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ORIGINAL ARTICLE

The effect of HIIT vs. SIT on muscle oxygenation in trained sprint kayakers

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Abstract

Purpose To assess the performance change and physiological adaptations following nine sessions of short high-intensity interval training (HIIT) or sprint-interval training (SIT) in sprint kayakers.

Methods Twelve trained kayakers performed an incremental test and 3 time trials (200 m, 500 m and 1000 m) on a kayak ergometer. Oxygen consumption ($\dot{V}O_2$) and muscle oxygenation of the latissimus dorsi, biceps brachii, and vastus lateralis were measured. Athletes were then paired for sex and $\dot{V}O_2$max and randomized into a HIIT or a SIT training group, and performed nine training sessions before repeating the tests.

Results Training improved performance in HIIT (200 m: $+3.8 \pm 3.1\%$, $p = 0.06$; 500 m: $+2.1 \pm 4.1\%$, $p = 0.056$; 1000 m: $+3.0 \pm 4.6\%$, $p = 0.13$) but changes in performance remained within the smallest worthwhile change in SIT (200 m: $+0.8 \pm 4.1\%$, $p = 0.59$; 500 m: $+0.5 \pm 4.1\%$, $p = 0.87$; 1000 m: $+1.3 \pm 4.6\%$, $p = 0.57$). In the 1000 m, training led to a greater deoxygenation in the biceps brachii and vastus lateralis in HIIT, and in the latissimus dorsi in SIT. In HIIT, the best predictors of improvements in 1000 m performance were increases in latissimus dorsi and vastus lateralis maximal deoxygenation.

Conclusion In a group of trained sprint kayakers, greater improvements in performance can be obtained with HIIT compared with SIT, for any distance. Training did not change $\dot{V}O_2$peak, but increased muscle maximal deoxygenation, suggesting both HIIT and SIT elicit peripheral adaptations. Performance improvement in the 1000 m was associated with increased maximal muscle deoxygenation, reinforcing the contribution of peripheral adaptations to performance in sprint kayaking.

Keywords Oxygen saturation · Peripheral adaptations · Aerobic fitness · Sprint kayak · Interval training

Abbreviations

HIIT	High-intensity interval training
SIT	Sprint interval training
$\dot{V}O_2$	Oxygen consumption
$\dot{V}O_2$max	Maximal oxygen consumption
NIRS	Near-infrared spectroscopy
TT	Time-trial
MAP	Maximal aerobic power
RPE	Rate of perceived exertion
sRPE	Session rate of perceived exertion
AU	Arbitrary units
SmO_2	Muscle O_2 saturation
total[heme]	Total heme concentration
ΔSmO_2min	Maximal deoxygenation
Δtotal[heme]	Change in total heme concentration
ES	Effect size
SWC	Smallest worthwhile change
oxy[heme]	Oxyhemoglobin and oxymyoglobin concentration
deoxy[heme]	Deoxyhemoglobin and deoxymyoglobin concentration

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Introduction

Interval training, which involves alternating short-to-long bouts (seconds to minutes) of high-intensity (usually above second ventilatory threshold) exercise with recovery periods (Weston et al. 2014), is considered one of the most effective training for improving performance in athletes from various sports (Buchheit and Laursen 2013). Interval training is frequently divided into two broad categories: high-intensity interval training (HIIT) and sprint-interval training (SIT). While there is yet to be a consensus on the exact definition of these terms, HIIT usually refers to “repeated bouts of exercise that occur at a power output or velocity within the severe-intensity domain, which occurs between the second ventilatory threshold and maximal oxygen consumption ($\dot{V}O_2$max)” (Rosenblat et al. 2020). While some authors define SIT as an interval training session involving repeated bouts of exercise performed in the extreme exercise domain (above power output or velocity associated with $\dot{V}O_2$max) (Rosenblat et al. 2020), it is generally admitted that SIT involves bouts of exercise far more intense, usually > 150% of power output associated with $\dot{V}O_2$max (Talanian 2016).

Both HIIT and SIT can produce physiological adaptations that are beneficial for endurance performance in trained athletes (Buchheit and Laursen 2013; Gist et al. 2014). A recent meta-analysis (Rosenblat et al. 2020) comparing their respective effects concluded that HIIT that includes work bouts between 4 and 6 min generally leads to greater increases in time-trial performance compared with SIT. However, most studies used medium (2–4 min) or long (4–6 min) HIIT and only one compared SIT to short (< 2 min) HIIT. Moreover, most studies used long (> 30 min) time trials in running or cycling as measures of endurance performance. One study used a 2-km rowing time trial and reported SIT to increase TT performance similarly to HIIT. To date, while the response to different types of interval training has been well studied for cycling and running, and for longer endurance performance, little is known about the comparative physiological and performance enhancing effects of short HIIT and SIT in sports that involve shorter race durations and where upper-body muscles are the principal propulsive muscles, like sprint kayaking.

In sprint kayaking, Olympic individual events are 200-m and 500-m (~ 38 to ~ 120 s) for women and 200-m and 1000-m (~ 34 to ~ 220 s) for men. The shorter race durations and reliance on upper body muscles, which typically display larger cross-sectional area of type II muscle fibers than lower-body muscles, increase anaerobic contribution in sprint kayak events, when compared with endurance sports. Furthermore, while $\dot{V}O_2$max is often considered a major performance factor in longer distance sprint canoe-kayak events (Michael et al. 2008), results from a recent study suggest that peripheral adaptations, as assessed via near-infrared spectroscopy (NIRS) derived changes in muscle oxygenation, may be stronger predictors of canoe-kayak performance in both short and long events (Paquette et al. 2018). According to that study, in a group of 30 trained kayakers, 90% of the variance in 200-m performance was explained by the maximal change in deoxyhemoglobin and deoxymyoglobin (deoxy[heme]) and the maximal change in muscle oxygenation (SmO_2) during the time trial, and 71% of the variance in 500-/1000-m performance was explained by the maximal change in deoxy[heme] in three active muscles (Paquette et al. 2018). Therefore, training sessions that elicit peripheral adaptations associated with O_2 extraction may be critical in this sport to enhance fitness and performance. In this perspective, an acute short HIIT session has recently been associated with longer time spent near $\dot{V}O_2$max, while a single SIT session led to the greatest levels of muscle deoxygenation (Paquette et al. 2019). Interestingly, it has been suggested that repeated fluctuations of muscular O_2 consumption during training sessions are necessary to induce muscular oxidative capacity adaptations (Daussin et al. 2008a, b) and that low O_2 partial pressure at the cellular level is needed to induce mitochondrial biogenesis (Hoppeler et al. 2003). Hence, we argue that training sessions that elicit repeated high levels of muscle deoxygenation might present an adequate stimulus to elicit peripheral adaptations. Therefore, it may be hypothesized that SIT would be an ideal training stimulus, when compared with HIIT, to induce peripheral adaptations that would lead to improved performance in trained sprint kayakers.

The purpose of this study was to assess the effect of nine sessions of either short HIIT or SIT on oxygen consumption ($\dot{V}O_2$), muscle oxygenation and performance in 200-m, 500-m and 1000-m time trials in trained kayakers, and to assess the associations between training-induced changes in physiology and performance.

Materials and methods

Subjects

Twelve trained sprint kayakers participated in this study (women, $n = 4$). Participants were 21 ± 3 years of age (range 18–27 years old) and weighted 73.9 ± 5.9 kg. Five of them were members of the Canadian National Team, four were members of a provincial team and three were club-level athletes. Subjects were recruited during their general preparatory phase, in the fall, where training is focused on base endurance (kayaking, swimming, running, cycling) and strength training. This study was approved by the ethical committee of Laval University and was conducted in accordance with the principles established in the Declaration of Helsinki, with verbal and written informed consent obtained from all participants.

Experimental design

Athletes performed a maximal incremental test on a kayak ergometer and time trials (TT) over the three racing distances of 200, 500 and 1000 m on the same kayak ergometer. Athletes were then paired for sex and $\dot{V}O_2$max and randomized into two training groups: a high-intensity interval training group (HIIT) and a sprint-interval training group (SIT). Both groups performed nine interval training sessions over 4 weeks, followed by a 4-day recovery period where load was reduced to allow athletes to recover before undertaking the incremental test and TTs again. Athletes were instructed to replace their usual high-intensity training sessions by the kayak ergometer interval sessions, and to perform their habitual strength training and low intensity aerobic activity. Previous studies have reported increases in $\dot{V}O_2$max and performance with 4 weeks of training with 2 to 3 HIIT or SIT sessions per week, suggesting the current design is an appropriate stimulus for adaptation (Rosenblat et al. 2020).

Procedures

Incremental test and time trials

Prior to and following the training program, all participants participated in two testing sessions. On the first testing session, they performed an incremental test followed by a 1000-m TT, to determine thresholds, VO_2max and maximal aerobic power (MAP). Then, 24 to 96 h later, the second testing session consisted in a 200-m, followed by a 20-min break and a 500-m TT. All tests were performed on a kayak ergometer (SpeedStroke Gym, KayakPro, Florida, USA). All participants but one were familiar with the kayak ergometer as they used it regularly to train during the winter months. The participant who was not familiar with the kayak ergometer participated in two familiarization sessions to rehearse the TTs before participating in the study to minimize the risk of a learning effect. The ergometer was calibrated before each test, according to the manufacturer recommendations and tension in the ergometer's ropes was verified regularly (Tanner and Gore 2013). Power output and stroke rate were recorded on a computer, using the eMonitorPro2 software (KayakPro, Florida, USA). During all sessions, expired air was continuously recorded using a breath-by-breath gas analyzer (Vmax Encore metabolic cart, CareFusion Corp, California, USA) and heart rate was measured using a chest strap and watch (Polar H10, Polar Electro, Kempele, Finland).

