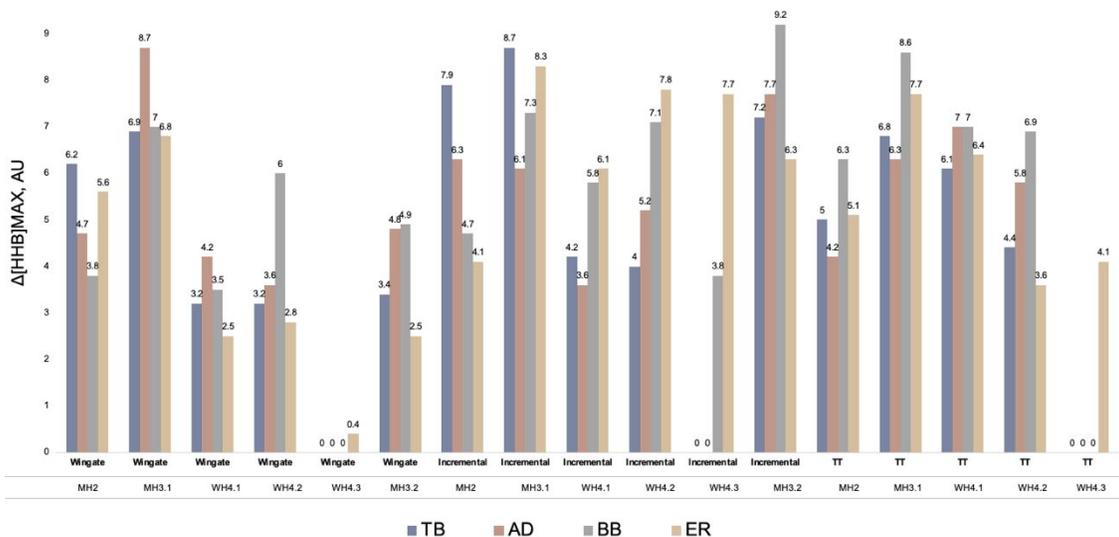


Figure 3. Changes in Maximal Muscle O<sub>2</sub> Extraction ([HHb]max) across Tests and Muscles in Handcyclists

## Wingate

Fatigue index was inversely related to  $SmO_2min$  in the TB (-.945 [-1.73, -0.38],  $P = .015$ ). Mean power (W) was related to maximal  $O_2$  extraction of the AD (1.000,  $P < .001$ ), to reoxygenation rate of the TB (.820 [-0.15, 0.99],  $P = .046$ ), of the AD (.916 [0.17, 1.57],  $P = .029$ ), of the ER (.829 [-0.13, 0.99],  $P = .042$ ) and of the average of the four muscles (.852 [0.13, 1.58],  $P = .031$ ), to deoxygenation rate of the AD (-.900 [-1.00, 0.17],  $P = .037$ ), of ER (-.867 [-1.56, -0.18],  $P = .025$ ), and of the average of the four muscles (-.921 [-1.46, -0.38],  $P = .009$ ). Mean power (W) was related to forearm girth (.832 [0.06, 1.60],  $P = .04$ ), to right prehension force (.879 [0.22, 1.54],  $P = .021$ ) and to left prehension force (.823 [0.033, 1.61],  $P = .044$ ). Mean power (W/kg) was related to reoxygenation rate of the AD (.884 [0.03, 1.72],  $P = .046$ ) and to the average of the four muscles (.860 [0.15, 1.57],  $P = .028$ ), as well as right prehension force (.957 [0.55, 1.36],  $P = .003$ ) and left prehension force (.860 [0.15, 1.57],  $P = .028$ ). Peak power (W) was related to maximal  $O_2$  extraction of the AD (.900 [-0.17, 1.00],  $P = .037$ ), to the deoxygenation rate of the ER (-.886 [-1.53, -0.24],  $P = .019$ ) and to the average of the four muscles (-.901 [-1.50, -0.30],  $P = .014$ ), to the reoxygenation rate of the AD (.948 [0.39, 1.62],  $P = .014$ ), ER (1.000,  $P < .001$ ) and to the average of the four muscles (.892 [0.26, 1.52],  $P = .017$ ). Peak power (W) was related to right prehension force (.870 [0.17, 1.55],  $P = .024$ ) and to forearm girth (.827 [0.05, 1.61],  $P = .042$ ). Peak power (W/kg) was related to upper arm girth (.829 [-0.13, 0.99],  $P = .042$ ), as well as to right prehension force (.943 [0.38, 1.00],  $P = .005$ ) and left prehension force (.886 [0.07, 0.99],  $P = .019$ ).

## Incremental Test

MAP (W) was related to maximal  $O_2$  extraction in the BB (.863 [0.16, 1.57],  $P = .027$ ), to upper arm girth (.965 [0.60, 1.33],  $P = .002$ ), to forearm girth (.947 [0.50, 1.39],  $P = .004$ ) and to  $VO_2max$  (L/min) (.928 [-0.53, 3.00],  $P = .023$ ). MAP (W/kg) was related to maximal blood lactate concentration (.829 [-0.13, 0.99],  $P = .042$ ) and to  $VO_2max$  (mL/kg/min) (.895 [0.08, 1.91],  $P = .04$ ).

## Time Trial

Mean power (W) was related to mean  $SmO_2min$  of the four muscles averaged (-.879 [-1.75, -0.01],  $P = .049$ ), to the deoxygenation rate of the AD (-.999 [-0.83, -0.63],  $P < .001$ ), of the BB (-.954 [-1.36, -0.03],  $P = .046$ ), of the ER (-.902 [-1.70, -0.11],  $P = .036$ ) and of the average of the four muscles (-.900 [-1.00, 0.17],  $P = .037$ ), to upper arm girth (.988 [0.71, 1.27],  $P = .002$ ), to forearm girth (.996 [0.83, 1.16],  $P < .001$ ), to the average total hemoglobin of the four muscles (.949 [0.15, 1.00],  $P = .014$ ) to  $VO_2peak$  (L/min) (.888 [0.03, 0.99],  $P = .044$ ) and to  $VO_2peak$  (mL/kg/min) (.889 [0.03, 0.99],  $P = .044$ ). Mean power (% MAP) was related to reoxygenation rate of the AD (.968 [0.18, 1.51],  $P = .032$ ), and to the level of experience in handcycling (.961 [0.45, 1.47],  $P = .009$ ). TT distance was related to the reoxygenation rate of the BB (-.985 [-0.98, -0.30],  $P = .015$ ).

Figure 4 illustrates the physiological demand of a 20-minute handcycling TT in minutes spent in the 3 intensity zones for the parameters examined: VO<sub>2</sub>, HR and SmO<sub>2</sub>. Overall, more time was spent in zone 3 for all variables, but more time was spent in zone 1 for ER SmO<sub>2</sub>.

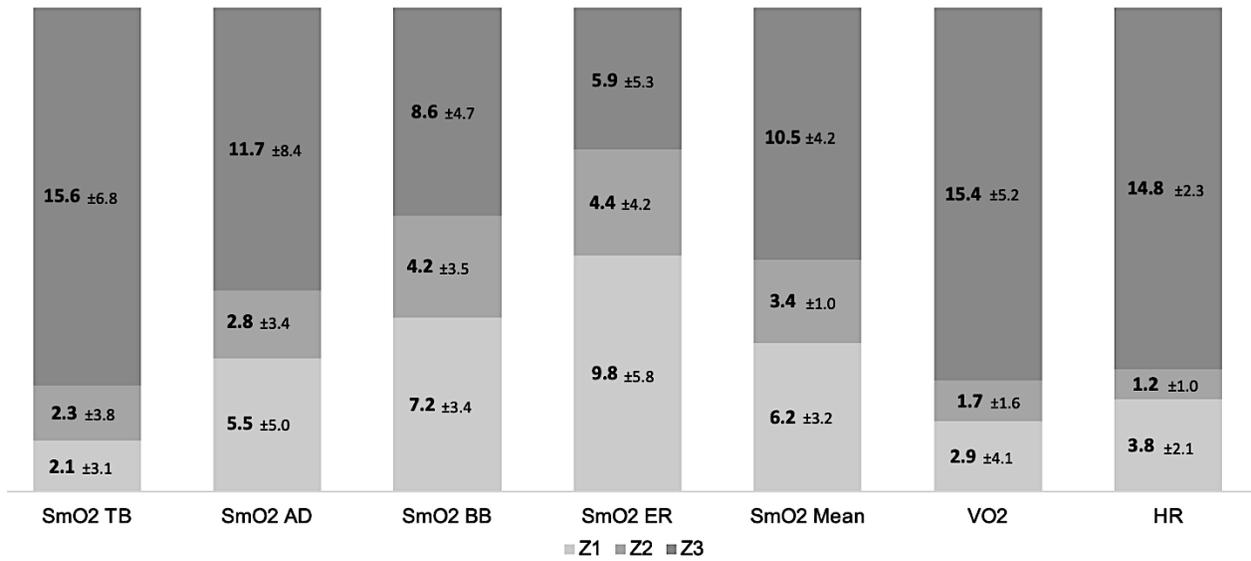


Figure 4. Physiological Response during a 20-minute Handcycling TT (Minutes in the 3 Intensity Zones), Mean ± SD

Figure 5 presents the evolution of  $SmO_2$  during the 20-minute TT, with the TT course (elevation) presented at the bottom. Figure 6 presents the evolution of HR in the TT and Figure 7 presents the evolution of  $VO_2$  in the TT. Figure 8 also presents the TT course at the bottom.

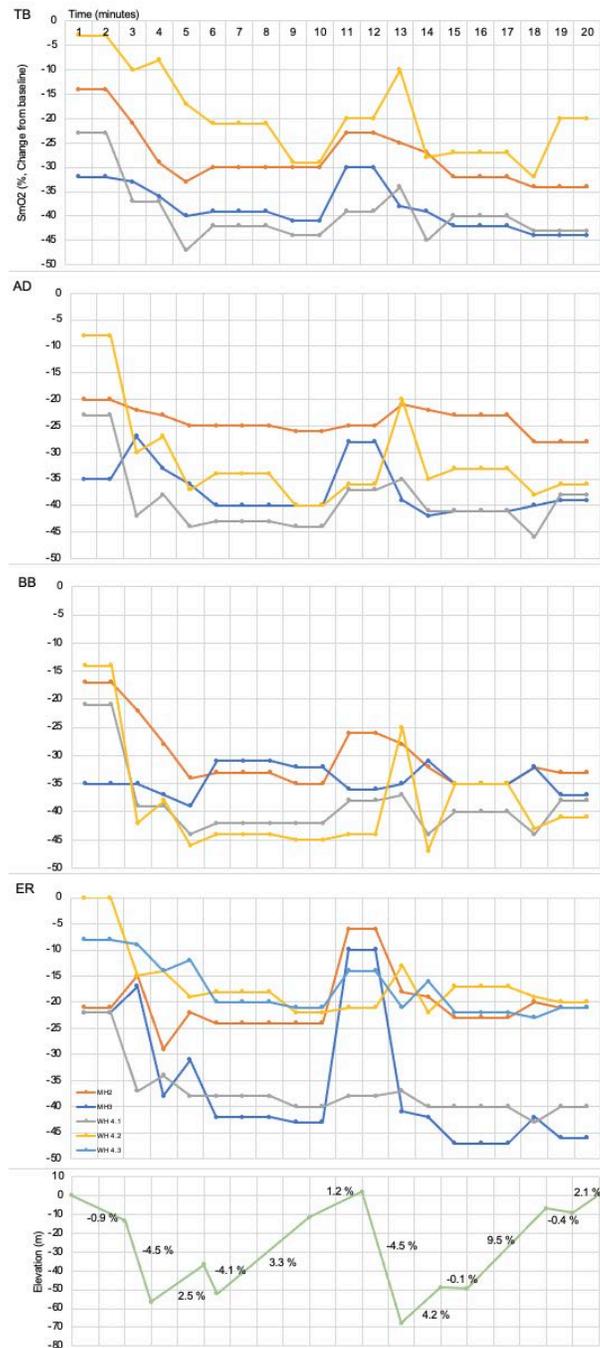


Figure 5. Evolution of  $SmO_2$  during a 20-minute Handcycling TT. Figure 5 presents the evolution of  $SmO_2$  (average of each TT segment) during the 20-minute TT, expressed in change from baseline  $SmO_2$  (%) in the four muscles.

