Effect of prior exercise on pulmonary O$_2$ uptake and estimated muscle capillary blood flow kinetics during moderate-intensity field running in men

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Buchheit M, Laursen PB, Ahmaidi S. Effect of prior exercise on pulmonary O$_2$ uptake and estimated muscle capillary blood flow kinetics during moderate-intensity field running in men. J Appl Physiol 107: 460–470, 2009. First published June 4, 2009; doi:10.1152/japplphysiol.91625.2008.—The effect of prior exercise on pulmonary O$_2$ uptake (V$_{O_2}$p) and estimated muscle capillary blood flow (Q_m) kinetics during moderate-intensity, field-based running was examined in 14 young adult men, presenting with either moderately fast (16 s < $\tau$V$_{O_2_p}$ < 30 s; MFK) or very fast V$_{O_2_p}$ kinetics ($\tau$V$_{O_2_p}$ < 16 s; VFK) (i.e., primary time constant, $\tau$V$_{O_2_p}$). On four occasions, participants completed a square-wave protocol involving two bouts of running at 90–95% of estimated lactate threshold (Mod1 and Mod2), separated by 2 min of repeated supramaximal sprinting. V$_{O_2_p}$ was measured breath by breath, heart rate (HR) beat to beat, and vastus lateralis oxygenation (deoxy-hemoglobin/myoglobin concentration [deoxy-[Hb+Mb]]) using near-infrared spectroscopy. Mean response time of Q_m (Q_m MRT) was estimated by rearranging the Fick equation, using V$_{O_2_p}$ and deoxy-[Hb+Mb] as proxies of muscle O$_2$ uptake (V$_{O_2}$) and arteriovenous difference, respectively. HR, blood lactate concentration, total hemoglobin, and Q_m were elevated before Mod2 compared with Mod1 (all $P < 0.05$). $\tau$V$_{O_2_p}$ was shorter in VFK compared with MFK during Mod1 (13.1 ± 1.8 vs. 21.0 ± 2.5 s, $P < 0.01$), but not in Mod2 (12.9 ± 1.5 vs. 13.7 ± 3.8 s, $P = 1.0$). Q_m MRT was shorter in VFK compared with MFK in Mod1 (8.8 ± 1.9 vs. 17.0 ± 3.4 s, $P < 0.01$), but not in Mod2 (10.1 ± 1.8 vs. 10.5 ± 3.5 s, $P = 1.0$). During Mod2, HR kinetics were slowed, whereas mean deoxy-[Hb+Mb] response time was unchanged. The difference in $\tau$V$_{O_2_p}$ between Mod1 and Mod2 was related to Q_m MRT measured at Mod1 ($r = 0.71$, $P < 0.01$). Present results suggest that local O$_2$ delivery (i.e., Q_m) may be a factor contributing to the V$_{O_2_p}$ kinetic during the onset of moderate-intensity, field-based running exercise, at least in subjects exhibiting moderately fast V$_{O_2_p}$ kinetics.

oxygen uptake dynamics; near-infrared spectroscopy; muscle deoxygenation; repeated sprint exercise; warm-up

COMMENCEMENT OF EXERCISE INSTIGATES an increase in O$_2$ delivery to working muscles to support the required increase in muscle oxygen uptake (V$_{O_2}$; V$_{O_2_m}$). However, the kinetics supporting the V$_{O_2_m}$ at exercise onset, now shown convincingly to be inferred from the pulmonary V$_{O_2}$ (V$_{O_2_p}$) (27, 37), are still debated (40). Resolution of our knowledge surrounding whether V$_{O_2_m}$ is limited by adequate delivery of O$_2$ and/or other substrates required for mitochondrial oxidative phosphorylation (i.e., metabolic inertia) is essential for understanding metabolic control in health, the mechanics of impaired (slowed) V$_{O_2_m}$ kinetics found in patient populations (25, 26), and, eventually, for the design of appropriate exercise training interventions (3).