The incremental test consisted of 4-min stages of increasing intensity, with an initial power output of 50 W for men and 40 W for women, and an increase of 20 W/stage for men and 15 W/stage for women. Participants received stroke-by-stroke feedback during the test and were asked to maintain the target power output during the whole 4-min stage. There was a 1-min break between stages where athletes stopped paddling and a blood sample was collected on the earlobe using a portable lactometer (The Edge Handheld Lactate Analyser, Woodley Equipment Company LTD, Lancashire, United Kingdom) to measure blood lactate concentration. Asepsis was performed with 70% ethylic solution on the distal portion of the earlobe prior to collection, and puncture was performed using disposable lancets. Power output and heart rate at blood lactate concentrations of 2 mmol and 4 mmol were determined using the Lactate-E software (Newell et al. 2007). The incremental test was ended when the athlete reached 4 mmol of lactate or higher, and was followed by a 20-min break, where athletes rested and then reactivated before performing the 1000-m TT. $\dot{V}O_2$max was the highest $\dot{V}O_2$ over a period of 30-s during the 1000-m TT, and MAP was the average power output over the 1000-m TT. This incremental testing protocol is frequently used by kayakers and has been described previously (Tanner and Gore 2013). On the second testing sessions, participants performed their habitual pre-race warm-up, and then did a 200-m TT, followed by a 20-min break, where they rested and reactivated, before performing the 500-m TT. Blood lactate concentration was measured 1, 3 and 5 min after the end of each TT and peak lactate concentration was defined as the highest of the three measurements. $\dot{V}O_2$peak for each TT was defined as the highest $\dot{V}O_2$ value achieved over a 30-s period during the test.

Muscle oxygenation

During all tests, NIRS monitors (Moxy monitor, Fortiori Design, Minnesota, USA) were used to assess changes in muscle oxygenation. The Moxy monitor employs four wave-lengths 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). Moxy monitors were placed on three active muscles: *latissimus dorsi*—midpoint between the inferior border of the scapula and posterior axillar fold (Borges et al. 2015)—, *biceps brachii*—middle of the BB muscle belly (8 to 12 cm above the elbow fold) —and *vastus lateralis*—distal part of the VL muscle belly (10 to 15 cm above the proximal border of the patella) (Billaut and Buchheit 2013). NIRS monitors were placed on the athlete's dominant side, parallel to the muscle fiber orientation. The skin at the location of probe placement was shaved when necessary. The NIRS monitors were attached and secured with a double-sided adhesive disk and an adhesive patch, and covered by a dark bandage, using the lightest pressure necessary to eliminate probe movement while not compressing the skin to reduce the intrusion of extraneous light. The monitors' position was recorded and replicated during following testing sessions. Skinfold thickness at the sites of NIRS measurement was measured using the thickness measured by a skinfold caliper (Harpenden Ltd) divided by 2. The raw muscle O_2 saturation (SmO_2) signal, which represents the balance between O_2 delivery and extraction by the muscle, and total heme concentration (total[heme]), an indicator of local blood volume (Ferrari et al. 2011), were visually assessed to remove outliers and treated using a rolling average to reduce the noise created by movement. Baseline SmO_2 and baseline total[heme] were computed as a 30-s average when subjects were seated still on the ergometer before the beginning of the test. In the incremental test, ΔSmO_2 was the difference between SmO_2 over the last minute of the stage and baseline SmO_2. As described elsewhere (Paquette et al. 2018), minimum SmO_2 (SmO_2min) was the lowest 5-s average SmO_2 reached during the TT. In the TT, maximal deoxygenation (ΔSmO_2min) and Δtotal[heme] were calculated as the difference between SmO_2min and baseline SmO_2, and the difference between total[heme] in the last 5-s of the effort and baseline total[heme]. At the onset of each TT, a linear model was used to assess rapid changes in SmO_2 (Bae et al. 2000). The SmO_2 versus time values were modeled from the start to the 12th second of exercise (Buchheit et al. 2012). The slope of the relationship was retained as an index of deoxygenation rate (deoxy rate) (Bae et al. 2000). During high-intensity exercise, muscle oxygenation decreases rapidly in the first seconds of exercise (phase I), then levels off for the last part of the effort (phase II) (Bae et al. 2000). Therefore, the phase II slope corresponded to the slope of the SmO_2 vs. time relationship during the second phase, from the 12th second to the end of the TT. Similarly, reoxygenation rate (reoxy rate) was taken as the slope of the linear part of the SmO_2 vs. time curve, within the first 30-s period following the end of exercise (Buchheit and Ufland 2011). Reoxygenation rate is recognized as an indicator of the muscle oxidative capacity, since it is related to both phosphocreatine restoration (Ryan et al. 2013) and changes in muscle oxidative enzyme concentrations (Puente-Maestu et al. 2003).

Training program

After initial testing, participants were paired for sex and VO_2max and randomized into the two training groups. Table 1 details the interval training sessions performed by both groups over the 4-week period. HIIT performed short interval training (15-s or 30-s efforts) at 110% maximal aerobic power (MAP), with 1:1 effort:rest ratio and active recovery at 50%MAP. SIT performed 150 m (~30 s) to 200 m (~40 s) all-out efforts, with long passive recovery between efforts. All sessions were performed on a kayak ergometer. The subjects were instructed to perform the nine training sessions over a 4-week period and to leave a minimum of 48 h and a maximum of 96 h between sessions. Sessions were not matched for volume or work, but were developed to be representative of sessions that would be programmed by kayak coaches.

Training load was recorded by self-reported questionnaire during the 4-week training period. Every evening, athletes filled an online questionnaire where they had to answer three questions for every training session performed on that day: session type (kayak, strength training or other

Table 1 Training sessions performed by HIIT and SIT groups

Session	HIIT						SIT					
	Sets	Reps/set	Work intervals (s)	Rest intervals (s)	Work intensity	Rest intensity	Sets	Reps/set	Work intervals (m)	Work + rest intervals (min)	Work intensity	Rest intensity
1	2	20	15	15	110% MAP	50% MAP	1	4	200	6	All-out	Passive
2	2	10	30	30			1	6	150	5		
3	2	20	15	15			1	5	200	6		
4	2	10	30	30			1	5	200	6		
5	3	16	15	15			1	7	150	5		
6	3	8	30	30			1	6	200	6		
7	3	20	15	15			1	7	200	6		
8	3	10	30	30			1	8	150	5		
9	3	20	15	15			1	7	200	6		

HIIT high-intensity interval training group, *SIT* sprint-interval training group, *MAP* maximal aerobic power cardio exercise), session duration (in min), and rate of perceived exertion (RPE, modified Borg scale) (Foster et al. 2001). Evening questionnaires were chosen for simplicity and because it was suggested that recall time had no effect on post-exercise RPE values in a study performed with top-class soccer referees (Castagna et al. 2017). For each training session, session RPE (sRPE) was calculated, as the product of session duration and RPE (Foster et al. 2001), in arbitrary units (AU). Weekly load was then computed as the sum of sRPE for the week, for each training session type. Weekly volume was the total training duration, in hours. Average RPE was calculated for each training week and considered as an index of training intensity. Athletes also completed a questionnaire every night to assess their subjective wellness (Hooper and Mackinnon 1995), where fatigue, soreness, sleep quality, motivation and health were rated on a 1 to 7 scale where 1 represents the worse situation (i.e. high fatigue, or low motivation). Average score was the average of the 5 scores for the day. Athletes also logged their sleep duration. Changes in subjective scores ratings following interval training days were calculated as the difference between scores logged on days where interval training sessions were performed minus the scores on the previous evening.

Statistical analysis

Means and standard deviations were calculated for performance and physiological parameters in pre- and post-training trials. Independent *t* test was used to assess the difference between groups for subjects' characteristics (age, weight, VO_2max) and training load. Paired *t* test was used to assess the effect of training in each group. Independent *t* test on change in parameters with training (absolute deltas) was used to assess the difference in training-induced change in performance and physiology between groups. An alpha level of 0.05 was used. The TTs were divided into 50-m segments and average power, stroke rate, SmO_2, total[heme] and VO_2 over these segments were compared between pre- and post-training trials in both groups using paired *t*-test. Cohen's *d* effect sizes (ES) and 90% confidence intervals were also computed for differences in means between pre- and post-training measures and for differences in means between training related change for HIIT and SIT. ES of 0.2, 0.6, 1.2, and 2.0 were considered small, moderate, large and very large differences, respectively. Pearson correlations were calculated to assess associations between pre-training characteristics and performance improvements. Correlation coefficients of >0.1, >0.3, >0.5 and >0.7 were considered small, moderate, large and very large (Hopkins et al. 2009). Stepwise multiple regression analyses were performed to determine the best possible combination of physiological variables to predict change in TT performance.

Results

Subjects characteristics

There were no differences between groups for subjects' age (HIIT: 21 ± 3; SIT: 22 ± 4 years, $t = -0.91$, $p = 0.38$), weight (HIIT: 73.2 ± 3.0; SIT: 74.6 ± 5.9 kg, $t = -0.37$, $p = 0.72$) and VO_2max (HIIT: 54.1 ± 9.3; SIT: 55.8 ± 6.1 mL/kg/min, $t = -0.39$, $p = 0.71$). Skinfolds thicknesses in HIIT were 1.7 ± 0.7 mm, 3.8 ± 1.8 mm and 5.7 ± 4.2 mm for the biceps brachii, latissimus dorsi and vastus lateralis, respectively, and were 2.6 ± 1.6 mm, 4.3 ± 2.2 mm and 5.6 ± 4.1 mm for the three muscles in SIT.