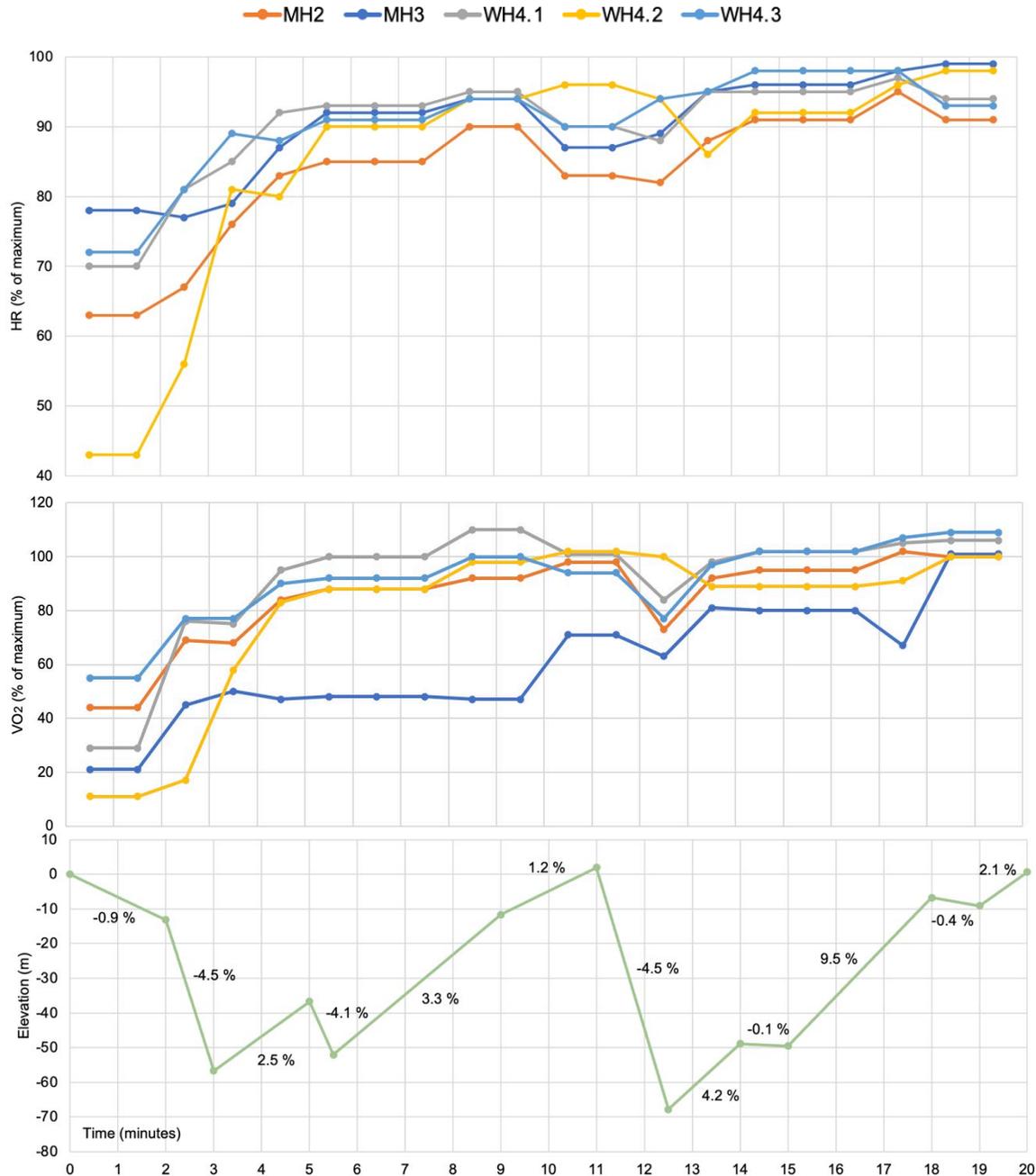


Figure 6 (top). Evolution of VO<sub>2</sub> during a 20-minute Handcycling TT. VO<sub>2</sub> is expressed in % of maximal VO<sub>2</sub>. Figure 7 (middle). Evolution of HR during a 20-minute Handcycling TT. HR is expressed in % of maximal HR. Figure 8 (bottom). TT Course.

## Discussion

The aim of the study was to assess muscle oxygenation changes during a 30-second arm cranking sprint (Wingate), a maximal incremental test and a 20-minute TT on an arm ergometer in well-trained recumbent handcyclists, and examined the associations between muscle oxygenation, VO<sub>2</sub>max, Qmax, power and TT distance. This is the first study to investigate the associations

between muscle oxygenation variables and performance in handcyclists using portable NIRS devices. Main results depicted these strong associations between the physiological and performance parameters examined:

- (1) High  $\text{VO}_2\text{max}$  and maximal blood lactate are associated to a high MAP
- (2) Large upper arm girths are associated to a high MAP, sprint peak PO and TT mean PO
- (3) Large forearm girths are associated to a high sprint peak and mean PO and TT mean PO
- (4) Strong prehension forces are associated to a high sprint peak and mean PO
- (5) High maximal BB  $\text{O}_2$  extraction is associated to a high MAP
- (6) High maximal AD  $\text{O}_2$  extraction is associated to a high sprint peak and mean PO
- (7) Low TB  $\text{SmO}_2\text{min}$  is associated to sprint lower resistance to fatigue
- (8) Low  $\text{SmO}_2\text{min}$  of muscles combined is associated to a high TT mean PO
- (9) Fast deoxygenation of muscles combined is associated to a high sprint peak and mean PO and TT mean PO
- (10) Fast reoxygenation of muscles combined is associated to a high sprint peak and mean PO
- (11) Fast reoxygenation of AD is associated to a high TT mean PO
- (12) Fast reoxygenation of BB is associated to greater TT distance
- (13) High total hemoglobin of the muscles combined is associated to a high TT mean PO
- (14) Superior experience level is associated to a high TT mean PO
- (15) Superior  $\text{VO}_2\text{peak}$  is associated to a high TT mean PO

Taken together, these results confirm the importance of central adaptations in handcycling but illustrate the contribution of peripheral adaptations to predict performance.

### **Central vs. peripheral contribution to performance**

Previous work depicted that high muscle  $\text{O}_2$  extraction contributes to a remarkable  $\text{VO}_2\text{max}$  in well-trained endurance athletes, as measured by arterial and femoral venous catheters (Skattebo et al., 2020).  $\text{O}_2$  extraction factor has been associated with peripheral adaptations such as, high muscle capillary density, elevated erythrocytes capillary mean transit time, enhanced  $\text{HbO}_2$  off-loading, thus greater ability of muscles to extract  $\text{O}_2$  (Larsen et al., 2020, Calbet et al., 2005 and Cardinale et al., 2019). In fact, our study demonstrates strong associations between maximal upper body  $\text{O}_2$  extraction and handcycling performance, especially of BB in the incremental test and AD in the sprint, which suggest enhanced peripheral characteristics in these specific muscles in elite handcyclists.

$\text{SmO}_2$  is the proportion of muscle  $\text{O}_2$  that is saturated with hemoglobin, and it represents the balance between  $\text{O}_2$  delivery and extraction by the muscle. In our study, muscle's levels of maximal deoxygenation ( $\text{SmO}_2\text{min}$ ) were similar in all 3 tests (~44% from baseline), suggesting there is no difference in maximal deoxygenation among these muscles from a sprint to longer efforts, at least in this group of athletes.

$\text{VO}_2\text{max}$  was similar, although a little lower than values reported in Stephenson's et al. review (2020) for H3-5 elite handcyclists (> 3.0 L/min), that could be attributed to the lack of available information regarding female handcyclists, whereas 3 females were included in our study with 1 training at a lower level than the other athletes. Similar  $\text{VO}_2$  was found for the H2 elite handcyclist included in this study than the values reported (~ 2.4 L/min) (Fischer, Figueiredo et Ardigo, 2015). To our knowledge, no published study ever examined  $\text{Qmax}$  and  $\text{SVmax}$  in elite handcyclists. We were able to measure  $\text{Qmax}$  and  $\text{SVmax}$  in 3 athletes and maximal (a-v) $\text{O}_2$  diff in 2 athletes.

Q<sub>max</sub> reported in our study were similar to the ones reported in highly trained kayakers measured with the Physioflow (23.9 L/min for men and 21.3 L/min for women), although these athletes have much higher VO<sub>2</sub>max (4.5 L/min for men and 3.2 L/min for women) reported by Paquette, Bieuzen & Billaut (2019). Even though both sports are upper body dominant, handcyclists have much less active muscle mass to contribute to a high VO<sub>2</sub>max due to physical impairment. Athletes in our study have lower (a-v)O<sub>2</sub> diff values than the ones reported in kayakers (19.2 mL/dL for men and 14.8 mL/dL for women), which would explain the major differences in VO<sub>2</sub>max despite very similar Q<sub>max</sub> measured (Paquette, Bieuzen & Billaut, 2018). Skattebo et al.'s review (2020) found high variability in (a-v)O<sub>2</sub> diff calculated on endurance athletes for a given VO<sub>2</sub>max, potentially explaining the differences with canoe-kayakers despite the very low sample size for this variable in our study. Authors also reported lower values of (a-v)O<sub>2</sub> diff for athletes with the highest VO<sub>2</sub>max and they explain that (a-v)O<sub>2</sub> diff is not only determined by the ability of the peripheries to extract O<sub>2</sub> but also by the arterial O<sub>2</sub> content during maximal exercise (Skattebo et al., 2019). Since plasma volume expands with endurance exercise, hemodilution occurs, Q<sub>max</sub> is increased, lowering arterial O<sub>2</sub> carrying capacity of the blood and (a-v)O<sub>2</sub> diff, complicating the finding in our study. Overall, RER, HR, maximal blood lactate and PO measures in handcyclists during the incremental test were similar to the ones reported previously (Strangier et al., 2019; Abel et al., 2006; Lovell et al., 2012; De Groot et al., 2014; Fisher et al., 2015).

An increase in Q<sub>max</sub> is generally considered the most important factor contributing to VO<sub>2</sub>max in endurance (Basset & Howley, 2000), and SV<sub>max</sub> is considered the most important factor contributing to an increase in Q (Basset & Howley, 2000 and Joyer & Coyle, 2008). Thus, lower SV<sub>max</sub> contributing to lower Q<sub>max</sub> and VO<sub>2</sub>max would be expected in handcyclists considering their physical impairment. SV<sub>max</sub> values reported in our study were similar to the ones reported in trained kayakers (129 mL/beat for men and 114 mL/beat for women), although a little high for one male athlete in our study (164.4 mL/beat), that could be explained by a lower maximal heart rate recorded for him (177 bpm), potentially due to fibers denervation caused by paraplegia (Paquette, Bieuzen & Billaut, 2018). Unfortunately, the VO<sub>2</sub>max data of that same athlete could not be retrieved, complicating the interpretation of this finding. Surprisingly, in our study, a lower SV<sub>max</sub> was very strongly associated to a higher MAP, maybe due to the small sample size for this variable (n=3) that could have conflicted the results. As expected, the Q<sub>max</sub> recovered from a female handcyclist in our study was lower than the Q<sub>max</sub> of female triathletes, which is expected again due to physical impairment (Mollard et al., 2007), however, Q<sub>max</sub> and max (a-v)O<sub>2</sub> diff of male handcyclists in our study were similar to the ones reported in male triathletes (Le Meur et al., 2014).

Overall, peripheral cardiovascular contributions to handcycling performance are confirmed. In terms of the central adaptations of VO<sub>2</sub>max, there seems to be some contribution to handcycling performance as Q<sub>max</sub> and SV<sub>max</sub> were strongly associated to MAP in the maximal incremental test but non-significantly (see Appendix for detailed results). For the TT, Q<sub>peak</sub> was non-significantly strongly associated to mean power.

### **Muscle recruitment vs. intensity**

Since SmO<sub>2</sub> parameters are associated to electromyography (EMG), and training intensity influences muscle recruitment and activity, changes in these parameters may differ between muscles, depending on the training intensity and type (Miura et al., 2000). This could explain some of the changes in muscle oxygenation among efforts found in our study. Previous work on EMG of handcycling revealed differences in muscle recruitment with increasing arm cycling intensity

(Quittman et al. 2019). Authors found an increased activity for the BB, posterior and anterior fibers of the deltoids, TB, ER and rectus abdominis muscles with an increase in workload (Quittman et al., 2019). The BB muscle group is the major force producer in the primary propulsion phase (pull) of handcycling and AD is highly activated in the pull and push phases (Quittman et al., 2020). These findings could explain the higher O<sub>2</sub> extraction capacity found in the BB during the maximal incremental test and of the AD during the sprint in our study. We did not find significant interaction of TB or ER O<sub>2</sub> extraction and performance, and TB low level of oxygenation (peak deoxygenation) was inversely associated to sprint performance (resistance to fatigue), showing that low oxygenation might be detrimental to sprint performance. Overall, all evaluated muscles combined ability to sustain low oxygenation seemed to be highly important to perform a good TT. Thus, the ability to highly desaturate, and to extensively use oxygen seems to be a key performance predictor, especially in long (aerobic) handcycling efforts.

The association between BB O<sub>2</sub> extraction and performance in the incremental test could suggest greater biceps brachii muscle fiber recruitment and activity in this test, although maximal O<sub>2</sub> extraction of the four muscles averaged and of each independent muscle were also correlated (non-significantly) to sprint performance. For the TT, most performance variables were correlated (non-significantly) to the muscles averaged maximal O<sub>2</sub> extraction, and BB maximal O<sub>2</sub> extraction was also correlated to mean power (non-significantly), making it difficult to assume greater biceps recruitment in the incremental test only, and greater peripheral adaptations in this muscle only. BB maximal O<sub>2</sub> extraction levels in our study were similar to the ones measured in highly trained canoe-kayakers with the Moxy monitors during an ergometer incremental test and on-water TTs (Paquette, Bieuzen & Billaut, 2018). With the strong association of AD maximal O<sub>2</sub> extraction and sprint performance found in our study, it could suggest greater anterior deltoid muscle fiber recruitment and activity in the sprint. However, TB, AD, and the muscles averaged maximal O<sub>2</sub> extraction were also correlated to MAP (non-significantly). In the TT, AD maximal O<sub>2</sub> extraction was non-significantly correlated to 2 of the 4 performance parameters examined, making it difficult to conclude greater recruitment and enhanced peripheral characteristics in the anterior deltoid in the sprint.