The current viewpoint surrounding the O$_2$ delivery/metabolic inertia debate is that it is a false dichotomy; that is, there may be a “tipping point” in the relationship between the speed of the V$_{O_2_p}$ kinetics [expressed using the time constant ($\tau$) of the primary component (i.e., phase II) of V$_{O_2_p}$] and muscle O$_2$ delivery (40). As highlighted by Poole et al. (40), the kinetics to the left of this tipping point (i.e., short $\tau$V$_{O_2_p}$) appear to be more essentially O$_2$-delivery dependent, whereas the right side is more likely determined by the availability of other substrates limiting O$_2$ utilization. It is, however, worth noting that the tipping point is more of a terminological concept than a real physiological variable or a parameter that we can currently measure. Mechanistic exploration into the basis of V$_{O_2_p}$ kinetics have used “priming” exercises in the past (e.g., Refs. 7, 10, 11, 19, 24, 28, 29). Compared with exercise transition without warm-up, V$_{O_2_p}$ kinetics upon initiation of heavy exercise subsequent to intense exercise appear consistently accelerated (the “overall” V$_{O_2_p}$ kinetics being significantly faster, due predominantly to a marked reduction in the amplitude of the so-called V$_{O_2_p}$ “slow component”). Mechanisms put forward to explain this phenomenon hint at enhanced muscle O$_2$ supply and delivery [i.e., increased cardiac output, muscle blood flow (Q_m) and total hemoglobin ([Hb]), or blood acidosis enhancing O$_2$ dissociation from Hb], and/or partial relief of muscle oxidative metabolic inertia [i.e., faster enzyme activation and improved substrate provision, illustrated by a shorter time delay (TD) before near-infrared spectroscopy (NIRS) muscle deoxygenation; deoxy-Hb/myoglobin concentration (deoxy-[Hb+Mb]), and/or alterations in motor unit recruitment profiles (for review, see Ref. 12).

Studies examining the effect of prior heavy exercise on V$_{O_2_p}$ adaptation at the onset of moderate-intensity exercise have been conflicting. Heavy-intensity warm-up exercise has been shown to have no effect on subsequent moderate-intensity V$_{O_2_p}$ exercise kinetics in young adults (11, 22, 24), yet quickened $\tau$V$_{O_2_p}$ in older adults (19, 44), reinforcing the assumption that V$_{O_2_p}$ kinetics in the moderate-intensity domain might be age- or at least “V$_{O_2_p}$ kinetic dependent” (i.e., related to the initial $\tau$V$_{O_2_p}$ value; the initial speed of V$_{O_2_p}$ adaptation). Indeed, subjects with a poor (slow) initial O$_2$ delivery capacity are most likely to present with shorter $\tau$V$_{O_2_p}$ after heavy exercise (19, 28, 29, 44). In contrast, Gurd et al. (29) recently reported that prior heavy cycling exercise hastened the V$_{O_2_p}$ adaptation, even in young subjects presenting with fast (i.e., <30 s) V$_{O_2_p}$ kinetics. However, subjects classified as having “fast V$_{O_2_p}$ kinetics” (i.e., 26 s on average) in the study by Gurd et al. had, in fact, rather moderately fast
kinetics compared with those reported in the literature for moderate exercise [i.e., ~20 s (14, 36, 41)]. Thus, whether subjects experiencing very fast VO2p kinetics (VK) [i.e., <16 s, presumably less likely to be limited by O2 delivery (40)], would respond in a similar fashion, is yet to be investigated. For example, during running exercise, young trained subjects generally present faster VO2p kinetics compared with cycling (14, 36). As such, it may be inappropriate to make inferences from the results of priming exercise studies involving cycling vs. running exercises. To date, only one study has investigated the effect of a priming exercise on the VO2 response following the onset of (high-intensity) treadmill running exercise (33); none has considered the transition to moderate-intensity running exercise in the field.

In an attempt to gain insight into the respective determinants regulating VO2 at the onset of moderate-intensity, field-running exercise, we sought to simultaneously investigate VO2p kinetics, muscular deoxygenation, and estimated Qm responses in subjects presenting either moderately fast VO2p kinetics (16 s < τVO2p < 30 s; MFK) or VK (τVO2p < 16 s), while using a supramaximal “priming exercise” [i.e., repeated sprints (38, 45)] that was likely to improve O2 delivery (9, 46, 50) and/or to speed muscle oxidative activity (7, 12). We expected that, irrespective of the mechanisms involved (i.e., increased O2 delivery and/or partial relief in metabolic inertia), VO2p kinetics in subjects exhibiting MFK before the warm-up would show a significantly accelerated response after the supramaximal warm-up (19, 28, 29, 44). Conversely, whether subjects exhibiting VK without warm-up would present a further quickening of VO2 adaptation after priming was difficult to predict. If the priming supramaximal exercise (SE) had somehow influenced enzymatic control of oxidative phosphorylation, then we would expect the kinetics on the subsequent moderate-intensity exercise bout to be quickened, at least as long as O2 delivery was not slower than muscle oxidative activity. Conversely, a lack of change in the VO2p kinetics in subjects exhibiting VK without warm-up would suggest that the warm-up did not likely affect metabolic inertia, and/or that O2 delivery was not likely a critical factor controlling VO2 adaptation following exercise onset in these subjects (40). As with other studies (e.g., Refs. 19, 20, 22, 29), we believed that investigation of changes in Qm and muscle deoxygenation on submaximal exercise onset following SE would assist with understanding of the mechanisms involved with the control of VO2p kinetics.