Change in time-trial performance

Figure 1 shows individual responses for change in performance with training. There were no differences in performance before training between groups for the 1000 m ($t = 0.92$, $p = 0.38$), 500 m ($t = 0.87$, $p = 0.41$) and 200 m ($t = 0.51$, $p = 0.63$). Performance in the 1000-m TT improved from 4:42.2 ± 0:33.7 min to 4:30.3 ± 0:18.8 min in HIIT (range: −41.9 to +1.7 s, ES: −0.44 [−1.40, 0.53], $t = 1.84$, $p = 0.13$) and from 4:27.1 ± 22.3 min to 4:25.1 ± 24.8 min in SIT (range: −9.9 to +12 s, ES: −0.08 [−1.03, 0.87], $t = 0.61$, $p = 0.57$). Performance in the 500-m improved from 2:10.0 ± 0:11.3 min to 2:07.3 ± 0:11.9 min in HIIT (range:

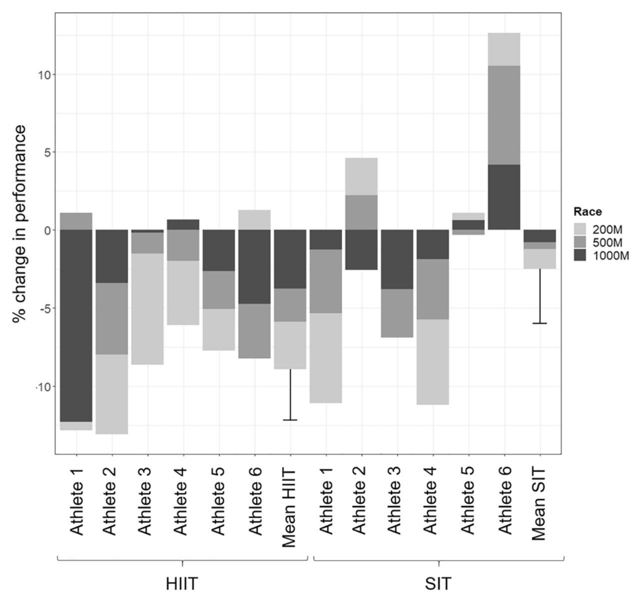


Fig. 1 Individual change in performance in the 200-, 500- and 1000-m TTs. Each bar represents the change in performance of an individual athlete. Mean HIIT and Mean SIT represent average change in performance for athletes in HIIT and SIT groups, respectively. Error bars represent group SD

−6.2 to +1.6 s, ES: −0.24 [−1.19, 0.21], $t=2.42$, $p=0.056$) and from 2:05.0 ± 0:08.4 min to 2:04.6 ± 0:12.7 min in SIT (range: −4.8 to +8.3 s, ES: −0.03 [−0.98, 0.92], $t=0.58$, $p=0.87$). Performance in the 200-m improved from 46.1 ± 4.5 s to 44.7 ± 5.3 s in HIIT (range: −3.1 to +0.6 s, ES: −0.27 [−1.22, 0.68], $t=2.42$, $p=0.06$) and from 44.8 ± 3.8 s to 44.3 ± 5.4 s in SIT (range: −2.4 to +1.2 s, ES: −0.10 [−1.05, 0.85], $t=0.58$, $p=0.59$). There was a moderate difference between groups for change in performance in favor of HIIT over SIT in the 1000-m (ES: −0.79 [−1.78, 0.19], $t=-1.37$, $p=0.22$), and a small difference for the 500-m (ES: −0.57 [−1.54, 0.40], $t=-0.99$, $p=0.40$) and the 200-m (ES: −0.56 [−1.53, 0.40], $t=-0.91$, $p=0.44$).

There was an almost perfect relationship between performance in the 1000-m TT pre-training and training related change in performance in HIIT ($R=-0.97$, $p=0.0013$), where athletes with the slowest performance before training improved the most with training, but not in SIT ($R=0.16$, $p=0.76$). On the contrary, in the 200-m TT, a faster performance pre-training was associated with a larger improvement with training, and this association was stronger in SIT ($R=0.83$, $p=0.081$) than HIIT ($R=0.55$, $p=0.26$). In the 500-m TT, there were no associations between initial performance and training-related change in performance ($R=0.32$, $p=0.32$).

Training load and subjective response to training

RPE for kayaking interval sessions was higher in SIT (9.0 ± 0.8 [range 8 to 10]) than HIIT (8.2 ± 0.8 [range 7 to 10], $p<0.01$). However, changes in subjective score ratings following interval training days were not different between groups (Table 2A). Over the 4-week training period, sleep quality, motivation, health and average subjective scores were lower in HIIT compared with SIT (Table 2B). These discrepancies in subjective scores between groups manifested in the second week of training, and in the last days of training, before the start of the recovery period.

Table 2 Average values for subjective scores during the 4-week training period in HIIT and SIT groups

	A. Change following interval sessions		B. Average of training period	
	HIIT	SIT	HIIT	SIT
Fatigue (/7)	0.1 ± 1.5	0.2 ± 1.2	4.1 ± 1.2	4.2 ± 1.4
Soreness (/7)	−0.2 ± 1.5	0.1 ± 1.8	4.4 ± 1.3	4.3 ± 1.4
Sleep quality (/7)	0.0 ± 1.2	0.4 ± 1.5	5.1 ± 1.4*	5.5 ± 1.4
Sleep duration (h)	0.1 ± 1.8	0.3 ± 1.4	7.9 ± 1.5	7.8 ± 1.1
Motivation (/7)	−0.2 ± 1.3	−0.1 ± 0.6	5.0 ± 1.3*	6.2 ± 1.2
Health (/7)	0.0 ± 0.7	0.0 ± 0.4	5.6 ± 1.2*	6.1 ± 1.3
Average scores (/7)	−0.1 ± 0.6	0.1 ± 0.6	4.8 ± 0.8*	5.3 ± 0.8

HIIT high-intensity interval training group, *SIT* sprint-interval training group

*$p<0.05$ compared with SIT; A. difference between score on the evening after the interval training was performed and the score of the previous evening; B. mean score over the 4-week training period

Figure 2 shows total training load performed by all subjects and average training load for both training groups for the different training modalities. Total training load during the 4-week training period was 10,390 ± 1361 AU in HIIT and 15,329 ± 6298 AU in SIT ($p=0.11$ between groups). While training load performed in kayaking (HIIT: 3331 ± 504 AU, SIT: 5273 ± 3432 AU, $p=0.23$) or in other cardio activities (HIIT: 1973 ± 837 AU, SIT: 2890 ± 973 AU, $p=0.11$) was not different between groups, strength training load was higher in SIT (8599 ± 2118 AU) compared with HIIT (5086 ± 1830 AU, $p=0.02$). While training load was higher in members of the National team (13,517 ± 7508 AU) and provincial team (14,530 ± 2568 AU) compared to club-level athletes (10,784 ± 2189 AU), this difference did not reach statistical significance (group effect, $p=0.469$). There were no associations between total training load (sRPE) over the study duration and change in performance in the 1000 m ($R=0.36$, $p=0.35$), but moderate associations between training load and both 500 m ($R=0.52$, $p=0.07$), and 200 m TT ($R=0.47$, $p=0.09$) that did not reach statistical significance. In both cases, a lower total training load was associated with a larger increase in performance.

Incremental test

Table 3 displays physiological parameters from the incremental test before and after training for both groups. $\dot{V}O_2$max, HRmax and thresholds did not change with

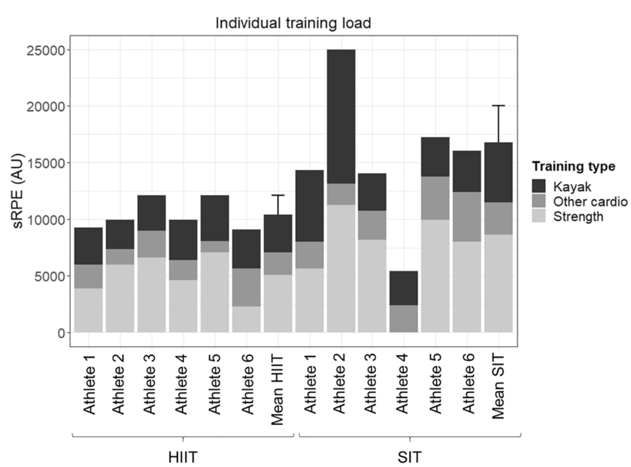


Fig. 2 Total training load by training type for all individuals and group averages for the 4-week training period. Each bar represents the total training load of an athlete over the 4-week training period. Mean HIIT and Mean SIT represent average training load for athletes in HIIT and SIT groups, respectively. Error bars represent group SD

Table 3 Parameters from the incremental test pre- and post-training in HIIT and SIT

	HIIT		SIT	
	Pre	Post	Pre	Post
VO_2max (mL/kg/min)	3.9 ± 0.7	3.9 ± 0.6	4.2 ± 0.7	4.2 ± 0.7
VO_2max (L/min)	54.1 ± 9.3	53.6 ± 8.3	55.8 ± 6.1	55.7 ± 6.8
HRmax (bpm)	192 ± 6	192 ± 6	190 ± 8	189 ± 8
MAP (W)	136 ± 41	146 ± 26	153 ± 31	156 ± 32
[Lactate] = 2 mmol				
Power (W)	57 ± 14	58 ± 23	66 ± 17	72 ± 13
%MAP	43 ± 13	39 ± 17	43 ± 7	47 ± 10
Heart rate (bpm)	159 ± 5	161 ± 11	154 ± 9	155 ± 7
%HRmax	82 ± 2	83 ± 5	81 ± 3	82 ± 3
[Lactate] = 4 mmol				
Power (W)	84 ± 14	87 ± 14	95 ± 19	98 ± 16
%MAP	66 ± 18	60 ± 10	43 ± 4	47 ± 8
Heart rate (bpm)	174 ± 9	175 ± 13	167 ± 8	168 ± 8
%HRmax	91 ± 4	91 ± 5	88 ± 3	89 ± 2

HIIT high-intensity interval training group, *SIT* sprint-interval training group, *VO_2max* maximal oxygen consumption, *HRmax* maximal heart rate, *MAP* maximal aerobic power

$^{\$}p$ between 0.05 and 0.10 compared with SIT

$^{*}p < 0.05$ compared with SIT

training in either group. Figure 3 shows ΔSmO_2 for the three muscles during the incremental test before and after training. ΔSmO_2 in submaximal stages was greater (lower deoxygenation) after training in HIIT in all muscles, but did not change with training in SIT. There was a group effect for change in ΔSmO_2 in the latissimus dorsi ($p = 0.04$), the group effect did not reach statistical significance for the biceps brachii ($p = 0.08$) and there was no group effect for the vastus lateralis ($p = 0.78$).