Maximal ER O<sub>2</sub> extraction was 37% higher in the incremental test than in the Wingate, and since exercise intensity impacts muscle recruitment, it could suggest that a maximal aerobic effort may induce superior O<sub>2</sub> extraction due to greater muscle recruitment than during a sprint, at least in the forearm extensors in this group of athletes. According to Quittmann et al. (2019), there is an increase in muscle activity as measured by EMG in the extensor carpi radialis muscle with an increase in handcycling intensity in able-bodied triathletes. Although it is difficult to compare able-bodied athletes to elite handcyclists, it supports our finding that a longer maximal effort may induce O<sub>2</sub> extraction due to greater muscle recruitment as compared to a short sprint. However, for TB, AD and BB, the capacity to extract O<sub>2</sub> did not differ across the 3 efforts, complicating the interpretation of this finding.

Total hemoglobin level ([THb]) serves as an indicator of local blood volume, and it did not differ between muscles from the sprint to the incremental test, suggesting there is no difference in total blood volume among these muscles from a sprint to a maximal incremental test at least in this group of athletes. For the TT, TB total hemoglobin was lower than the BB (-200%) and the ER (-308%), and AD total hemoglobin was lower than the BB (-140%), suggesting that TB and AD may have lower levels of blood volume during a 20-minute-long TT, at least in this group of handcyclists. This could imply a greater blood distribution to the dominant muscles during a handcycling TT, the BB and ER, suggesting greater biceps brachii and extensor carpi radialis

muscles recruitment and activity, perhaps due to fatigue or weakness of other main muscles such as the elbow extensors. In contrast to our findings, Quittmann and colleagues (2019) found an increased activity for the biceps brachii, triceps brachii and extensor carpi radialis muscles with an increase in workload in able-bodied athletes during handcycling. Even though no significant differences in oxygenation levels and in O<sub>2</sub> extraction were found among muscles, a study found significantly higher BB O<sub>2</sub> extraction as compared to AD, and lower BB tissue saturation index as compared to AD, TB, and forearm muscle (brachioradialis) during a maximal incremental arm cranking test in healthy young men (Lusina et al., 2008). Although it is difficult to compare elite handcyclists to able-body sedentary participants, authors suggested that BB was the main muscle involved in arm cranking propulsion with supinated grip, reinforcing the importance of this key muscle in handcycling (Lusina et al., 2008). However, in our study athletes cycled in neutral grip, slightly changing muscle involvement.

### **Deoxygenation rate and performance**

Rapid deoxygenation rate was highly associated to sprint and TT performance in our study. Nioka et al. (1998) suggested that greater muscle capillary density and O<sub>2</sub> extraction may result in ability to deoxygenate faster and more extensively in athletes, thus an important peripheral adaptation for performance. Faster negative SmO<sub>2</sub> slope is generally attributed to an acceleration in muscle O<sub>2</sub> demand and energy consumption, that is greater than the acceleration of O<sub>2</sub> supply (Ferrari, Muthalib & Quaresima, 2011). ER deoxygenated 88% faster in the Wingate than in the TT, suggesting that for this muscle group, faster deoxygenation occurs in an all-out sprint as compared to a 20-minute effort, due to a disproportional increase in O<sub>2</sub> demand vs. supply to the muscle, and a shift to anaerobic energy needs. Even though deoxygenation rate did not statistically differ across the efforts for TB, AD and BB, the deoxygenation rates are much faster in the Wingate than in the TT in our study. In Varsity sprinters, a 30-second leg cycling sprint triggered a faster muscle deoxygenation in the vastus lateralis, as compared to an incremental test starting at low intensity and where deoxygenation was delayed (Nioka et al., 1998). The rapid deoxygenation was attributed to immediate energy cost and muscle O<sub>2</sub> demand in the sprint (Nioka et al., 1998). This response to immediate oxygen needs during an arm cranking sprint could also be true for other muscles and could be an important aspect of sprint training. Muscles deoxygenation rates were similar in the sprint and in the 20-minute TT (~1.3%/sec) suggesting there is no difference in deoxygenation rates among these 4 muscles from a handcycling sprint to a TT, at least in this group of athletes.

### **Reoxygenation rate and performance**

Rapid reoxygenation rate was highly associated to sprint and TT performance in our study. The ability of muscles to reoxygenate quickly is known to be an important physiological adaptation and had been previously associated to better recovery (Buchheit & Ufland, 2011). The metabolic-related mechanisms involved in faster recovery (reduce fatigue development) can be the phosphocreatine recovery, systemic oxygen availability, muscle oxygenation and H<sup>+</sup> buffering (Bogdanis et al., 1996, Balsom et al., 1994, Buchheit et al., 2009). A faster muscle reoxygenation post-effort has also been associated to improved muscle oxidative capacity (Puente-Maestu et al., 2003), muscle blood flow and capillarization (Kime et al., 2003). In fact, a study measured the distance decrements between 15-second shuttle running sprints in athletic males and found faster reoxygenation assessed by NIRS between bouts in athletes who recovered well, as muscles reached baseline oxygenation quickly (Buchheit & Ufland, 2011). In our study, muscles reoxygenated at similar rates in all 3 tests (~0.6%/sec) suggesting there is no difference in

reoxygenation pattern among these 4 muscles, thus no change in muscle recovery capacity from a handcycling sprint to longer efforts, at least in this group of athletes.

### **Peripheral adaptations and training**

Paquette, Bieuzen & Billaut (2020) demonstrated that sprint and high intensity interval training can significantly induce peripheral adaptations measured by portable NIRS devices in highly trained canoe-kayakers. The same authors (2021) discovered significant improvements in maximal deoxygenation in various muscles in elite kayakers after nine sessions of high intensity interval training (HIIT) and sprint interval training programs, and a greater improvement in 200m, 500m and 1000m kayak performances after the HIIT sessions. These adaptations could also be seen in handcyclists after a specific HIIT training block, and future studies should consider trying this method.

### **Grip strength and arm girths**

Strong associations were found between grip strength and Wingate peak and mean PO. Grip strength was also positively correlated to high peak PO in amputee football players who performed a 30-second arm cranking Wingate (Nowak et al., 2021). This association suggests that strength training may be an important contributor of high PO during handcycling sprints also. Previous work demonstrated the importance of strength training with improvements in MAP, body composition, mechanical efficiency, and 30 km TT performance after an 8-week strength and endurance program in handcyclists (Nevin, 2017). Moreover, the study observed a small increase in arm girth after the 8-week strength and endurance program, which could potentially contribute to the improvements in MAP explained by muscle growth (Nevin, 2017). In our study, arm girths were also strongly associated to handcycling performance.

### **Physiological Response of a Handcycling TT**

We observed the evolution of 3 physiological parameters during the laboratory 20-minute TT:  $VO_2$ , HR and  $SmO_2$ . On average, athletes spent more time in zone 3 for all variables, representing efforts with intensities above the 2<sup>nd</sup> lactate threshold, except for one variable,  $SmO_2$  of ER that spent more time in the first intensity zone (below the 1<sup>st</sup> lactate threshold). For one athlete, only a slight desaturation was seen from stage to stage for ER  $SmO_2$  in the incremental test, complicating the computation of  $SmO_2$  zones. For this same athlete, the two  $SmO_2$  thresholds are very close to each other, and it resulted in a very large time spent in zone 1 for the TT. Overall, it is expected to observe more time spent in the high intensity zone in a short TT of 20 minutes, with climbs in 6 of the 12 segments, starting as early as by the 3<sup>rd</sup> minute, which could have elevated  $VO_2$  and HR early during the test. Athlete's rating of perceived exertion was 8 or 8.5/10, confirming the difficulty of the course. One study examined the demand of a 90 km cross-country skiing TT where  $SmO_2$  of the TB spent over 70% of the course in zone 3 (defined as  $\geq 90\% VO_{2max}$ ), while HR spent over 80% in zone 1 ( $\leq 80\% VO_{2max}$ ) (Stöggl & Born, 2021). This finding suggests that in very long TTs (over 5 hours), TB  $O_2$  saturation can be in the highest intensity zone for the majority of the duration of the effort, in contrast to HR that can probably only stay in the high intensity zone during short TTs (~20 minutes), and this could potentially be seen in other muscles. However, this comparison should be carefully examined in athletes specialized in the same sport and in different muscles.

## Limitations

This study included a highly heterogeneous group of handcycling athletes (N=6) including three females and three males classified in 3 different categories (H2, H3 or H4). Thus, it was difficult to provide differences between sub-groups with such a small and heterogeneous sample size. Future studies should include larger samples of trained handcyclists, preferably from the same handcycling class (H1-5) and gender, and conduct field testing, to make thorough training recommendations, as laboratory settings may not represent true racing conditions (e.g., environmental conditions, tactics, position in the handcycle, driving skills, psychological pressure during competition, etc.).

Even though the Moxy monitor from Fortiori design has a similar reliability to other traditional NIRS devices, and was previously validated in sports science (Feldmann, Schmitz & Erlacher, 2019), previous research has identified that the reliability of the SmO<sub>2</sub> measurements of the Moxy monitor decreases as the intensity of exercise increases, possibly due to greater movement artefact and tissue ischemia (Crum et al., 2017). In addition, skinfold thickness can be a limiting factor while using NIRS monitors if it exceeds penetration depth of the emitted photons, half the distance between the emitter and the detector as the muscular effects will be blunted (Stöcker et al., 2016). This aspect can be quite limiting if the study participants recruited have a higher fat mass level and a high BMI. Thicker tissue may alter the scattering of light and inhibit the return of light to the sensor (Crum et al., 2017). Therefore, skinfold thickness of the four muscle sites were assessed prior to testing and any site surpassing 15 mm of skin thickness was excluded (Feldmann, Schmitz & Erlacher, 2019). The recommended penetration depth threshold identified by previous research is 15 mm maximum (adipose tissue thickness < 7.5 mm) and any SmO<sub>2</sub> values obtained over this threshold must be considered suspect (Feldmann, Schmitz & Erlacher, 2019). The Moxy monitor has a small source-detector separation (2.5 cm), resulting in lower tissue penetration into the muscle in subjects with higher adipose tissue thickness (McMannus, Collison & Cooper, 2018).

## Practical applications and perspectives

This project provided a complete physiological profile of highly trained handcyclists including various parameters that were never assessed before with this specific group of athletes such as muscle oxygenation kinetics and Q. By providing these various physiological markers, this study was the first to measure peripheral adaptations (changes in muscle O<sub>2</sub> saturation and in O<sub>2</sub> extraction) and central adaptation (VO<sub>2</sub>max, Q and SV) in handcyclists. It was possible to confirm the hypothesis that muscle oxygenation capacity is important in an upper body sport like handcycling. Thus, this project clarifies the physiological needs of the sport, and reinforces the utility of portable NIRS monitors in para-cycling that can be used to optimize training and testing protocols, by analysing the evolution of muscle oxygenation characteristics before, during and after training blocks. Results of our study could also be used for further talent identification in development athletes, with the examination of superior peripheral abilities at younger ages (e.g., ability of muscles to extract oxygen, and rapid muscle deoxygenation and reoxygenation patterns, that are linked to muscle recruitment efficiency and superior oxidative capacity) possibly helping team recruitment.

## Conclusion

To summarize, our study suggest that peripheral adaptations are key predictors of handcycling performance. Consequently, these should be considered in training plans of elite handcyclists. Although this research project is carried out in para-cycling, results could be generalizable to other Paralympic sports, where efforts are generated by the upper limbs, such as para canoe-kayak, para-swimming, or wheelchair sports where athletes have lower limb restrictions.