Physiological response to time trials

Tables 4, 5 and 6 and Figs. 4, 5 and 6 display the muscle oxygenation response to the 1000-m TT, 500-m TT and 200-m TT, respectively, before and after training.

Table 7 depicts VO_2peak and blood lactate concentrations for the three TTs. There was no change in VO_2peak with training in any group during the 500-m and 1000-m TT. VO_2peak was lower post-training in the 200-m TT in SIT, and VO_2peak response to training was different between groups.

Predicting change in performance

The best stepwise multiple regression equation for predicting 1000-m performance improvement in both groups was Δ1000 m performance (s) = 0.48 × $\Delta(\Delta SmO_2$min, latissimus dorsi) + 0.53 × $\Delta(\Delta SmO_2$min, vastus lateralis) + 0.04. This equation yielded an R^2 value of 0.65, a standard error of the estimate (SEE) of 7.67 s and a p value of 0.004. In SIT, the best equation was Δ1000 m performance (s) = − 29.14 × ΔVO_2max (L/min) − 1.12 × ΔPeak lactate concentration + 0.17, $R^2 = 0.94$, SEE = 1.86 s, $p = 0.006$. In HIIT, the best equation was Δ1000 m performance (s) = 0.64 × $\Delta(\Delta SmO_2$min, latissimus dorsi) + 0.45 × $\Delta(\Delta SmO_2$min, vastus lateralis) − 3.17, $R^2 = 0.87$, SEE = 5.7 s, $p = 0.022$.

The best stepwise multiple regression equation for predicting 500-m performance improvement in both groups was Δ500 m performance (s) = − 18.67 × Δ(phase II slope, latissimus dorsi) × 1.34, $R^2 = 0.492$, SEE = 2.99 s, $p = 0.014$. In SIT, the best equation was Δ500 m performance (s) = − 0.39 × ΔPeak lactate concentration − 4.74 × Δ(Reoxy rate, vastus lateralis) − 9.08 × Δ(phase II slope, latissimus dorsi), $R^2 = 0.998$, SEE = 0.22 s, $p = 0.001$. In HIIT, the best equation was Δ500 m performance (s) = 2.14 × Reoxy rate (biceps brachii) − 0.34 × Deoxy rate (biceps brachii) − 3.85, $R^2 = 0.98$, SEE = 0.29 s, $p = 0.012$.

In SIT or when pooling both groups, there were no multiple regression equations that could help predicting change in performance from the available physiological variables. The best stepwise multiple regression equation for predicting 200-m performance improvement in HIIT was Δ200 m performance (s) = − 6.77 × $\Delta(\Delta$total[heme], vastus lateralis) − 1.91, $R^2 = 0.71$, SEE = 0.75 s, $p = 0.019$.

Discussion

The goal of this study was to compare the effects of a HIIT and a SIT training program on performance and physiology, especially muscle oxygenation parameters, in trained kayakers. We had hypothesized that SIT would induce greater peripheral adaptations, and would therefore lead to greater increases in performance when compared with short HIIT. We rather found that: (1) HIIT led to greater improvements in performance in the 200-m, 500-m and 1000-m TT compared with SIT; (2) HIIT induced lower muscle deoxygenation at submaximal intensity in all three investigated muscles; (3) training led to a greater deoxygenation in the biceps brachii and vastus lateralis during the 1000-m in HIIT, and to a greater deoxygenation in the latissimus dorsi during the 1000-m in SIT, but did not change muscles maximal deoxygenation in the 500-m TT or 200-m TT; (4) the best predictors of the increase in 1000-m performance were training-related increases in maximal deoxygenation in the latissimus dorsi and vastus lateralis.

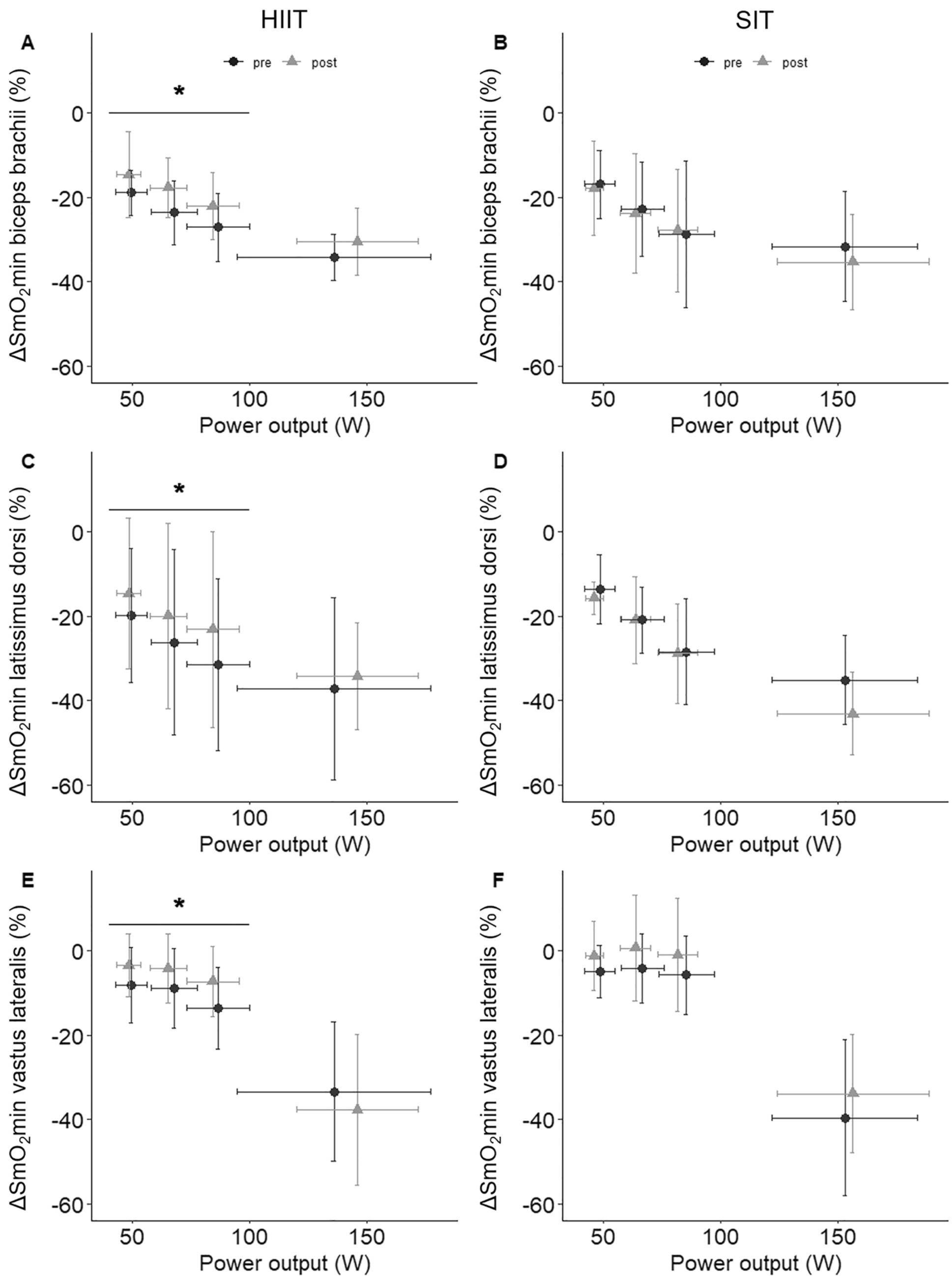


Fig. 3 SmO_2 in the last minute of each stage of the incremental test and the 1000-m TT before and after training in HIIT and SIT. *$p < 0.05$ compared with pre-training; **A** HIIT, ΔSmO_2 of the bieps brachii; **B** SIT, ΔSmO_2 of the bieps brachii; **C** HIIT, ΔSmO_2 of the latissimus dorsi; **D** SIT, ΔSmO_2 of the latissimus dorsi; **E** HIIT, ΔSmO_2 of the vastus lateralis; **D** SIT, ΔSmO_2 of the vastus lateralis. First 3 points represent the last minute of the submaximal stages of the incremental test; last point represents the last minute of the 1000-m TT

Training-induced changes in performance

Performance improved from 2.1 to 3.8% in HIIT, while it improved by 0.5% to 1.3% in SIT. The typical variability of performance for top athletes in sprint canoe-kayak over a season is 1.0% (Malcata and Hopkins 2014) and so increases in performance were larger than the season variability following HIIT, but not SIT. Performance improvements in SIT were smaller compared to the increases of 1.4% to 4.3% reported in previous studies among trained rowers, runners and cyclists undertaking 3 to 6 weeks of SIT (Laursen et al. 2002; Esfarjani and Laursen 2007; Akca and Aras 2015). In HIIT, performance improvements were similar to what has been reported following 3 to 6 weeks of HIIT in a trained population (~3.7%, range 0.3 to 7.4%, Rosenblat et al. 2020).