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## References

1. Abel, T., Schneider, S., Platen, P. & Strüder, H. K. (2006). Performance diagnostics in handbiking during competition. *Spinal Cord*, 44:211-216. <https://doi.org/10.1038/sj.sc.3101845>
2. Akoglu H. (2018). User's guide to correlation coefficients. *Turkish journal of emergency medicine*, 18(3), 91–93. <https://doi.org/10.1016/j.tjem.2018.08.001>
3. Balsom, P. D., Ekblom, B., & Sjodin, B. (1994). Enhanced oxygen availability during high intensity intermittent exercise decreases anaerobic metabolite concentrations in blood. *Acta physiologica Scandinavica*, 150(4), 455–456. <https://doi.org/10.1111/j.1748-1716.1994.tb09711.x>
4. Bassett, D. R. Jr. & Howley, E. T. (2000). Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Med Sci Sports Exerc*, 32(1):70-84. doi: 10.1097/00005768-200001000-00012. PMID: 10647532
5. Bogdanis, G. C., Nevill, M. E., Boobis, L. H., & Lakomy, H. K. (1996). Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. *Journal of applied physiology (Bethesda, Md. : 1985)*, 80(3), 876–884. <https://doi.org/10.1152/jappl.1996.80.3.876>
6. Bruce R. M. (2017). The control of ventilation during exercise: a lesson in critical thinking. *Advances in physiology education*, 41(4), 539–547. <https://doi-org.lib-ezproxy.concordia.ca/10.1152/advan.00086.2017>
7. Buchheit, M., Cormie, P., Abbiss, C. R., Ahmaidi, S., Nosaka, K. K., & Laursen, P. B. (2009). Muscle deoxygenation during repeated sprint running: Effect of active vs. passive recovery. *International journal of sports medicine*, 30(6), 418–425. <https://doi.org/10.1055/s-0028-1105933>
8. Buchheit, M., Abbiss, C. R., Peiffer, J. J., & Laursen, P. B. (2012). Performance and physiological responses during a sprint interval training session: relationships with muscle oxygenation and pulmonary oxygen uptake kinetics. *European journal of applied physiology*, 112(2), 767–779. <https://doi-org.lib-ezproxy.concordia.ca/10.1007/s00421-011-2021-1>
9. Calbet, J. A., Holmberg, H. C., Rosdahl, H., van Hall, G., Jensen-Urstad, M., & Saltin, B. (2005). Why do arms extract less oxygen than legs during exercise?. *American journal of physiology. Regulatory, integrative and comparative physiology*, 289(5), R1448–R1458. <https://doi.org/10.1152/ajpregu.00824.2004>

10. Cardinale, D. A., Larsen, F. J., Jensen-Urstad, M., Rullman, E., Søndergaard, H., Morales-Alamo, D., Ekblom, B., Calbet, J. A. L., & Boushel, R. (2019). Muscle mass and inspired oxygen influence oxygen extraction at maximal exercise: Role of mitochondrial oxygen affinity. *Acta physiologica (Oxford, England)*, 225(1), e13110. <https://doi.org/10.1111/apha.13110>
11. Crenshaw, A. G., Elcadi, G. H., Hellstrom, F. & Mathiassen, S. E. (2011). Reliability of near-infrared spectroscopy for measuring forearm and shoulder oxygenation in healthy males and females. *Eur J Appl Physiol*;112(7):2703-15. doi: 10.1007/s00421-011-2244-1. Epub 2011 Nov 23. PMID: 22109701
12. Crum, E. M., O'Connor, W. J., Van Loo, L., Valckx, M. & Stannard, S. R. (2017). Validity and reliability of the Moxy oxygen monitor during incremental cycling exercise. *Eur J Sport Sci.*; 17(8):1037-1043. doi: 10.1080/17461391.2017.1330899. Epub 2017 May 30. PMID: 28557670
13. De Groot, S., Postma, K., Vliet, L. V., Timmermans, R. & Valent, L. J. M. (2014). Mountain time trial in handcycling: exercise intensity and predictors of race time in people with spinal cord injury. *International Spinal Cord Society*, 52:455-461. doi:10.1038/sc.2014.58
14. Faupin A., Gorce P., Watelain E., Meyer C. & Thevenon A. (2010). A biomechanical analysis of handcycling: a case study. *J Appl Biomech*, (2):240-5. doi: 10.1123/jab.26.2.240. PMID: 20498497
15. Feldmann, A., Schmitz, R., Erlacher, D. (2019). Near-infrared spectroscopy-derived muscle oxygen saturation on a 0% to 100% scale: reliability and validity of the Moxy Monitor. *J Biomed Opt.*, 24(11):1-11. doi: 10.1117/1.JBO.24.11.115001. PMID: 31741352; PMCID: PMC7003144
16. Ferrari, M., Muthalib, M. & Quaresima, V. (2011). The use of near-infrared spectroscopy in understanding skeletal muscle physiology: recent developments. *Philos Trans A Math Phys Eng Sci*, 369(1955):4577-90. doi: 10.1098/rsta.2011.0230. PMID: 22006907
17. Fisher, G., Figueiredo, P. & Ardigò, L. P. (2015). Physiological Performance Determinants of a 22-km Handbiking Time Trial. *International Journal of Sports Physiology and Performance*, 10(8): 965-971. <http://dx.doi.org/10.1123/ijsp.2014-0429>
18. Kime, R., Karlsen, T., Nioka, S., Lech, G., Madsen, Ø., Sæterdal, R., Im, J., Chance, B., & Stray-Gundersen, J. (2003). Discrepancy between cardiorespiratory system and skeletal muscle in elite cyclists after hypoxic training. *Dynamic medicine : DM*, 2(1), 4. <https://doi.org/10.1186/1476-5918-2-4>
19. Kounalakis, S. N., Bayios, I. A., Koskolou, M. D., & Geladas, N. D. (2008). Anaerobic Capacity of the Upper Arms in Top-Level Team Handball Players, *International Journal of Sports Physiology and Performance*, 3(3), 251-261. doi: 10.1123/ijsp.3.3.251
20. Larsen, F. J., Schiffer, T. A., Zinner, C., Willis, S. J., Morales-Alamo, D., Calbet, J. A. L., Boushel, R., & Holmberg, H. C. (2020). Mitochondrial oxygen affinity increases after sprint interval training and is related to the improvement in peak oxygen uptake. *Acta physiologica (Oxford, England)*, 229(3), e13463. <https://doi.org/10.1111/apha.13463>
21. Le Meur, Y., Louis, J., Aubry, A., Guéron, J., Pichon, A., Schaal, K., Corcuff, J. B., Hatem, S. N., Isnard, R., & Hausswirth, C. (2014). Maximal exercise limitation in functionally overreached triathletes: role of cardiac adrenergic stimulation. *Journal of applied physiology (Bethesda, Md. : 1985)*, 117(3), 214–222. <https://doi-org.lib-ezproxy.concordia.ca/10.1152/jappphysiol.00191.2014>
22. Lepêtre, P-M., Weissland, T., Slawinski, J. & Lopes, P. (2012). Para-cycling performance was rather limited by physiological than functional factors. *Frontiers in Physiology*, 3(327). <https://doi.org/10.3389/fphys.2012.00327>

23. Lovell D., Shields D., Beck B., Cuneo R. & McLellan C. (2012). The aerobic performance of trained and untrained handcyclists with spinal cord injury. *Eur J Appl Physiol.*, 112(9):3431-7. doi: 10.1007/s00421-012-2324-x. Epub 2012 Jan 26. PMID: 22278391
24. Lusina, S. J., Warburton, D. E., Hatfield, N. G., & Sheel, A. W. (2008). Muscle deoxygenation of upper-limb muscles during progressive arm-cranking exercise. *Applied physiology, nutrition, and metabolism = Physiologie appliquee, nutrition et metabolisme*, 33(2), 231–238. <https://doi-org.lib-ezproxy.concordia.ca/10.1139/h07-156>
25. McManus, C. J., Collison, J., & Cooper, C. E. (2018). Performance comparison of the MOXY and PortaMon near-infrared spectroscopy muscle oximeters at rest and during exercise. *Journal of biomedical optics*, 23(1), 1–14. <https://doi.org/10.1117/1.JBO.23.1.015007>
26. Miura, H., Araki, H., Matoba, H., & Kitagawa, K. (2000). Relationship among oxygenation, myoelectric activity, and lactic acid accumulation in vastus lateralis muscle during exercise with constant work rate. *International journal of sports medicine*, 21(3), 180–184. <https://doi.org/10.1055/s-2000-301>
27. Mollard, P., Woorons, X., Letournel, M., Lamberto, C., Favret, F., Pichon, A., Beaudry, M., & Richalet, J. P. (2007). Determinant factors of the decrease in aerobic performance in moderate acute hypoxia in women endurance athletes. *Respiratory physiology & neurobiology*, 159(2), 178–186. <https://doi-org.lib-ezproxy.concordia.ca/10.1016/j.resp.2007.06.012>
28. Muchaxo, R. A., de Groot, S., van der Woude, L. V., Janssen, T. J., & Nooijen, C. (2020). Do Handcycling Time-Trial Velocities Achieved by Para-Cycling Athletes Vary Across Handcycling Classes? *Adapted Physical Activity Quarterly*, 37(4), 461-480. Retrieved Feb 1, 2021, from <https://journals.humankinetics.com/view/journals/apaq/37/4/article-p461.xml>
29. Mygind E. (1995). Fibre characteristics and enzyme levels of arm and leg muscles in elite cross-country skiers. *Scandinavian journal of medicine & science in sports*, 5(2), 76–80. <https://doi.org/10.1111/j.1600-0838.1995.tb00016.x>
30. Nevin, J., Smith, P., Waldron, M., Patterson, S., Price, M., Hunt, A. & Blagrove, R. (2018). Efficacy of an 8-Week Concurrent Strength and Endurance Training program on Hand Cycling Performance. *J Strength Cond Res.* 32(7):1861-1868. doi: 10.1519/JSC.0000000000002569. PMID: 29561384
31. Nioka, S., Moser, D., Lech, G., Evengelisti, M., Verde, T., Chance, B., & Kuno, S. (1998). Muscle deoxygenation in aerobic and anaerobic exercise. *Advances in experimental medicine and biology*, 454, 63–70. [https://doi-org.lib-ezproxy.concordia.ca/10.1007/978-1-4615-4863-8\\_8](https://doi-org.lib-ezproxy.concordia.ca/10.1007/978-1-4615-4863-8_8)
32. Norton, K. (2018). Standards for Anthropometry Assessment. *Kinanthropometry and Exercise Physiology*. London. <https://doi.org/10.4324/9781315385662>
33. Nowak, A. M., Molik, B., Kosmol, A., Szczepaniak, M., & Marszałek, J. (2021). Application of the arm-cranking 30-second Wingate Anaerobic Test (the WAnT) to assess power in amputee football players. *Acta of bioengineering and biomechanics*, 23(3), 13–23. PMID: 34978315
34. Paquette, M., Bieuzen & F., Billaut, F. (2018). Muscle Oxygenation Rather Than VO<sub>2</sub>max as a Strong Predictor of Performance in Sprint Canoe-Kayak. *Int J Sports Physiol Perform*, 1-9. doi: 10.1123/ijsp.2018-0077. Epub ahead of print. PMID: 29745773
35. Paquette, M., Bieuzen, F., & Billaut, F. (2019). Sustained Muscle Deoxygenation vs. Sustained High VO<sub>2</sub> During High-Intensity Interval Training in Sprint Canoe-Kayak. *Frontiers in sports and active living*, 1, 6. <https://doi-org.lib-ezproxy.concordia.ca/10.3389/fspor.2019.00006>

36. Paquette, M., Bieuzen, F., & Billaut, F. (2020). Effect of a 3-Weeks Training Camp on Muscle Oxygenation,  $\dot{V}O_2$  and Performance in Elite Sprint Kayakers. *Frontiers in Sports and Active Living*, 2, 47. <https://doi.org/10.3389/fspor.2020.00047>
37. Paquette, M., Bieuzen, F., & Billaut, F. (2021). The effect of HIIT vs. SIT on muscle oxygenation in trained sprint kayakers. *European journal of applied physiology*, 121(10), 2743–2759. <https://doi-org.lib-ezproxy.concordia.ca/10.1007/s00421-021-04743-z>
38. Puente-Maestu, L., Tena, T., Trascasa, C., Pérez-Parra, J., Godoy, R., García, M. J., & Stringer, W. W. (2003). Training improves muscle oxidative capacity and oxygenation recovery kinetics in patients with chronic obstructive pulmonary disease. *European journal of applied physiology*, 88(6), 580–587. <https://doi.org/10.1007/s00421-002-0743-9>
39. Quittmann, O. J., Abel, T., Albracht, K. & Strüder, H. K. (2019). Reliability of muscular activation patterns and their alterations during incremental handcycling in able-bodied participants, *Sports Biomechanics*, DOI: 10.1080/14763141.2019.1593496
40. Quittmann, O. J., Abel, T., Albracht, K., Meskemper, J., Foitschik, T., & Strüder, H. K. (2020). Biomechanics of handcycling propulsion in a 30-min continuous load test at lactate threshold: Kinetics, kinematics, and muscular activity in able-bodied participants. *European journal of applied physiology*, 120(6), 1403–1415. <https://doi-org.lib-ezproxy.concordia.ca/10.1007/s00421-020-04373-x>
41. Roca, J., Hogan, M. C., Story, D., Bebout, D. E., Haab, P., Gonzalez, R., Ueno, O. & Wagner, P. D. (1985). Evidence for tissue diffusion limitation of  $\dot{V}O_{2max}$  in normal humans. *J Appl Physiol*, 67(1):291-9. doi: 10.1152/jappl.1989.67.1.291. PMID: 2759955.
42. Saltin B. (1985). Hemodynamic adaptations to exercise. *Am J Cardiol*, 55(10):42D-47D doi: 10.1016/0002-9149(85)91054-9. PMID: 3993550
43. Schumacher, Y. O., Ruthardt, S., Schmidt, M., Ahlgrim, C., Roecker, K. & Pottgiesser, T. (2009). Total haemoglobin mass but not cardiac volume adapts to long-term endurance exercise in highly trained spinal cord injured athletes. *Eur J Appl Physiol*; 105(5):779-85. doi: 10.1007/s00421-008-0963-8. Epub 2009 Jan 6. PMID: 19125284
44. Seo, M. W., Jung, S. W., Kim, S. W., Lee, J. M., Jung, H. C., & Song, J. K. (2021). Effects of 16 Weeks of Resistance Training on Muscle Quality and Muscle Growth Factors in Older Adult Women with Sarcopenia: A Randomized Controlled Trial. *International journal of environmental research and public health*, 18(13), 6762. <https://doi-org.lib-ezproxy.concordia.ca/10.3390/ijerph18136762>
45. Skattebo, Ø., Calbet, J. A. L., Rud, B., Capelli, C., Hallén, J. (2020). Contribution of oxygen extraction fraction to maximal oxygen uptake in healthy young men. *Acta Physiol (Oxf)*. 230(2):e13486. doi: 10.1111/apha.13486. Epub 2020 May 30. PMID: 32365270; PMCID: PMC7540168
46. Souissi, A., Haddad, M., Dergaa, I., Ben Saad, H., & Chamari, K. (2021). A new perspective on cardiovascular drift during prolonged exercise. *Life sciences*, 287, 120109. <https://doi-org.lib-ezproxy.concordia.ca/10.1016/j.lfs.2021.120109>
47. Stephenson, B., Stone, B., Mason, B. & Goosey-Tolfrey, V. (2020). Physiology of Handcycling: A Current Sports Perspective. doi: 10.1111/SMS.13835
48. Stöcker, F., Von Oldershausen, C., Paternoster, F. K, Schulz, T. & Oberhoffer, R. (2016). Relationship of post-exercise muscle oxygenation and duration of cycling exercise. *BMC Sports Sci Med Rehabil*, 8, 9. <https://doi.org/10.1186/s13102-016-0036-y> 3
49. Stöggl, T., & Born, D. P. (2021). Near Infrared Spectroscopy for Muscle Specific Analysis of Intensity and Fatigue during Cross-Country Skiing Competition-A Case Report. *Sensors (Basel, Switzerland)*, 21(7), 2535. <https://doi-org.lib-ezproxy.concordia.ca/10.3390/s21072535>

50. Stone, B., Mason, B. S., Stephenson, B. T. & Goosey-Tolfrey V. L. (2020). Physiological responses during simulated 16 km recumbent handcycling time trial and determinants of performance in trained handcyclists. *Eur J Appl Physiol.*, 120(7):1621-1628. doi: 10.1007/s00421-020-04390-w. Epub 2020 May 20. PMID: 32435985; PMCID: PMC7295712
51. Strangier, C., Abel, T., Zeller, S., Quittmann, O. J., Perret, C., & Strüder, H. K. (2019). Comparison of Different Blood Lactate Threshold Concepts for Constant Load Performance Prediction in Spinal Cord Injured Handcyclists. *Frontiers in physiology*, 10, 1054. <https://doi-org.lib-ezproxy.concordia.ca/10.3389/fphys.2019.01054>
52. Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) project, retrieved March 30<sup>th</sup>, 2021 from <http://www.seniam.org/>

## Appendix

### Associations between Physiological and Performance Parameters

Table 4, Table 5 and Table 6 present correlations between physiological parameters and performance in the Wingate, in the incremental test, and in the TT respectively.

Variable	Average P (W)	Average P (W/kg)	PPO (W)	PPO (W/kg)	Fatigue Index
<b>ΔSmO<sub>2</sub>min, %</b>					
TB	-0.379 [-2.186, 1.389]	-0.468 [-2.119, 1.170]	-0.459 [-2.210, 1.241]	-0.553 [-2.168, 1.017]	-0.945* [-1.729, -0.384] ( <i>P</i> = 0.015)
AD	-0.6 (S) [-0.976, 0.671]	-0.154 (S) [-0.914, 0.845]	-0.3 (S) [-0.939, 0.803]	-0.2 (S) [-0.922, 0.833]	-0.5 (S) [-0.965, 0.726]
BB	0.178 [-2.088, 1.714]	0.622 [-2.087, 0.828]	-0.183 [-2.102, 1.715]	0.527 [-2.173, 1.076]	0.115 [-2.169, 1.912]
ER	0.028 [-1.359, 1.416]	0.002 [-1.386, 1.390]	-0.035 [-1.422, 1.352]	-0.092 [-1.474, 1.291]	-0.701 [-1.691, 0.289]
Mean	-0.172 [-1.540, 1.195]	-0.248 [-1.593, 1.097]	-0.228 [-1.580, 1.123]	-0.323 [-1.637, 0.991]	-0.643 [-1.706, 0.419]
<b>Δ[HHb]max, AU</b>					
TB	0.777 [-0.400, 2.042]	0.513 [-1.188, 2.336]	0.763 [-0.452, 2.073]	0.543 [-1.119, 2.331]	-0.031 [-2.087, 2.019]
AD	1.000** (S) ( <i>P</i> = .001)	0.462 (S) [0.743, 0.961]	0.9* (S) [0.169, 0.996] ( <i>P</i> = 0.037)	0.6 (S) [0.671, 0.976]	0.3 (S) [-0.803, 0.939]
BB	0.657 [-0.764, 2.147]	0.875 [-0.017, 1.788]	0.690 [-0.677, 2.135]	0.870 [-0.036, 1.847]	0.383 [-1.470, 2.326]
ER	0.598 [-0.515, 1.711]	0.646 [-0.414, 1.706]	0.643 [-0.419, 1.706]	0.685 [-0.326, 1.696]	0.678 [-0.342, 1.698]
Mean	0.704 [-0.282, 1.690]	0.776 [-0.98, 1.651]	0.731 [-0.217, 1.678]	0.784 [-0.078, 1.646]	0.493 [-0.715, 1.701]
<b>Reoxy rate, %/sec</b>					
TB	0.820* (S) [-0.15, 0.99] ( <i>P</i> = 0.046)	0.585 (S) [-0.504, 0.956]	0.698 (S) [-0.38, 0.97]	0.577 (S) [-0.511, 0.955]	0.698 (S) [-0.38, 0.97]
AD	0.916* [0.168, 1.574] ( <i>P</i> = 0.029)	0.884* [0.027, 1.72] ( <i>P</i> = 0.046)	0.948* [0.387, 1.617] ( <i>P</i> = 0.014)	0.7 (S) [-0.591, 0.984]	0.547 [-1.107, 2.331]
BB	0.625 [-0.77, 1.959]	0.411 [-1.249, 2.06]	0.617 [-0.73, 2.148]	0.359 (S) [-0.783, 0.947]	0.624 [-0.909, 2.303]
ER	0.829* (S) [-0.13, 0.99] ( <i>P</i> = 0.042)	0.667 (S) [-0.42, 0.97]	1.000** (S) ( <i>P</i> = < .001)	0.714 (S) [-0.36, 0.97]	0.429 (S) [-0.619, 0.928]
Mean	0.852* [0.125, 1.579] ( <i>P</i> = 0.031)	0.860* [0.151, 1.569] ( <i>P</i> = 0.028)	0.892* [0.264, 1.52] ( <i>P</i> = 0.017)	0.657 (S) [-0.43, 0.97]	0.639 [-0.429, 1.707]

Table 4.1. Correlations between Performance and Physiological Parameters for the Wingate in Handcyclists (n = 6). Abbreviations: AU, arbitrary units; P, power; ΔSmO<sub>2</sub>min, minimal muscle O<sub>2</sub> saturation (min - baseline); Δ[HHb]max, maximal muscle O<sub>2</sub> extraction (max - baseline). Note: values are Pearson correlations and Spearman's rho (S), with 95% confidence limits. \**P* < .05. \*\**P* < .01.

Variable	Average P (W)	Average P (W/kg)	PPO (W)	PPO (W/kg)	Fatigue Index
<b>Deoxy rate, %/sec</b>					
TB	-0.795 [-1.637, 0.047]	-0.553 [-1.71, 0.604]	-0.802 [-1.631, 0.026]	-0.6 (S) [-0.976, 0.617]	-0.260 [-1.601, 1.080]
AD	-0.900* (S) [-0.996, 0.169] ( <i>P</i> = 0.037)	-0.308 (S) [-0.940, 0.801]	-0.800 (S) [-0.991, 0.457]	-0.5 (S) [-0.965, 0.726]	0.100 (S) [-0.859, 0.903]
BB	-0.586 [-1.973, 0.858]	-0.148 [-1.94, 1.648]	-0.512 [-2.209, 1.126]	-0.6 (S) [-0.976, 0.617]	0.642 [-0.857, 2.293]
ER	-0.867* [-1.558, -0.177] ( <i>P</i> = 0.025)	-0.696 [-1.693, 0.3]	-0.886* [-1.53, -0.241] ( <i>P</i> = 0.019)	-0.6 (S) [-0.976, 0.617]	-0.406 [-1.675, 0.863]
Mean	-0.921** [-1.462, -0.381] ( <i>P</i> = 0.009)	-0.658 [-1.703, 0.388]	-0.901* [-1.504, -0.298] ( <i>P</i> = 0.014)	-0.714 (S) [-0.97, 0.36]	0.025 [-1.363, 1.413]
Weight, kg	0.768 [-0.12, 1.657]	0.340 [-0.965, 1.646]	0.739 [-0.196, 1.674]	0.429 (S) [-0.619, 0.928]	-0.197 [-1.558, 1.164]
Training, hours/week	-0.269 [-1.606, 1.068]	0.068 [-1.317, 1.453]	-0.283 [-1.615, 1.048]	0.314 (S) [-0.683, 0.902]	-0.055 [-1.441, 1.331]
<b>Right arm girth, cm</b>					
Upper arm	0.768 [-0.073, 1.645]	0.711 [-0.265, 1.687]	0.772 [-0.11, 1.654]	0.829* (S) [-0.13, 0.99] ( <i>P</i> = 0.042)	0.049 [-1.338, 1.435]
Forearm	0.832* [0.061, 1.602] ( <i>P</i> = 0.04)	0.757 [-0.149, 1.664]	0.827* [0.048, 1.607] ( <i>P</i> = 0.042)	0.771 (S) [-0.26, 0.98]	0.169 [-1.200, 1.537]
<b>Prehension force, kg</b>					
Right	0.879* [0.218, 1.541] ( <i>P</i> = 0.021)	0.957** [0.554, 1.36] ( <i>P</i> = 0.003)	0.870* [0.168, 1.554] ( <i>P</i> = 0.024)	0.943** (S) [0.38, 1.00] ( <i>P</i> = 0.005)	0.093 [-1.290, 1.475]
Left	0.823* [0.033, 1.612] ( <i>P</i> = 0.044)	0.860* [0.15, 1.569] ( <i>P</i> = 0.028)	0.794 [-0.05, 1.638]	0.886* (S) [0.07, 0.99] ( <i>P</i> = 0.019)	-0.124 [-1.501, 1.254]
Handcycling experience, y	0.341 [-0.964, 1.646]	-0.045 [-1.432, 1.342]	0.311 [-1.009, 1.63]	0.239 (S) [-0.718, 0.883]	-0.213 [-1.569, 1.144]

Table 4.2. Correlations between Performance and Physiological Parameters for the Wingate in Handcyclists (n = 6). Abbreviations: AU, arbitrary units; P, power;  $\Delta$ SmO<sub>2</sub>min, minimal muscle O<sub>2</sub> saturation (min - baseline);  $\Delta$ [HHb]max, maximal muscle O<sub>2</sub> extraction (max - baseline). Note: values are Pearson correlations and Spearman's rho (S), with 95% confidence limits. \**P* < .05. \*\**P* < .01.

Variable	MAP (W)	MAP (W/kg)	Variable	MAP (W)	MAP (W/kg)
$\Delta\text{SmO}_2\text{min, \%}$			$\text{VO}_2\text{max, mL/kg/min}$	0.733 [-0.461, 1.772]	0.895* [0.084, 1.907] ( $P = 0.04$ )
TB	-0.757 [-1.545, 0.349]	0.371 [-0.554, 0.863]	$\text{VO}_2\text{max L/min}$	0.928* [-0.528, 2.997] ( $P = 0.023$ )	0.601 [-1.624, 1.207]
AD	-0.604 [-1.633, 0.680]	0.389 [-0.541, 0.864]	HRmax, bpm	-0.075 [-1.460, 1.309]	-0.463 [-1.693, 0.767]
BB	-0.734 [-1.677, 0.208]	-0.666 [-1.702, 0.370]	RERmax, AU	-0.531 [-1.707, 0.645]	-0.574 [-1.711, 0.562]
ER	0.103 [-1.277, 1.484]	0.116 [-1.263, 1.495]	Max (a-v) $\text{O}_2$ diff, mL/dL	-0.364 [-14.031, 13.194]	0.886 [-1.559, 2.111]
Mean	-0.635 [-1.707, 0.438]	-0.123 [-1.500, 1.255]	Last stage $\Delta[\text{THb}]$ , AU		
$\Delta[\text{HHb}]\text{max, AU}$			TB	-0.086 [-1.513, 1.378]	-0.834 [-0.767, 0.075]
TB	0.682 [-0.522, 1.599]	-0.482 [-0.869, 0.468]	AD	-0.017 [-1.464, 1.437]	-0.389 [-0.864, 0.541]
AD	0.726 [-0.426, 1.571]	-0.434 [-0.867, 0.507]	BB	0.618 (S) [-0.472, 0.961]	0.088 (S) [-0.780, 0.840]
BB	0.863* [0.160, 1.565] ( $P = 0.027$ )	0.691 [-0.313, 1.695]	ER	0.408 [-0.859, 1.676]	0.061 [-1.325, 1.447]
ER	0.012 [-1.376, 1.400]	0.037 [-1.350, 1.425]	Mean	0.507 [-0.689, 1.704]	0.330 [-0.980, 1.641]
Mean	0.809 [-0.008, 1.625]	0.252 [-1.091, 1.595]	$\text{Qmax, L/min}$	0.936 [-3.066, 4.694]	-0.937 [-1.677, 1.093]
Reoxy rate, %/sec			SVmax, mL/beat	0.781 [-6.215, 7.574]	-0.999* [-4.937, -1.478] ( $P = 0.027$ )
TB	0.382 [-1.667, 2.635]	-0.031 [-4.498, 4.348]	VEmax, mL/min	0.807 [-0.248, 1.691]	0.814 [-0.283, 2.094]
AD	0.631 [-0.709, 1.838]	-0.022 [-1.671, 1.631]	V Tmax, mL	-0.096 [-1.478, 1.286]	0.235 [-1.114, 1.584]
BB	0.712 [-0.263, 1.687]	0.295 [-1.031, 1.622]	$\Delta\text{SmO}_2$ last stage, %		
ER	0.705 [-0.28, 1.69]	0.389 [-0.890, 1.668]	TB	-0.800 [-1.502, 0.240]	0.331 [-0.582, 0.857]
Mean	0.646 [-0.414, 1.706]	0.260 [-1.080, 1.601]	AD	-0.451 [-1.651, 0.939]	0.485 [-0.465, 0.869]
Weight, kg	0.727 [-0.227, 1.68]	-0.028 [-1.416, 1.359]	BB	-0.680 [-1.698, 0.337]	-0.657 [-1.703, 0.389]
Training, hours/week	0.021 [-1.367, 1.409]	0.681 [-0.334, 1.697]	ER	0.261 [-1.079, 1.601]	0.226 [-1.126, 1.578]
Right arm girth, cm			Mean	-0.608 [-1.710, 0.494]	-0.123 [-1.500, 1.255]
Upper arm	0.965** [0.6, 1.33] ( $P = 0.002$ )	0.769 [-0.117, 1.656]			
Forearm	0.947** [0.501, 1.393] ( $P = 0.004$ )	0.714 [-0.259, 1.686]			
Prehension force, kg					
Right	0.807 [-0.012, 1.627]	0.626 [-0.456, 1.709]			
Left	0.758 [-0.148, 1.664]	0.539 [-0.631, 1.708]			
Handcycling experience, y	0.593 [-0.524, 1.711]	0.182 [-1.183, 1.547]			
RRmax, breaths/min	-0.794 (S) [-0.983, 0.212]	-0.265 (S) [-0.890, 0.706]			
Lactate max, mmol/L	0.086 (S) [-0.781, 0.840]	0.829* (S) [-0.126, 0.987] ( $P = 0.042$ )			