In the current study, HIIT led to increases in performance that were 2 to 5 times bigger compared with SIT, the largest difference being for the 1000-m. In their recent meta-analysis, Rosenblat et al. (2020) compared the effect of SIT and HIIT on TT performance and concluded that long HIIT was superior to SIT to improve performance over long TT (> 35 min). Only one study had compared SIT to short HIIT in trained athletes, on a very small group of subjects, and found a 3-week SIT program to improve cycling 40-km TT

Table 4 Muscle oxygenation response to the 1000-m time trial before and after training

	HIIT			SIT			Group difference
	Pre	Post	ES	Pre	Post	ES	ES
ΔSmO_2min—biceps brachii	−50.9 ± 9.1	−59.0 ± 9.1*	**−0.97 [−1.97, 0.04]**	−51.8 ± 12.1	−54.8 ± 9.7	−0.27 [−1.22, 0.68]	**−1.04 [−2.05, −0.03]**
ΔSmO_2min—latissimus dorsi	−56.7 ± 15.1	−58.5 ± 6.5	−0.16 [−1.11, 0.80]	−55.7 ± 12.3	−62.9 ± 9.8	−0.65 [−1.63, 0.32]	0.38 [−0.58, 1.34]
ΔSmO_2min—vastus lateralis	−51.6 ± 15.5	−68.5 ± 9.3$	**−1.32 [−2.37, −0.28]**	−56.1 ± 17.5	−57.5 ± 11.9	−0.09 [−1.04, 0.86]	**−1.09 [−2.10, −0.07]**
Δtotal[heme]—biceps brachii	0.3 ± 0.1	0.2 ± 0.2	−0.38 [−1.34, 0.58]	0.2 ± 0.3	0.2 ± 0.3	0.07 [−0.88, 1.02]	−0.38 [−1.34, 0.58]
Δtotal[heme]—latissimus dorsi	0.0 ± 0.3	0.1 ± 0.2	0.51 [−0.46, 1.47]	0.1 ± 0.3	−0.1 ± 0.2	−0.64 [−1.61, 0.34]	**1.11 [0.09, 2.13]**
Δtotal[heme]—vastus lateralis	0.0 ± 0.3	−0.1 ± 0.2	−0.30 [−1.26, 0.65]	−0.2 ± 0.4	−0.1 ± 0.3	0.20 [−0.75, 1.16]	−0.77 [−1.75, 0.22]
Deoxy rate—biceps brachii (%/s)	−3.1 ± 1.5	−4.0 ± 1.4	−0.61 [−1.58, 0.36]	−3.6 ± 1.5	−4.7 ± 2.4	−0.57 [−1.53, 0.40]	0.12 [−0.83, 1.07]
Deoxy rate—latissimus dorsi (%/s)	−3.1 ± 1.9	−3.9 ± 2.1	−0.42 [−1.38, 0.54]	−4.0 ± 1.6	−3.4 ± 1.7	0.41 [−0.55, 1.37]	**−1.12 [−2.14, −0.1]**
Deoxy rate—vastus lateralis (%/s)	−1.9 ± 1.8	−2.5 ± 1.7	−0.33 [−1.28, 0.63]	−1.3 ± 0.9	−1.6 ± 1	−0.30 [−1.25, 0.66]	−0.40 [−1.36, 0.56]
Slope phase II—biceps brachii (%/s)	−0.017 ± 0.033	0.005 ± 0.038*	0.62 [−0.35, 1.59]	−0.003 ± 0.036	−0.015 ± 0.039	−0.32 [−1.27, 0.64]	**1.05 [0.04, 2.06]**
Slope phase II—latissimus dorsi (%/s)	−0.033 ± 0.032	−0.045 ± 0.040	−0.34 [−1.29, 0.62]	−0.040 ± 0.031	−0.058 ± 0.061	−0.38 [−1.34, 0.58]	0.11 [−0.84, 1.06]
Slope phase II—vastus lateralis (%/s)	−0.039 ± 0.061	−0.061 ± 0.063	−0.36 [−1.31, 0.6]	−0.107 ± 0.121	−0.068 ± 0.080	0.38 [−0.58, 1.34]	−0.89 [−1.89, 0.11]
Reoxy rate—biceps brachii (%/s)	0.8 ± 0.4	0.7 ± 0.3	−0.15 [−1.1, 0.80]	0.9 ± 0.7	1.3 ± 0.7$	0.58 [−0.39, 1.55]	−1.00 [−2.00, 0.01]
Reoxy rate—latissimus dorsi (%/s)	1.1 ± 0.8	1.0 ± 0.4	−0.19 [−1.14, 0.76]	1.0 ± 0.5	0.8 ± 0.3	−0.63 [−1.61, 0.34]	0.15 [−0.80, 1.10]
Reoxy rate—vastus lateralis (%/s)	1.2 ± 0.5	1.9 ± 0.3*	**1.41 [0.35, 2.48]**	1.5 ± 0.4	1.7 ± 0.6	0.28 [−0.67, 1.24]	0.52 [−0.44, 1.49]

ES: effect size and 90% confidence interval for difference in means between pre- and post-training measurements in each group and between groups for training induced change, HIIT: high-intensity interval training group, SIT: sprint-interval training group, ΔSmO_2min: maximal deoxygenation (change from baseline) in the muscle, Δ[THb]: change in [THb] between baseline and end of the TT, Deoxy rate: slope of muscle oxygenation at exercise onset, Slope phase II: slope of muscle oxygenation from 12 s after exercise onset to the end of the TT, Reoxy rate: slope of muscle oxygenation at exercise cessation. Bold values indicate significant effect (90% confidence interval does not cross 0)

$ p between 0.05 and 0.10 compared with pre

* $p < 0.05$ compared with pre

to a greater extent compared with a short HIIT program (Stepto et al. 1999). We had hypothesized that, in sprint kayaking, SIT would elicit greater gains in performance compared to SIT, through greater peripheral adaptations.

If both training protocols induced changes in muscle oxygenation parameters, HIIT appears to have induced the largest peripheral adaptations. HIIT induced lower muscle deoxygenation at submaximal intensity, likely due to an increased O_2 delivery in the microcirculation. HIIT also induced moderate and large increases in deoxygenation in the biceps brachii and vastus lateralis in the 1000-m TT. Since performance was improved and increases in

Table 5 Muscle oxygenation response to the 500-m time trial before and after training

	HIIT			SIT			Group
	Pre	Post	ES	Pre	Post	ES	ES
ΔSmO_2min—biceps brachii	−45.8 ± 7.8	−47.7 ± 2.6	−0.32 [−1.27, 0.64]	−49.4 ± 9.5	−52.5 ± 8.1	−0.35 [−1.31, 0.6]	0.18 [−0.78, 1.13]
ΔSmO_2min—latissimus dorsi	−52.8 ± 7.2	−51.6 ± 11.7	0.13 [−0.82, 1.08]	−53.5 ± 13.5	−50.0 ± 6.7	0.33 [−0.63, 1.28]	−0.20 [−1.15, 0.76]
ΔSmO_2min—vastus lateralis	−59.7 ± 6.8	−59.8 ± 13.2	−0.01 [−0.96, 0.94]	−62.0 ± 13.9	−59.6 ± 11.3	0.18 [−0.77, 1.14]	−0.18 [−1.13, 0.77]
Δtotal[heme]—biceps brachii	0.3 ± 0.1	0.3 ± 0.1	−0.23 [−1.18, 0.72]	0.2 ± 0.1	0.2 ± 0.2	−0.04 [−0.99, 0.90]	−0.15 [−1.10, 0.8]
Δtotal[heme]—latissimus dorsi	0.0 ± 0.1	0.1 ± 0.1*	0.58 [−0.39, 1.55]	0.1 ± 0.3	0.0 ± 0.3	−0.42 [−1.38, 0.54]	**1.02 [0.01, 2.03]**
Δtotal[heme]—vastus lateralis	0.1 ± 0.1	0.1 ± 0.2	0.00 [−0.95, 0.95]	0.0 ± 0.2	0.1 ± 0.2	0.42 [−0.54, 1.38]	−0.25 [−1.20, 0.71]
Deoxy rate—biceps brachii (%/s)	−3.4 ± 1.9	−3.5 ± 0.5	−0.05 [−1.0, 0.9]	−5.0 ± 2.1	−5.4 ± 1.3	−0.23 [−1.18, 0.73]	0.18 [−0.77, 1.14]
Deoxy rate—latissimus dorsi (%/s)	−4.7 ± 0.9	−4.7 ± 1.4	−0.04 [−0.99, 0.91]	−6.9 ± 2.6	−4.6 ± 2.3*	0.93 [−0.07, 1.93]	**−2.49 [−3.75, −1.22]**
Deoxy rate—vastus lateralis (%/s)	−3.1 ± 1.6	−4.3 ± 2.1$	−0.68 [−1.65, 0.3]	−3.8 ± 1.4	−2.7 ± 1.5*	0.73 [−0.25, 1.71]	**−2.24 [−3.45, −1.03]**
Slope phase II—biceps brachii (%/s)	−0.087 ± 0.245	0.025 ± 0.137	0.57 [−0.40, 1.54]	0.164 ± 0.241	0.006 ± 0.140	−0.80 [−1.79, 0.19]	**1.40 [0.34, 2.47]**
Slope phase II—latissimus dorsi (%/s)	0.061 ± 0.299	0.098 ± 0.120	0.17 [−0.79, 1.12]	0.029 ± 0.222	0.009 ± 0.188	−0.10 [−1.05, 0.85]	0.33 [−0.62, 1.29]
Slope phase II—vastus lateralis (%/s)	−0.320 ± 0.115	−0.187 ± 0.281	0.62 [−0.35, 1.59]	−0.420 ± 0.372	−0.710 ± 1.072	−0.36 [−1.32, 0.6]	0.71 [−0.27, 1.69]
Reoxy rate—biceps brachii (%/s)	0.7 ± 0.5	0.8 ± 0.4	0.22 [−0.73, 1.17]	0.9 ± 0.7	1.1 ± 0.7	0.28 [−0.67, 1.24]	−0.18 [−1.13, 0.77]
Reoxy rate—latissimus dorsi (%/s)	0.8 ± 0.5	1.1 ± 0.7	0.44 [−0.52, 1.4]	1.0 ± 0.5	0.6 ± 0.3*	−0.91 [−1.91, 0.09]	**1.79 [0.67, 2.92]**
Reoxy rate—vastus lateralis (%/s)	1.6 ± 0.6	1.3 ± 0.6	−0.50 [−1.46, 0.47]	1.6 ± 0.4	1.7 ± 0.4	0.19 [−0.76, 1.15]	−0.62 [−1.59, 0.35]