Table 5. Correlations between Performance and Physiological Parameters for the Incremental Test in Handcyclists (n = 6). Abbreviations: AU, arbitrary units; MAP, max aerobic power; SmO<sub>2</sub>, muscle O<sub>2</sub> saturation;  $\Delta[\text{HHb}]\text{max}$ , maximal muscle O<sub>2</sub> extraction (max - baseline); last stage  $\Delta[\text{THb}]$ , average total hemoglobin (average - baseline) of last stage; SV, stroke volume; VO<sub>2</sub>max, maximal oxygen uptake; RRmax, maximal respiratory rate; RERmax; maximal respiratory exchange ratio; Qmax, maximal cardiac output; VE<sub>max</sub>, maximal minute ventilation. Note: Values are Pearson correlations and Spearman's rho (S), with 95% confidence limits. \*P < .05. \*\*P < .01. As a result of missing data, n is lower for SVmax, Qmax (n=3), and max(a-v)O<sub>2</sub> diff (n=2), and for VO<sub>2</sub>max (n=5).

Variable	Distance (m)	Mean P (%MAP)	Average P (W)	Average P (W/kg)
<b>ΔSmO<sub>2</sub>min, %</b>				
TB	-0.439 [-2.061, 1.491]	-0.011 [-2.662, 2.643]	-0.729 [-2.050, 0.988]	-0.445 [-1.288, 0.926]
AD	-0.873 [-1.531, 0.397]	0.491 [-1.883, 2.739]	-0.391 [-2.326, 1.756]	-0.830 [-1.027, 0.353]
BB	-0.524 [-2.024, 1.343]	0.559 [-1.711, 2.687]	-0.790 [-1.937, 0.786]	-0.789 [-1.080, 0.439]
ER	-0.451 [-2.091, 1.190]	-0.436 [-2.090, 1.217]	-0.774 [-1.937, 0.389]	-0.463 [-2.092, 1.165]
Mean	-0.810 [-1.888, 0.268]	-0.386 [-2.081, 1.310]	-0.879* [-1.754, -0.005] (P = 0.049)	-0.819 [-1.873, 0.236]
<b>Δ[HHb]max, AU</b>				
TB	0.272 [-1.725, 2.079]	0.171 [-2.465, 2.763]	0.776 [-0.834, 1.965]	0.290 [-1.065, 1.301]
AD	0.934 [-0.102, 1.315]	-0.491 [-2.739, 1.882]	0.134 [-2.100, 2.296]	0.788 [-0.441, 1.081]
BB	0.446 [-1.480, 2.059]	-0.375 [-2.786, 2.133]	0.875 [-0.435, 1.711]	0.681 [-0.629, 1.182]
ER	0.423 [-1.243, 2.088]	0.472 [-1.148, 2.092]	0.789 [-0.341, 1.918]	0.453 [-1.186, 2.091]
Mean	0.760 [-0.435, 1.954]	0.413 [-1.261, 2.086]	0.873 [-0.22, 1.768]	0.769 [-0.407, 1.944]
<b>Reoxy rate, %/sec</b>				
TB	-0.579 [-1.988, 1.236]	0.491 [-2.476, 3.603]	0.833 [-0.62, 1.834]	-0.314 [-7.881, 6.336]
AD	-0.634 [-1.941, 1.118]	0.968* [0.184, 1.505] (P = 0.032)	0.240 [-3.721, 4.381]	-0.750 [-1.123, 0.513]
BB	-0.985* [-0.979, -0.301] (P = 0.015)	0.803 [-0.879, 2.28]	0.180 [-3.858, 4.352]	-0.878 [-0.948, 0.235]
ER	-0.270 [-2.039, 1.499]	0.774 [-0.389, 1.937]	0.386 [-1.310, 2.081]	-0.001 [-1.838, 1.836]
Mean	-0.174 [-1.983, 1.636]	0.818 [-0.237, 1.847]	0.510 [-1.07, 2.09]	0.088 [-1.742, 1.918]
<b>Deoxy rate, %/sec</b>				
TB	0 (S) [-0.961, 0.961]	-0.2 (S) [-0.975, 0.944]	-0.8 (S) [-0.998, 0.819]	-0.105 (S) [-0.969, 0.953]
AD	-0.008 [-4.696, 4.670]	-0.008 [-3.498, 3.481]	-0.999** [-0.827, -0.63] (P < .001)	-0.291 [-7.879, 6.447]
BB	-0.256 [-4.921, 4.134]	0.134 [-3.305, 3.611]	-0.954* [-1.358, -0.033] (P = .046)	-0.482 [-7.746, 5.375]
ER	-0.273 [-2.040, 1.495]	-0.365 [-2.076, 1.345]	-0.902* [-1.696, -0.107] (P = .036)	-0.455 [-2.091, 1.182]
Mean	-0.5 (S) [-0.965, 0.726]	-0.6 (S) [-0.976, 0.671]	-0.900* (S) [-0.996, 0.169]	-0.564 (S) [-0.972, 0.693]

Table 6.1. Correlations between Performance and Physiological Parameters for the TT in Handcyclists (n = 5). Abbreviations: AU, arbitrary units; P, power; MAP, max aerobic power; SmO<sub>2</sub>, muscle O<sub>2</sub> saturation; Δ[HHb]max, maximal muscle O<sub>2</sub> extraction (max - baseline); Δ[THb] average, average total hemoglobin (average - baseline). Note: Values are Pearson correlation and Spearman's rho (S), with 95% confidence limits. \*P < .05. \*\*P < .01.

Variable	Distance (m)	Mean P (%MAP)	Average P (W)	Average P (W/kg)
Weight, kg	-0.296 [-2.051, 1.458]	0.386 [-1.341, 2.076]	0.539 [-1.008, 2.087]	-0.102 [-1.930, 1.725]
Training, hours/week	0.862 [-0.071, 1.794]	0.319 [-1.423, 2.060]	0.281 [-1.483, 2.044]	0.734 [-0.512, 1.981]
Right arm girth, cm				
Upper arm	0.666 [-0.704, 2.036]	0.569 [-0.941, 2.08]	0.988** [0.705, 1.271]	0.834 [-0.18, 1.848]
Forearm	0.583 [-0.909, 2.076]	0.575 [-0.928, 2.078]	0.996** [0.831, 1.161]	0.758 [-0.44, 1.956]
Prehension force, kg				
Right	0.624 [-0.811, 2.06]	-0.066 [-1.899, 1.767]	0.795 [-0.319, 1.909]	0.649 [-0.748, 2.047]
Left	0.615 [-0.834, 2.064]	-0.186 [-1.991, 1.620]	0.662 [-0.714, 2.039]	0.566 [-0.949, 2.081]
Handcycling experience, y	-0.025 [-1.861, 1.812]	0.961** [0.45, 1.471]	0.449 [-1.193, 2.091]	0.178 [-7.171, 8.738]
VO <sub>2</sub> peak (L/min)	0.464 [-0.708, 0.955]	0.719 [-0.446, 0.980]	0.888* [0.029, 0.993] (P = 0.044)	0.575 [-0.623, 0.967]
VO <sub>2</sub> peak (mL/kg/min)	0.464 [-0.708, 0.955]	0.716 [-0.451, 0.980]	0.889* [0.031, 0.993] (P = 0.044)	0.575 [-0.623, 0.967]
Q <sub>peak</sub>	0.335 [-0.923, 0.980]	0.931 [-0.289, 0.999]	0.813 [-0.677, 0.996]	0.532 [-0.878, 0.988]
ΔSmO <sub>2</sub> average, %				
TB	-0.201 [-2.067, 1.805]	-0.373 [-2.786, 2.136]	-0.578 [-2.231, 1.388]	-0.095 [-1.269, 1.192]
AD	-0.840 [-1.618, 0.526]	0.377 [-2.129, 2.786]	-0.357 [-2.332, 1.812]	-0.746 [-1.127, 0.520]
BB	-0.948 [-1.245, 0.013]	0.747 [-1.111, 2.414]	0.349 [-1.824, 2.333]	-0.781 [-1.089, 0.454]
ER	-0.557 [-2.083, 0.968]	-0.418 [-2.087, 1.252]	-0.670 [-2.034, 0.694]	-0.503 [-2.091, 1.086]
Mean	-0.825 [-1.863, 0.212]	-0.484 [-2.092, 1.123]	-0.774 [-1.938, 0.390]	-0.794 [-1.911, 0.323]
Δ[THb] average, AU				
TB	-0.672 [-1.901, 1.028]	0.694 [-1.305, 2.515]	0.712 [-1.037, 2.076]	-0.499 [-1.274, 0.869]
AD	-0.447 (S) [-0.988, 0.918]	0 (S) [-0.961, 0.961]	-0.447 (S) [-0.988, 0.918]	-0.236 (S) [-0.977, 0.941]
BB	-0.258 (S) [-0.978, 0.939]	-0.258 (S) [-0.978, 0.939]	0.775 (S) [-0.834, 0.997]	0.544 (S) [-0.903, 0.991]
ER	0.707 (S) [-0.584, 0.985]	0.707 (S) [-0.584, 0.985]	0.707 (S) [-0.584, 0.985]	0.725 (S) [-0.564, 0.986]
Mean	0.105 (S) [-0.858, 0.904]	0.738 (S) [-0.549, 0.987]	0.949* (S) [0.151, 0.998] (P = 0.014)	0.433 (S) [-0.756, 0.957]

Table 6.2. Correlations between Performance and Physiological Parameters for the TT in Handcyclists (n = 5). Abbreviations: AU, arbitrary units; P, power; MAP, max aerobic power; SmO<sub>2</sub>, muscle O<sub>2</sub> saturation; Δ[HHb]max, maximal muscle O<sub>2</sub> extraction (max - baseline); Δ[THb] average, average total hemoglobin (average - baseline). Note: Values are Pearson correlation and Spearman's rho (S), with 95% confidence limits. \*P < .05. \*\*P < .01.

## CHAPTER 3: SUMMARY, CONCLUSIONS AND FUTURE DIRECTIONS

### Summary, Conclusion, and Future Directions

This project provided a complete physiological profile of highly trained handcyclists with various parameters including some that were never assessed before with this specific group of athletes (muscle oxygenation parameters). By providing these various physiological markers, this study was the first to measure peripheral and central cardiovascular changes in this group of paracyclists. It was possible to confirm the hypothesis that peripheral cardiovascular adaptations are important in an upper body sport like handcycling. Thus, this project clarifies the physiological determinants of the sport, and reinforces the usefulness of portable NIRS monitors in para-cycling to optimize training and testing protocols. By providing a thorough analysis of muscles oxygenation characteristics of the athletes throughout the year (before, during and after specific training blocks), it is possible to target the development of peripheral adaptations such as muscle recruitment and activity optimization, capillary density, and oxidative capacity, which can orientate the choice of training and testing methods and can contribute to the development of norms for the different tests and physiological measures used in handcycling. Results can provide insights regarding training protocols and interventions better suited to improve handcycling performance (e.g., heat training, altitude training, ischemic preconditioning, etc.). Although this research project is carried out in para-cycling, the results could potentially be generalizable to other Paralympic sports, where efforts are generated by the upper limbs, such as para canoe-kayak, para-swimming, or wheelchair sports where athletes have lower limb restrictions, and where peripheral cardiovascular adaptations may predominate over the central  $\text{VO}_2$  adaptations to predict performance. Results of our study could also be used for further talent identification in development athletes, or in the identification of physiological strengths and weaknesses with the examination of superior peripheral abilities at younger ages (e.g., ability of muscles to extract oxygen, and rapid muscle deoxygenation and reoxygenation patterns, that are linked to muscle recruitment efficiency and superior oxidative capacity) possibly helping team recruitment.