ES: effect size and 90% confidence interval for difference in means between pre- and post-training measurements in each group and between groups for training induced change, HIIT: high-intensity interval training group, SIT: sprint-interval training group, ΔSmO_2min: maximal deoxygenation (change from baseline) in the muscle, Δ[THb]: change in [THb] between baseline and end of the TT, Deoxy rate: slope of muscle oxygenation at exercise onset, Slope phase II: slope of muscle oxygenation from 12 s after exercise onset to the end of the TT, Reoxy rate: slope of muscle oxygenation at exercise cessation. Bold values indicate significant effect (90% confidence interval does not cross 0)

$ p between 0.05 and 0.10 compared with pre

* $p < 0.05$ compared with pre

performance were strongly associated with increases in vastus lateralis deoxygenation, the most plausible explanation for the decreased SmO_2 is an increase in muscle O_2 extraction/consumption, due to training-induced increase in mitochondrial content (Granata et al. 2018). HIIT also led to a large increase in vastus lateralis reoxy rate, which also suggest an increase in the muscle oxidative capacity with training (Puente-Maestu et al. 2003). In SIT, there were no changes in muscle oxygenation at submaximal intensity, and a moderate increase in latissimus dorsi deoxygenation in the

Table 6 Muscle oxygenation response to the 200-m time trial before and after training

	HIIT			SIT			Group difference
	Pre	Post	ES	Pre	Post	ES	ES
ΔSmO_2min—biceps brachii	−45.7 ± 10.9	−53.0 ± 6.6	−0.81 [−1.80, 0.18]	−49.2 ± 15.2	−55.2 ± 11.9	−0.43 [−1.40, 0.53]	−0.13 [−1.08, 0.82]
ΔSmO_2min—latissimus dorsi	−51.1 ± 6.5	−49.4 ± 5.5	0.28 [−0.67, 1.24]	−51.8 ± 10.8	−50.9 ± 13.2	0.08 [−0.87, 1.03]	0.07 [−0.88, 1.02]
ΔSmO_2min—vastus lateralis	−59.7 ± 16.5	−62.2 ± 14.3	−0.17 [−1.12, 0.79]	−67.6 ± 13.6	−65.1 ± 11.7	0.20 [−0.76, 1.15]	−0.33 [−1.28, 0.63]
Δtotal[heme]—biceps brachii	0.3 ± 0.1	0.3 ± 0.2	0.21 [−0.74, 1.16]	0.3 ± 0.1	0.2 ± 0.3	−0.28 [−1.23, 0.68]	0.58 [−0.39, 1.55]
Δtotal[heme]—latissimus dorsi	0.3 ± 0.2	0.4 ± 0.3$	0.58 [−0.39, 1.55]	0.1 ± 0.2	0.1 ± 0.4	0.02 [−0.93, 0.97]	0.48 [−0.49, 1.44]
Δtotal[heme]—vastus lateralis	0.2 ± 0.1	0.1 ± 0.2	−0.42 [−1.38, 0.54]	0.1 ± 0.2	0.0 ± 0.2	−0.38 [−1.34, 0.58]	0.07 [−0.88, 1.02]
Deoxy rate—biceps brachii (%/s)	−4.9 ± 1.7	−4.7 ± 1.1	0.14 [−0.81, 1.09]	−4.6 ± 2.8	−5.2 ± 2.5	−0.23 [−1.19, 0.72]	0.56 [−0.41, 1.52]
Deoxy rate—latissimus dorsi (%/s)	−6.1 ± 0.6	−6.5 ± 3.4	−0.15 [−1.10, 0.80]	−5.7 ± 2.9	−4.5 ± 2.4	0.45 [−0.52, 1.41]	−0.65 [−1.63, 0.32]
Deoxy rate—vastus lateralis (%/s)	−6.8 ± 3.6	−4.6 ± 2.3	0.73 [−0.25, 1.71]	−4.4 ± 2.4	−2.6 ± 0.6	**1.00 [0.00, 2.01]**	0.14 [−0.81, 1.09]
Slope phase II—biceps brachii (%/s)	−0.065 ± 0.507	−0.396 ± 0.393	−0.73 [−1.71, 0.25]	−0.165 ± 0.262	−0.064 ± 0.294	0.36 [−0.60, 1.32]	**−1.25 [−2.28, −0.21]**
Slope phase II—latissimus dorsi (%/s)	0.087 ± 0.230	−0.016 ± 0.186	−0.49 [−1.46, 0.47]	−0.009 ± 0.161	−0.210 ± 0.321$	−0.79 [−1.78, 0.19]	0.48 [−0.48, 1.44]
Slope phase II—vastus lateralis (%/s)	−0.350 ± 0.335	−0.654 ± 0.404	−0.82 [−1.81, 0.17]	−0.390 ± 0.223	−0.703 ± 0.716	−0.59 [−1.56, 0.38]	0.01 [−0.94, 0.96]
Reoxy rate−biceps brachii (%/s)	0.9 ± 0.3	0.8 ± 0.6	−0.10 [−1.05, 0.85]	1.3 ± 1.0	1.2 ± 0.9	−0.11 [−1.06, 0.84]	0.07 [−0.88, 1.02]
Reoxy rate−latissimus dorsi (%/s)	1.0 ± 0.6	0.9 ± 0.4	−0.13 [−1.08, 0.82]	0.7 ± 0.3	0.7 ± 0.3	0.03 [−0.92, 0.98]	−0.28 [−1.24, 0.67]
Reoxy rate—vastus lateralis (%/s)	1.9 ± 1	2.2 ± 0.8	0.31 [−0.64, 1.27]	1.6 ± 0.9	1.5 ± 0.7	−0.14 [−1.09, 0.81]	0.39 [−0.57, 1.35]

ES: effect size and 90% confidence interval for difference in means between pre- and post-training measurements in each group and between groups for training induced change, HIIT: high-intensity interval training group, SIT: sprint-interval training group, ΔSmO_2min: maximal deoxygenation (change from baseline) in the muscle, Δ[THb]: change in [THb] between baseline and end of the TT, Deoxy rate: slope of muscle oxygenation at exercise onset, Slope phase II: slope of muscle oxygenation from 12 s after exercise onset to the end of the TT, Reoxy rate: slope of muscle oxygenation at exercise cessation. Bold values indicate significant effect (90% confidence interval does not cross 0)

$ p between 0.05 and 0.10 compared with pre

* $p < 0.05$ compared with pre

1000 m TT. However, this increase in deoxygenation was not associated with increases in performance, was concomitant with a moderate, but non-significant decrease in total[heme] and a deceleration in latissimus dorsi reoxy rate in the 500 m TT. Therefore, it is possible that the increase in deoxygenation observed following SIT is due to decreased O_2 delivery, rather than an increased extraction, and therefore that peripheral adaptations following SIT were limited.

Fig. 4 ΔSmO_2 response during 1000-m time trial pre- and post-training in both groups. *$p < 0.05$ compared with pre-training; during the race, each point represents an average over a 50-m segment; before and after the TT, each point represents a 10-s average. **A** HIIT, ΔSmO_2 of the bieps brachii; **B** SIT, ΔSmO_2 of the bieps brachii; **C** HIIT, ΔSmO_2 of the latissimus dorsi; **D** SIT, ΔSmO_2 of the latissimus dorsi; **E** HIIT, ΔSmO_2 of the vastus lateralis; **D** SIT, ΔSmO_2 of the vastus lateralis

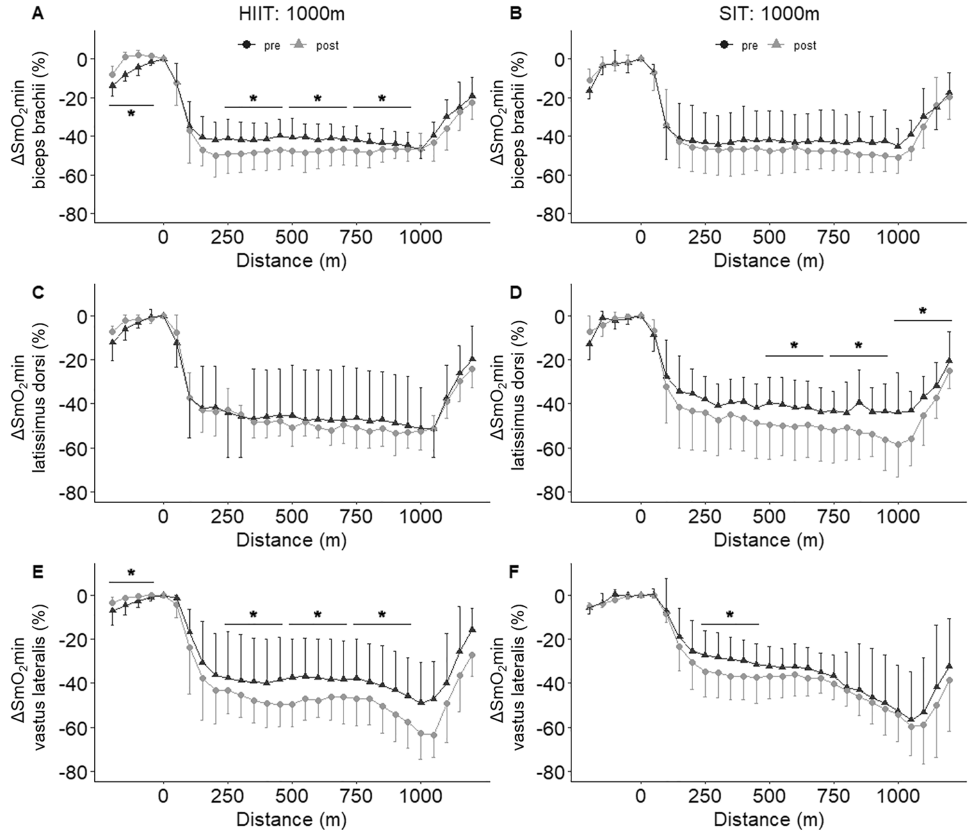
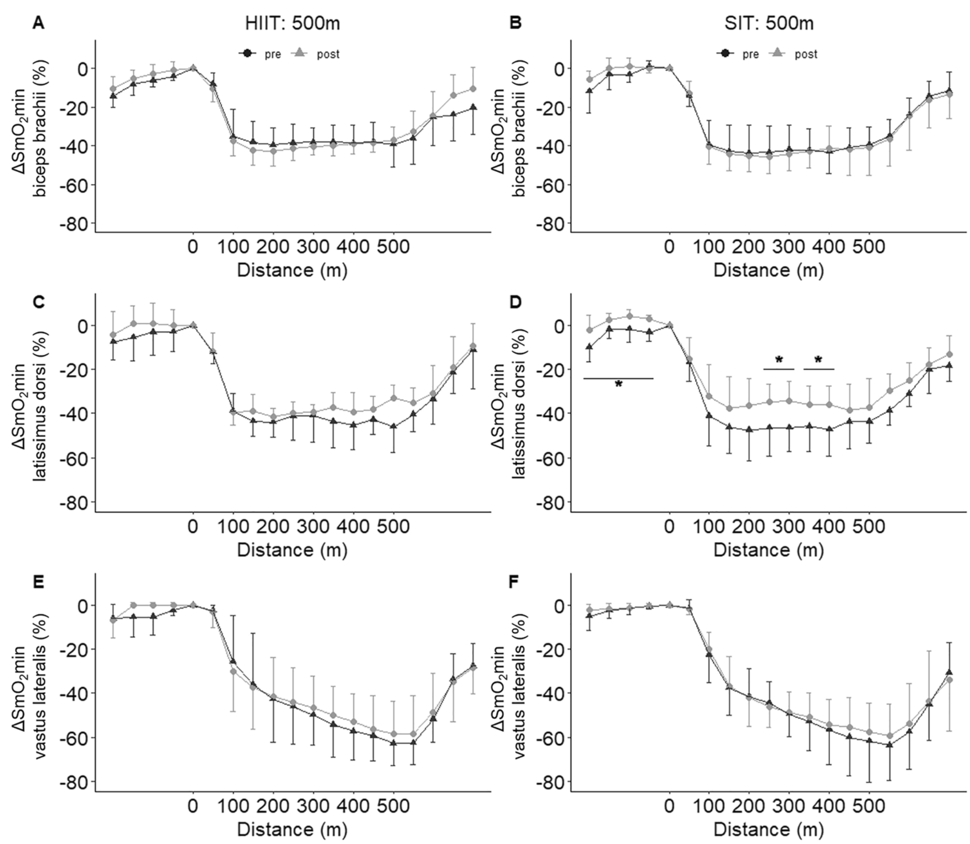


Fig. 5 ΔSmO_2 response during 500-m time-trial pre- and post-training in both groups. *$p < 0.05$ compared with pre-training; during the race, each point represents an average over a 50-m segment; before and after the TT, each point represents a 10-s average. **A** HIIT, ΔSmO_2 of the bieps brachii; **B** SIT, ΔSmO_2 of the bieps brachii; **C** HIIT, ΔSmO_2 of the latissimus dorsi; **D** SIT, ΔSmO_2 of the latissimus dorsi; **E** HIIT, ΔSmO_2 of the vastus lateralis; **D** SIT, ΔSmO_2 of the vastus lateralis



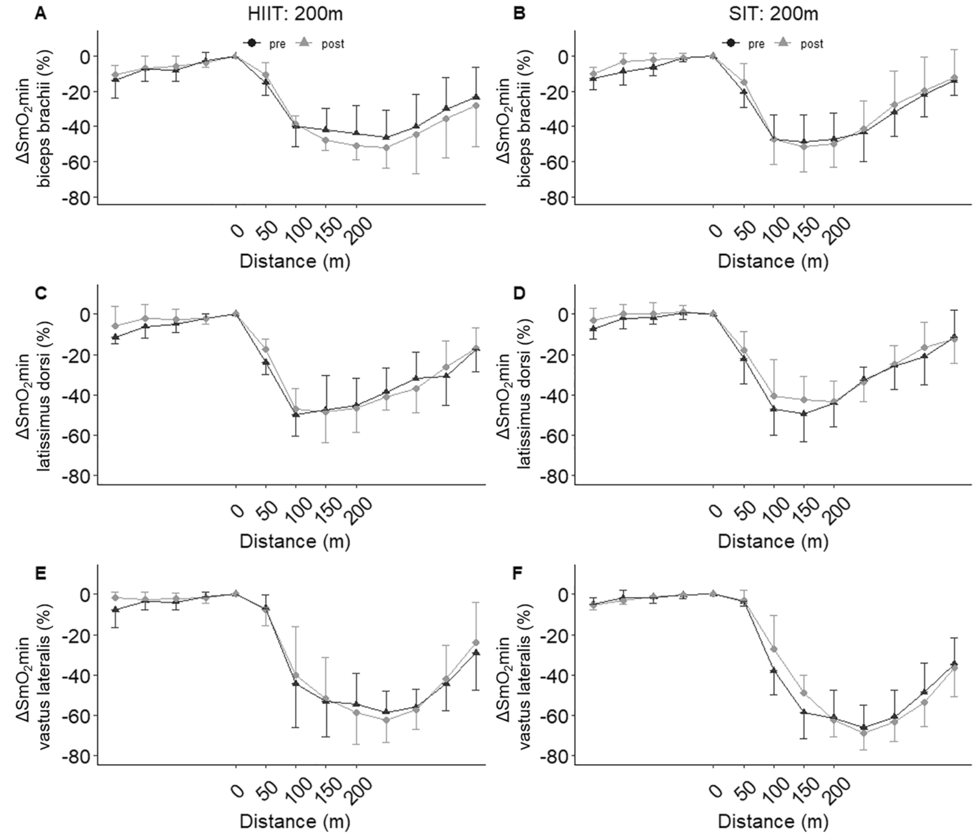


Fig. 6 ΔSmO_2 response during 200-m time-trial pre- and post-training in both groups. *$p < 0.05$ compared with pre-training; during the race, each point represents an average over a 50-m segment; before and after the TT, each point represents a 10-s average. **A** HIIT, ΔSmO_2 of the bieps brachii; **B** SIT, ΔSmO_2 of the bieps brachii; **C** HIIT, ΔSmO_2 of the latissimus dorsi; **D** SIT, ΔSmO_2 of the latissimus dorsi; **E** HIIT, ΔSmO_2 of the vastus lateralis; **D** SIT, ΔSmO_2 of the vastus lateralis

Table 7 Aerobic and anaerobic contribution to the time trials before and after training