### Training recommendations

#### Adaptations derived from near-infrared spectroscopy

- Very low maximal deoxygenation ( $\text{SmO}_2\text{min}$  - nadir) is the ability to sustain very low levels of muscle  $\text{O}_2$  and should be developed for TT performance (by aerobic adaptations - oxidative capacity, capillarization, etc.). This adaptation could be developed through different methods such as high intensity interval training and sprint interval training;
- Maximal muscle  $\text{O}_2$  extraction seems to be necessary in both aerobic and glycolytic efforts, although more important for aerobic performance;
- Total hemoglobin content is important for aerobic performance, and ER and BB may have higher levels in a TT, confirming their high irrigation, and their high blood and  $\text{O}_2$  local delivery during this type of effort, thus a significant aerobic contribution. This is in line with the strong association between  $\text{VO}_2\text{peak}$  and TT power, confirming the importance of central aerobic adaptations to TT performance.
- Fast muscle deoxygenation is an important peripheral adaptation for both aerobic and sprint performance, although deoxygenation is generally much faster in higher-intensity (shorter) efforts as supported by previous literature in the field<sup>78</sup>.
- Fast muscle reoxygenation following an effort is a crucial adaptation for the development of peripheral characteristics such as capillarization and oxidative capacity and is associated to both aerobic and sprint performance. However, it seems to be more important for sprint performance and should be developed with various training protocols

such as repeated sprint training. This peripheral adaptation should be used as a marker to follow athlete's progress over time.

#### Other physiological adaptations

- Strength training should be included in training plans of elite handcyclists, as strong grip strength is necessary for handcycling sprint performance;
- Large arm girths because of muscle hypertrophy contribute to the generation of superior power output during aerobic and sprint handcycling efforts, confirming the importance of resistance training in this sport;
- High  $VO_{2peak}$  is necessary in handcycling aerobic efforts;
- Level of experience in the sport is an important aspect of TT performance;
- Maximal blood lactate contributes to the development of a high power during incremental handcycling performance;
- $Q_{peak}$  and  $SV_{peak}$  seem to be important for aerobic performance;

Overall, peripheral cardiovascular contributions to handcycling performance are confirmed and should be included in training plans of elite handcyclists. In terms of the central adaptations of  $VO_{2max}$ , they also seem to contribute largely to handcycling performance, thus more classic cardiovascular variables should be measured in elite handcyclists to monitor their progress.

Future studies with elite handcyclists should focus on the development of new training protocols that can improve peripheral adaptations (muscle oxygenation kinetics) in this sport. The training recommendations from this study need to be confirmed with future studies and these should also aim to confirm the muscle recruitment and activity during different handcycling efforts (glycolytic and aerobic) with NIRS and electromyography. Future studies should also evaluate the strength training benefits on handcycling performance.

#### **Difficulties encountered due to the COVID-19 pandemic**

Special sanitary measures were put in place for this research due to the COVID-19 pandemic. Athletes, research personnel, coaches, and any other person allowed to attend the testing sessions had to fill an assessment of symptoms questionnaire before coming to the testing centers (National Sport Institute of Quebec and Calgary) and had to show their complete vaccination passport at the entrance and prior to each day. A person without his or hers two shots of vaccine against COVID-19 at least administered within the past 14 days did not have access to the training center. This aspect significantly affected the recruitment of participants for the study. All persons attending the research had to wear a surgical mask and these were changed every two hours. Traveling and access to the training centers was highly restricted (sometimes even closed) for the majority of the duration of the study, so it was difficult to have opportunities to collect data and the plans changed frequently at the last minute. As soon as a study participant or a person from the research staff felt any symptoms, they had to isolate, and so this aspect significantly complicated the data collection. All of these aspects can explain the small sample size of the study.

## CHAPTER 1 REFERENCES

1. Liljedahl, J. B., Bjerkefors, A., Arndt, A. & Nooijen, C. F. J. (2020). Para-cycling race performance in different sport classes, *Disability and Rehabilitation*, DOI: 10.1080/09638288.2020.1734106
2. Nevin, J., Smith, P., Waldron, M., Patterson, S., Price, M., Hunt, A. & Blagrove, R. (2018). Efficacy of an 8-Week Concurrent Strength and Endurance Training program on Hand Cycling Performance. *J Strength Cond Res.* 32(7):1861-1868. doi: 10.1519/JSC.0000000000002569. PMID: 29561384
3. Stephenson, B., Stone, B., Mason, B. & Goosey-Tolfrey, V. (2020). Physiology of Handcycling: A Current Sports Perspective. doi: 10.1111/SMS.13835
4. Abel, T., Schneider, S., Platen, P. & Strüder, H. K. (2006). Performance diagnostics in handbiking during competition. *Spinal Cord*, 44:211-216. <https://doi.org/10.1038/sj.sc.3101845>
5. Stone, B., Mason, B. S., Stephenson, B. T. & Goosey-Tolfrey V. L. (2020). Physiological responses during simulated 16 km recumbent handcycling time trial and determinants of performance in trained handcyclists. *Eur J Appl Physiol.*, 120(7):1621-1628. doi: 10.1007/s00421-020-04390-w. Epub 2020 May 20. PMID: 32435985; PMCID: PMC7295712
6. Canadian Government. (2020). Retrired October 12<sup>th</sup>, 2020 from <https://www.canada.ca/en/canadian-heritage/services/sport-policies-acts-regulations/high-performance-strategy.html>
7. Canadian Paralympic Committee. *Para-Cycling*. Retrieved October 12<sup>th</sup>, 2020 from <https://paralympic.ca/paralympicsports/paracycling#:~:text=Para%20cycling%20was%20first%20developed,the%202004%20Games%20in%20Athens>
8. Lepêtre, P-M., Weissland, T., Slawinski, J. & Lopes, P. (2012). Para-cycling performance was rather limited by physiological than functional factors. *Frontiers in Physiology*, 3(327). <https://doi.org/10.3389/fphys.2012.00327>
9. Muchaxo, R. A., de Groot, S., van der Woude, L. V., Janssen, T. J., & Nooijen, C. (2020). Do Handcycling Time-Trial Velocities Achieved by Para-Cycling Athletes Vary Across Handcycling Classes? *Adapted Physical Activity Quarterly*, 37(4), 461-480. Retrieved Feb 1, 2021, from <https://journals.humankinetics.com/view/journals/apaq/37/4/article-p461.xml>
10. Lovell D., Shields D., Beck B., Cuneo R. & McLellan C. (2012). The aerobic performance of trained and untrained handcyclists with spinal cord injury. *Eur J Appl Physiol.*, 112(9):3431-7. doi: 10.1007/s00421-012-2324-x. Epub 2012 Jan 26. PMID: 22278391
11. Fisher, G., Figueiredo, P. & Ardigo, L. P. (2015). Physiological Performance Determinants of a 22-km Handbiking Time Trial. *International Journal of Sports Physiology and Performance*, 10(8): 965-971. <http://dx.doi.org/10.1123/ijsp.2014-0429>
12. Faupin A., Gorce P., Watelain E., Meyer C. & Thevenon A. (2010). A biomechanical analysis of handcycling: a case study. *J Appl Biomech*, (2):240-5. doi: 10.1123/jab.26.2.240. PMID: 20498497
13. Quittmann, O. J., Abel, T., Albracht, K. & Strüder, H. K. (2019). Reliability of muscular activation patterns and their alterations during incremental handcycling in able-bodied participants, *Sports Biomechanics*, DOI: 10.1080/14763141.2019.1593496
14. Stone, B., Mason, B. S., Warner, M. B. & Goosey-Tolfrey, V. L. (2019). Shoulder and thorax kinematics contribute to increased power output of competitive handcyclists. *Scand J Med Sci Sports*, 29(6):843-853. doi: 10.1111/sms.13402. Epub 2019 Mar 18. PMID: 30739351; PMCID: PMC6850573

15. Wright, R. L. (2016). Positive Pacing Strategies Are Utilized by Elite Male and Female Para-cyclists in Short Time Trials in the Velodrome. *Front Physiol.*, 18(6):425. doi: 10.3389/fphys.2015.00425. PMID: 26834643; PMCID: PMC4716664
16. Bassett, D. R. Jr. & Howley, E. T. (2000). Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Med Sci Sports Exerc*, 32(1):70-84. doi: 10.1097/00005768-200001000-00012. PMID: 10647532
17. Saltin B. (1985). Hemodynamic adaptations to exercise. *Am J Cardiol*, 55(10):42D-47D. doi: 10.1016/0002-9149(85)91054-9. PMID: 3993550
18. Vincent, J. L. (2008). Understanding cardiac output. *Crit Care*, 12(4):174. doi: 10.1186/cc6975. Epub 2008 Aug 22. PMID: 18771592; PMCID: PMC2575587
19. Joyner, M. J. & Coyle, E. F. (2008). Endurance exercise performance: the physiology of champions. *J Physiol*, 586(1):35-44. doi: 10.1113/jphysiol.2007.143834. Epub 2007 Sep 27. PMID: 17901124; PMCID: PMC2375555
20. De Groot, S., Postma, K., Vliet, L. V., Timmermans, R. & Valent, L. J. M. (2014). Mountain time trial in handcycling: exercise intensity and predictors of race time in people with spinal cord injury. *International Spinal Cord Society*, 52:455-461. doi:10.1038/sc.2014.58
21. Vikmoen, O., Ellefsen, S., Trøen, Ø., Hollan, I., Hanestadhaugen, M., Raastad, T. & Rønnestad, B. R. (2016). Strength training improves cycling performance, fractional utilization of VO<sub>2</sub>max and cycling economy in female cyclists. *Scand J Med Sci Sports*, 26(4):384-96. doi: 10.1111/sms.12468. Epub 2015 Apr 18. PMID: 25892654
22. Hasnan, N., Mohamad, S. N. S, Hamzaid, N. A, Teoh, M. X., Ahmadi, S. & Davis, G. M. (2018). Muscle oxygenation during hybrid arm and functional electrical stimulation-evoked leg cycling after spinal cord injury. *Medicine (Baltimore)*, 97(43): e12922. doi: 10.1097/MD.00000000000012922. PMID: 30412097; PMCID: PMC6221724
23. Ferrari, M., Muthalib, M. & Quaresima, V. (2011). The use of near-infrared spectroscopy in understanding skeletal muscle physiology: recent developments. *Philos Trans A Math Phys Eng Sci*, 369(1955):4577-90. doi: 10.1098/rsta.2011.0230. PMID: 22006907
24. Shibuya, K. & Tanaka, J. (2003). Skeletal muscle oxygenation during incremental exercise. *Arch Physiol Biochem*, 111(5):475-8. doi: 10.3109/13813450312331342355. PMID: 16026037
25. Handrakis, J. P., Trbovich, M., Hagen, E. M. & Price, M. (2017). Thermodyregulation in persons with spinal cord injury: case series on use of the autonomic standards. *Spinal Cord Ser Cases*, 3:17086. doi: 10.1038/s41394-017-0026-7. PMID: 29423292; PMCID: PMC5798926
26. Trbovich, M., Ortega, C., Schroeder, J. & Fredrickson, M. (2014). Effect of a cooling vest on core temperature in athletes with and without spinal cord injury. *Top Spinal Cord Inj Rehabil*, 20(1):70-80. doi: 10.1310/sci2001-70. PMID: 24574824; PMCID: PMC3919696
27. Paquette, M., Bieuzen & F., Billaut, F. (2018). Muscle Oxygenation Rather Than VO<sub>2</sub>max as a Strong Predictor of Performance in Sprint Canoe-Kayak. *Int J Sports Physiol Perform*, 1-9. doi: 10.1123/ijsp.2018-0077. Epub ahead of print. PMID: 29745773
28. Boushel, R. & Piantadosi, C. A. (2000). Near-Infrared spectroscopy for monitoring muscle oxygenation. *Scandinavian Physiological Society*, 168(4): 615-622. doi: 10.1046/j.1365-201x.2000.00713.x
29. Stöcker, F., Von Oldershausen, C., Paternoster, F. K, Schulz, T. & Oberhoffer, R. (2016). Relationship of post-exercise muscle oxygenation and duration of cycling exercise. *BMC Sports Sci Med Rehabil*, 8, 9. <https://doi.org/10.1186/s13102-016-0036-y> 3
30. Levine, B. D. (2008). VO<sub>2</sub>max: what do we know, and what do we still need to know? *J Physiol*;586(1):25-34. doi: 10.1113/jphysiol.2007.147629. Epub 2007 Nov 15. PMID: 18006574; PMCID: PMC2375567