	HIIT			SIT			Group difference
	Pre	Post	ES	Pre	Post	ES	ES
VO_2peak (L/min)							
200 m	3.3 ± 0.5	3.5 ± 0.7	0.40 [− 0.56, 1.35]	3.6 ± 0.6	3.4 ± 0.5*	− 0.31 [− 1.26, 0.65]	**1.76 [0.64, 2.88]**
500 m	3.8 ± 0.5	3.9 ± 0.6	0.28 [− 0.67, 1.23]	4.0 ± 0.7	4.0 ± 0.7	0.00 [− 0.95, 0.95]	0.71 [− 0.27, 1.69]
1000 m	3.9 ± 0.7	3.9 ± 0.6	0.03 [− 0.92, 0.98]	4.2 ± 0.7	4.2 ± 0.7	0.03 [-0.92, 0.98]	0.00 [− 0.95, 0.95]
VO_2peak (mL/kg/min)							
200 m	45.3 ± 6.2	48.4 ± 9.4	0.39 [− 0.57, 1.35]	47.9 ± 5.3	45.4 ± 3.8*	− 0.53 [− 1.49, 0.44]	**1.34 [0.29, 2.39]**
500 m	51.3 ± 5.7	53.6 ± 8	0.34 [− 0.62, 1.29]	54.1 ± 6.5	53.8 ± 6	− 0.05 [− 1.00, 0.90]	0.94 [− 0.06, 1.94]
1000 m	54.1 ± 9.3	53.6 ± 8.3	− 0.05 [− 1.00, 0.90]	55.8 ± 6.1	55.7 ± 6.8	− 0.02 [− 0.97, 0.93]	− 0.09 [− 1.04, 0.86]
Peak blood lactate concentration (mmol)							
200 m	15.5 ± 2.0	14.6 ± 3.5	− 0.30 [− 1.25, 0.66]	14.3 ± 2.2	14.3 ± 3.6	− 0.02 [− 0.97, 0.93]	− 0.35 [− 1.31, 0.61]
500 m	17.6 ± 2.1	17.4 ± 3.6	− 0.09 [− 1.04, 0.86]	15.5 ± 1.8	16.3 ± 4.6	0.24 [− 0.71, 1.20]	− 0.31 [− 1.27, 0.64]
1000 m	13.0 ± 4.7	14.7 ± 3.0	0.43 [− 0.53, 1.39]	13.6 ± 2.2	15.1 ± 3.9	0.46 [− 0.51, 1.42]	0.08 [− 0.87, 1.03]

ES: effect size and 90% confidence interval for difference in means between pre- and post-training measurements in each group and between groups for training induced change,, HIIT: high-intensity interval training group, SIT: sprint-interval training group, VO_2peak: highest oxygen consumption over a period of 30-s during the TT. Bold values indicate significant effect (90% confidence interval does not cross 0)

$^{\$}$p between 0.05 and 0.10 compared with pre

*$p < 0.05$ compared with Pre

As per our hypothesis, the training protocol eliciting the greater peripheral adaptations led to the biggest improvements in performance. In addition, changes in latissimus dorsi and vastus lateralis maximal deoxygenation with training were the best predictors of change in performance. These findings support the role of peripheral adaptations as key physiological determinants in sprint kayaking. But contrary to our hypothesis, HIIT was a stronger stimulus than SIT in the current study to elicit peripheral adaptations in sprint kayakers. SIT has been shown to induce similar increases in oxidative capacity compared to HIIT (MacInnis and Gibala 2017), and there are a few parameters that can explain the different findings of the current study. Strength training load was superior in SIT, and recent studies suggest that strength sessions may have detrimental effects on the quality of endurance training sessions, that can limit the adaptations to endurance training (Doma et al. 2017), and could have contributed to the lower improvements in performance in the SIT group. It is also possible that the training volume associated with the SIT sessions was not high enough to induce adaptations in already highly trained athletes. While observations from studies on SIT suggest increasing the number of SIT high-intensity bouts is associated with similar or diminishing returns in sedentary or moderately active subjects (MacInnis and Gibala 2017), the optimal number of SIT repetitions for an athletic population remains uncertain. Finally, a faster performance pre-training was associated with a bigger improvement with training in the 200-m in SIT. Hence, it may be speculated that a high level of anaerobic fitness is required to benefit from such training modality. Anecdotally, the two athletes who experienced decreases in performance following SIT (athletes 2 and 6 in Fig. 1) were female athletes, and so there could also be a gender effect on the adaptations to SIT.

The metabolic perturbations that lead to the activation of coactivators and transcription factors, activating mitochondrial protein synthesis and ultimately increasing muscle oxidative function are well understood (Bishop et al. 2014). Among these, some are proportional to O_2 consumption, like ROS production, redox status or cellular O_2 partial pressure, and therefore would potentially be maximal when the training intensity is around VO_2max. Others are proportional to exercise intensity, like calcium ions concentration and AMP or ADP/ATP, and would probably be maximized at supramaximal intensities. It is hence possible that the combination of HIIT performed near the speed at VO_2max and SIT, performed at supramaximal intensities, would be an optimal stimulus to induce increases in muscle oxidative capacity, and studies looking at the effect of combining different training stimulus on peripheral adaptations are required.

Training-induced changes in physiological parameters

$\dot{V}O_2$peak did not change with training in any group. In previous studies, changes in $\dot{V}O_2$peak or $\dot{V}O_2$max following SIT in trained subjects have varied from −2 to +5% (Gist et al. 2014; Rosenblat et al. 2020). A recent meta-analysis revealed no significant effect of SIT or short HIIT on $\dot{V}O_2$max in an athletic population (Wen et al. 2019), and confirmed that longer work intervals were needed to increase $\dot{V}O_2$max in such population. Change in $\dot{V}O_2$peak and peak blood lactate concentration were the best predictors of change in 1000-m performance in SIT, suggesting SIT may increase performance through both aerobic and anaerobic adaptations. While VO_2 kinetics were not measured in this study, changes in deoxy rate following SIT may suggest increases in VO_2 kinetics, as a slowing of SmO_2 kinetics is usually associated with an acceleration of VO_2 kinetics (Grassi et al. 2003). An acceleration of VO_2 kinetics could lead to a smaller accumulation of anaerobic by-products and a reduction in neuromuscular fatigue, inceasing performance in high-intensity efforts (Temesi et al. 2017).

The muscle oxygenation response to the training protocols varied between muscles, HIIT being associated with greater maximal deoxygenation in the biceps brachii and vastus lateralis while SIT was associated with greater maximal deoxygenation in the latissimus dorsi. The difference in maximal deoxygenation between muscles was at least partly due to differences in total[heme]. In the 500-m and 1000-m, there was a significant group effect for latissimus dorsi Δtotal[heme], which increased with training in HIIT and decreased with training in SIT. There was also a non-significant decrease in biceps brachii and vastus lateralis Δtotal[heme] in the 1000-m for HIIT, while it increased slightly or did not change with SIT. In a previous study, electromyography (EMG) patterns have revealed differences in muscle recruitment with increasing paddling intensity (Fleming et al. 2012). Hence, since training intensity influences muscle recruitment, we can therefore speculate that peripheral adaptations may differ between muscles, according to training type. This is supported by studies on rat muscle, where fast-twitch, white skeletal muscle exhibited the largest increase in oxidative capacity, capillary density and blood flow capacity with SIT, while endurance training adaptations were exhibited primarily in slow-twitch, red skeletal muscle (Laughlin and Roseguini 2008). While total[heme] is not a measure of blood flow, it could suggest that these two training regimens induced vascular adaptations differently between investigated muscles, and these differences may be due to differences in muscle typology between muscle groups.

Maximal muscle deoxygenation did not change with training in the 200-m and 500-m TT, despite increased performance. Previous studies have found increased deoxygenation during short anaerobic performances (Jones and Cooper 2014; Jones et al. 2015; Delextrat et al. 2018). In particular, in 10 elite women hockey players, 6 weeks of cycling SIT were associated with an increased deoxygenation (larger decrease in tissue saturation index, ΔTSI) and an increased O_2 extraction (larger increase in deoxy[heme]) during repeated 30-s all-out sprints (Jones et al. 2015). Increases in muscle deoxygenation happened in all participants in the first 30-s sprint. The lack of change in maximal deoxygenation in the current study could come from the shorter training protocol, the kayak athletes being more aerobically trained than hockey players, and the upper-body dominant exercise modality used in this study.

Limits

Results must be interpreted with the following limits. Although the chosen statistical approach is suited for the analysis of small sample size data and elite-level athletic performance (Hopkins et al. 2009), we acknowledge that the current power may not have permitted to detect some of the subtle training-induced physiological changes associated with the 1–4% improvement in TT performance. However, we still detected valuable training-induced changes in this very specific population of trained kayakers. As it is often the case in training studies performed in athletic populations, there was no control group in the current study. It is possible that normal training would also elicit changes in physiology and performance, and it is not possible to assess how the changes observed in this study would compare to habitual training adaptations in these athletes. Furthermore, while the use of session RPE for training load quantification has been validated against more objective training load measurements (van Erp et al. 2019), and was used in this study to allow for comparison of training load from different training modalities, it should be acknowledged that RPE is not a direct measure of training intensity, as it is also influenced by training volume. The heterogeneity in competitive level of kayakers involved in this study has led to important variability in fitness and training load between participants. Therefore, it cannot be excluded that competitive level and training load acted as confounding factors in this study. However, a similar increase in deoxygenation in a 200-m and a 1000-m race were reported in a group of 8 international level male kayakers in a previous study (Paquette et al. 2020), suggesting the adaptations described in this study could realistically manifest in a group of elite athletes.

Conclusion

The current data set on trained kayakers indicates that there was a greater increase in performance over 200 m, 500 m and 1000 m after nine sessions of HIIT vs. SIT. Training did not change $\dot{V}O_2$peak, but increased muscle maximal deoxygenation, suggesting HIIT and SIT elicit peripheral adaptations in this group. Performance improvement in the 1000 m was associated with increased maximal muscle deoxygenation, reinforcing the contribution of peripheral adaptations to performance in a sport like sprint kayaking. While NIRS has not been used widely on the field with high performance athletes, the current results suggest that portable NIRS could be a helpful additional monitoring tool to track training effects on muscle oxygen extraction in elite athletes.

Authors' contributions All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by MP. The first draft of the manuscript was written by MP and all authors commented on previous versions of the final manuscript. All authors read and approved the final manuscript.

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Availability of data and materials The datasets generated during the current study are available from the corresponding author on reasonable request.

Declarations

Conflict of interest The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Ethics approval This research was approved by the Comité d'éthique de la recherche en sciences de la santé de l'Université Laval, file number 2016-089 Phase 2.

Consent to participate Informed consent was obtained from all individual participants included in the study.

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