31. Saltin, B., Henriksson, J., Nygaard, E., Andersen, P. & Jansson, E. (1977). Fiber types and metabolic potentials of skeletal muscles in sedentary man and endurance runners. *Ann N Y Acad Sci*; 301:3-29. doi: 10.1111/j.1749-6632.1977.tb38182.x. PMID: 73362
32. Crum, E. M., O'Connor, W. J., Van Loo, L., Valckx, M. & Stannard, S. R. (2017). Validity and reliability of the Moxy oxygen monitor during incremental cycling exercise. *Eur J Sport Sci.*; 17(8):1037-1043. doi: 10.1080/17461391.2017.1330899. Epub 2017 May 30. PMID: 28557670
33. Griggs, K. E., Price, M. J. & Goosey-Tolfrey, V. L. (2015). Cooling athletes with a spinal cord injury. *Sports Med*; 45(1):9-21. doi: 10.1007/s40279-014-0241-3. PMID: 25119157
34. Schumacher, Y. O., Ruthardt, S., Schmidt, M., Ahlgrim, C., Roecker, K. & Pottgiesser, T. (2009). Total haemoglobin mass but not cardiac volume adapts to long-term endurance exercise in highly trained spinal cord injured athletes. *Eur J Appl Physiol*; 105(5):779-85. doi: 10.1007/s00421-008-0963-8. Epub 2009 Jan 6. PMID: 19125284
35. Crenshaw, A. G., Elcadi, G. H., Hellstrom, F. & Mathiassen, S. E. (2011). Reliability of near-infrared spectroscopy for measuring forearm and shoulder oxygenation in healthy males and females. *Eur J Appl Physiol*; 112(7):2703-15. doi: 10.1007/s00421-011-2244-1. Epub 2011 Nov 23. PMID: 22109701
36. Kim, J., Kim, M., Ahn, D., & Oh, J. (2019). Comparison of Shoulder Protraction Strength and Electromyography Activity of Serratus Anterior and Pectoralis Major in Subjects With or Without a Winged Scapula, *Journal of Sport Rehabilitation*, 28(3), 272-277. Retrieved Mar 30, 2021, from <http://journals.humankinetics.com/view/journals/jsr/28/3/article-p272.xml>
37. Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) project, retrieved March 30<sup>th</sup>, 2021 from <http://www.seniam.org/>
38. Kounalakis, S. N., Bayios, I. A., Koskolou, M. D., & Geladas, N. D. (2008). Anaerobic Capacity of the Upper Arms in Top-Level Team Handball Players, *International Journal of Sports Physiology and Performance*, 3(3), 251-261. Retrieved Mar 30, 2021, from <http://journals.humankinetics.com/view/journals/ijsp/3/3/article-p251.xml>
39. Noakes, T. D. (2000). Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. *Scand J Med Sci Sports*, 10(3):123-45. doi: 10.1034/j.1600-0838.2000.010003123.x. PMID: 10843507
40. Saltin, B. (1977). The interplay between peripheral and central factors in the adaptive response to exercise and training. *Ann N Y Acad Sci*, 301:224-31. doi: 10.1111/j.1749-6632.1977.tb38201.x. PMID: 270918
41. Clausen, J. P., Klausen, K., Rasmussen, B., Trap-Jensen, J. (1973). Central and peripheral circulatory changes after training of the arms or legs. *Am J Physiol*, 225(3):675-82. doi: 10.1152/ajplegacy.1973.225.3.675. PMID: 4726503
42. Janssen, T. W., Dallmeijer, A. J., Van Der Woude, L. H. (2001). Physical capacity and race performance of handcycle users. *J Rehabil Res Dev.*, 38(1):33-40. PMID: 11322469
43. Costill, D. L., Thomason, H., Roberts, E. (1973). Fractional utilization of the aerobic capacity during distance running. *Med Sci Sports*, 5(4):248-52. PMID: 4774203
44. Stephenson, B. T., Shill, A., Lenton, J., Goosey-Tolfrey, V. (2020). Physiological Correlates to In-race Paratriathlon Cycling Performance. *Int J Sports Med.*, 41(8):539-544. doi: 10.1055/a-1103-2001. Epub 2020 Apr 14. PMID: 32289842
45. Howley, E. T., Bassett, D. R. Jr, Welch, H. G. (1995). Criteria for maximal oxygen uptake: review and commentary. *Med Sci Sports Exerc.*, 27(9):1292-301. PMID: 8531628
46. Dempsey, J. A., Hanson, P. G., Henderson, K. S. (1984). Exercise-induced arterial hypoxaemia in healthy human subjects at sea level. *J Physiol.*, 355:161-75. doi: 10.1113/jphysiol.1984.sp015412. PMID: 6436475; PMCID: PMC1193484

47. Ekblom, B., Wilson, G., Astrand, P. O. (1976). Central circulation during exercise after venesection and reinfusion of red blood cells. *J Appl Physiol.*, 40(3):379-83. doi: 10.1152/jappl.1976.40.3.379. PMID: 931852
48. Hoppeler, H. (1990). The different relationship of VO<sub>2</sub>max to muscle mitochondria in humans and quadrupedal animals. *Respir Physiol.*, 80(2-3):137-45. doi: 10.1016/0034-5687(90)90077-c. PMID: 2218095
49. Huertas, J. R., Ruiz-Ojeda, F. J., Plaza-Díaz, J., Nordsborg, N. B., Martín-Albo, J., Rueda-Robles, A., Casuso, R.A. (2019). Human muscular mitochondrial fusion in athletes during exercise. *FASEB J.*, 33(11):12087-12098. doi: 10.1096/fj.201900365RR. Epub 2019 Aug 9. PMID: 31398297
50. Neuffer, P. D. (1989). The effect of detraining and reduced training on the physiological adaptations to aerobic exercise training. *Sports Med.*, 8(5):302-20. doi: 10.2165/00007256-198908050-00004. PMID: 2692122
51. Holloszy, J. O., Coyle, E. F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *J Appl Physiol Respir Environ Exerc Physiol.*, 56(4):831-8. doi: 10.1152/jappl.1984.56.4.831. PMID: 6373687
52. Henriksson, J., Reitman, J. S. (1977). Time course of changes in human skeletal muscle succinate dehydrogenase and cytochrome oxidase activities and maximal oxygen uptake with physical activity and inactivity. *Acta Physiol Scand.*, 99(1):91-7. doi: 10.1111/j.1748-1716.1977.tb10356.x. PMID: 190867
53. Gollnick, P. D., Armstrong, R. B., Saltin, B., Saubert, C. W. (1973). Effect of training on enzyme activity and fiber composition of human skeletal muscle. *J Appl Physiol.*, 34(1):107-11. doi: 10.1152/jappl.1973.34.1.107. PMID: 4348914
54. Howley, E. T., Bassett, D. R. Jr., Welch, H. G. (1995). Criteria for maximal oxygen uptake: review and commentary. *Med Sci Sports Exerc.*, 27(9):1292-301. PMID: 8531628
55. Houtman, S., Oeseburg, B., Hopman, M. T. (2000). Blood volume and hemoglobin after spinal cord injury. *Am J Phys Med Rehabil.*, 79(3):260-5. doi: 10.1097/00002060-200005000-00008. PMID: 10821312
56. Price, M. J., Campbell, I. G. (2003). Effects of spinal cord lesion level upon thermoregulation during exercise in the heat. *Med Sci Sports Exerc.*, 35(7):1100-7. doi: 10.1249/01.MSS.0000074655.76321.D7. PMID: 12840629
57. Eldahan, K. C., Rabchevsky, A. G. (2018). Autonomic dysreflexia after spinal cord injury: Systemic pathophysiology and methods of management. *Auton Neurosci.*, 209:59-70. doi: 10.1016/j.autneu.2017.05.002. Epub 2017 May 8. PMID: 28506502; PMCID: PMC5677594
58. Perrey, S., Ferrari, M. (2018). Muscle Oximetry in Sports Science: A Systematic Review. *Sports Med.*, 48(3):597-616. doi: 10.1007/s40279-017-0820-1. PMID: 29177977
59. Fortiori Design LLC. (2015). Introduction to muscle oxygen monitoring with Moxy. Retrieved from <http://cdn2.hubspot.net/hub/188620/file-433442739-pdf/docs/moxy-ebook-intro-to-muscle-oxygen.pdf?t=1488816603832>
60. Roca, J., Hogan, M. C., Story, D., Bebout, D. E., Haab, P., Gonzalez, R., Ueno, O. & Wagner, P. D. (1985). Evidence for tissue diffusion limitation of VO<sub>2</sub>max in normal humans. *J Appl Physiol.*, 67(1):291-9. doi: 10.1152/jappl.1989.67.1.291. PMID: 2759955
61. Hopman, M. T., Dueck, C., Monroe, M., Philips, W. T., Skinner, J. S. (1998). Limits to maximal performance in individuals with spinal cord injury. *Int J Sports Med.*, 19(2):98-103. doi: 10.1055/s-2007-971889. PMID: 9562217
62. Stangier, C., Abel, T., Zeller, S., Quittmann, O. J., Perret, C., Strüder, H. K. (2019). Comparison of Different Blood Lactate Threshold Concepts for Constant Load Performance Prediction in Spinal Cord Injured Handcyclists. *Front Physiol.*, 10:1054. doi: 10.3389/fphys.2019.01054. PMID: 31611803; PMCID: PMC6777042

63. Flandrois, R., Grandmontagne, M., Gerin, H., Mayet, M. H., Jehl, J. L., Eyssette, M. (1986). Aerobic performance capacity in paraplegic subjects. *Eur J Appl Physiol Occup Physiol.*, 55(6):604-9. doi: 10.1007/BF00423204. PMID: 3780703
64. Bhambhani, Y. (2002). Physiology of wheelchair racing in athletes with spinal cord injury. *Sports Med.*, 32(1):23-51. doi: 10.2165/00007256-200232010-00002. PMID: 11772160
65. Feldmann, A., Schmitz, R., Erlacher, D. (2019). Near-infrared spectroscopy-derived muscle oxygen saturation on a 0% to 100% scale: reliability and validity of the Moxy Monitor. *J Biomed Opt.*, 24(11):1-11. doi: 10.1117/1.JBO.24.11.115001. PMID: 31741352; PMCID: PMC7003144
66. Lovell, D. I., Mason, D., Delphinus, E., McLellan, C. (2011). A comparison of asynchronous and synchronous arm cranking during the Wingate test. *Int J Sports Physiol Perform.*, 6(3):419-26. doi: 10.1123/ijsp.6.3.419. PMID: 21911866
67. Norton, K. (2018). Standards for Anthropometry Assessment. *Kinanthropometry and Exercise Physiology*. London. <https://doi.org/10.4324/9781315385662>
68. Noordhof, D. A., Skiba, P. F., de Koning, J. J. (2013). Determining anaerobic capacity in sporting activities. *Int J Sports Physiol Perform.*, 8(5):475-82. doi: 10.1123/ijsp.8.5.475. PMID: 24026759
69. Van der Woude, L. H., Bakker, W. H., Elkhuzen, J. W., Veeger, H. E., Gwinn, T. (1997). Anaerobic work capacity in elite wheelchair athletes. *Am J Phys Med Rehabil.*, 76(5):355-65. doi: 10.1097/00002060-199709000-00002. PMID: 9354488
70. Zupan, M. F., Arata, A. W., Dawson, L. H., Wile, A. L., Payn, T. L., Hannon, M. E. (2009). Wingate Anaerobic Test peak power and anaerobic capacity classifications for men and women intercollegiate athletes. *J Strength Cond Res.* 23(9):2598-604. doi: 10.1519/JSC.0b013e3181b1b21b. PMID: 19910814
71. Skattebo, Ø., Calbet, J. A. L., Rud, B., Capelli, C., Hallén, J. (2020). Contribution of oxygen extraction fraction to maximal oxygen uptake in healthy young men. *Acta Physiol (Oxf)*. 230(2):e13486. doi: 10.1111/apha.13486. Epub 2020 May 30. PMID: 32365270; PMCID: PMC7540168
72. Lin, C. W., Huang, C. F., Wang, J. S., Fu, L. L., & Mao, T. Y. (2020). Detection of ventilatory thresholds using near-infrared spectroscopy with a polynomial regression model. *Saudi journal of biological sciences*, 27(6), 1637–1642. <https://doi-org.lib-ezproxy.concordia.ca/10.1016/j.sjbs.2020.03.005>
73. McManus, C. J., Collison, J., & Cooper, C. E. (2018). Performance comparison of the MOXY and PortaMon near-infrared spectroscopy muscle oximeters at rest and during exercise. *Journal of biomedical optics*, 23(1), 1–14. <https://doi-org.lib-ezproxy.concordia.ca/10.1117/1.JBO.23.1.015007>
74. Paquette, M., Bieuzen, F., & Billaut, F. (2019). Sustained Muscle Deoxygenation vs. Sustained High VO<sub>2</sub> During High-Intensity Interval Training in Sprint Canoe-Kayak. *Frontiers in sports and active living*, 1, 6. <https://doi-org.lib-ezproxy.concordia.ca/10.3389/fspor.2019.00006>
75. Paquette, M., Bieuzen, F., & Billaut, F. (2020). Effect of a 3-Weeks Training Camp on Muscle Oxygenation, V̇O<sub>2</sub> and Performance in Elite Sprint Kayakers. *Frontiers in Sports and Active Living*, 2, 47. <https://doi.org/10.3389/fspor.2020.00047>
76. Barstow T. J. (2019). Understanding near infrared spectroscopy and its application to skeletal muscle research. *Journal of applied physiology (Bethesda, Md.: 1985)*, 126(5), 1360–1376. <https://doi-org.lib-ezproxy.concordia.ca/10.1152/jappphysiol.00166.2018>
77. Results Archive Tokyo 2020. *Paralympic.org*. Retrieved on December 23<sup>rd</sup>, 2022. <https://www.paralympic.org/tokyo-2020/results/cycling>

78. Nioka, S., Moser, D., Lech, G., Evengelisti, M., Verde, T., Chance, B., & Kuno, S. (1998). Muscle deoxygenation in aerobic and anaerobic exercise. *Advances in experimental medicine and biology*, 454, 63–70. [https://doi-org.lib-ezproxy.concordia.ca/10.1007/978-1-4615-4863-8\\_8](https://doi-org.lib-ezproxy.concordia.ca/10.1007/978-1-4615-4863-8_8)