METHODS

Subjects

Based on the assumption that a 10 ± 5 s difference in the τ for the primary phase of the VO2 response upon initiation of exercise is meaningful (19, 24), we used Minitab 14.1 software (Minitab, Paris, France) to determine that a sample size of at least eight participants would provide a statistical power of 0.8 at an α-level of 0.05. To further increase the power of our study, we recruited 14 moderately trained athletes (23 ± 4 yr, 77 ± 9 kg, 179 ± 5 cm). All participants were involved (5.1 ± 2.8 h/wk) in running-based sporting activities (running, soccer, handball, or basketball) and had no history or clinical signs of cardiovascular or pulmonary diseases. Participants were not currently taking prescribed medications and presented with normal blood pressure levels and electrocardiographic patterns. The study conformed to the recommendations of the Declaration of Helsinki, and participants gave voluntary, written consent to participate in the experiment, which was approved by the local institute’s human research ethics committee.

Study Overview

Participants were tested on five separate occasions on an indoor synthetic track, where ambient temperature ranged from 18 to 22°C. To minimize possible circadian effects, all tests were initiated at the same time of day (±1 h). Subjects first performed a graded maximal aerobic test [30–15 Intermittent Fitness Test (30–15IFT) (4)] for the determination of the estimated lactate threshold (τ01), peak VO2 (VO2peak), and a reference velocity for the ongoing moderate-intensity exercise bouts (Vmax). Following this test, subjects returned to the track on four separate occasions to perform two moderate-intensity exercise transients (Mod1 and Mod2) at 90–95% of estimated τ01, interspersed with a 2-min SE following by 5 min of passive recovery (Fig. 1). Each transient was 5 min in duration and was preceded by 2 min of standing rest. No warm-up was allowed before Mod1. The time between the end of Mod1 and SE was 5 min (i.e., standardized warm-up consisting of a few athletic drills and short bursts of progressive accelerations on the track). Time between the end of SE and Mod2 was chosen to ensure an optimal priming effect on the following moderate exercise bout (8, 10). Respiratory gas exchange, heart rate (HR), and Hb variables of the vastus lateralis (NIRS) were recorded during the entire session. Blood lactate was also measured after each test. Participants indicated their rating of perceived exertion (0–10 Borg’s scale) immediately at the end of each test. Subjects were told not to perform exercise on the day before testing, and to consume their (usual) last meal at least 3 h before the scheduled test time.

Exercise Testing

Maximal graded aerobic test. Maximal aerobic performance of each subject was assessed using a 30–15IFT (5). This intermittent shuttle field test elicits similar levels of VO2peak compared with a standard, continuous incremental test [i.e., r = 0.71; 95% confidence interval (CI) for mean difference, −1.0−3.5 ml·min−1·kg−1] (5) and has been shown to be accurate to estimate τ01 (5). Elicitation of the final running speed (Vmax) during this test has also been shown to be reliable [intraclass correlation coefficient = 0.96; typical error = 0.33 (95% CI, 0.26–0.46) km/h] (4). The 30–15IFT consisted of 30 s shuttle runs interspersed with 15-s passive recovery periods. For this test, velocity was set at 8 km/h for the first 30-s run, and speed was increased by 0.5 km/h every 30-s stage thereafter. The velocity attained during the last completed stage was determined as the subject’s Vmax.

Moderate-running exercise bouts. Exercise intensity for Mod1 and Mod2 was set at 45% of Vmax and was chosen for subjects to reach a VO2p corresponding to ~90–95% of that observed at τ01 (5). An audio time countdown was given to the subjects 3 s before the commencement of the test. Running pace was governed by a prerecorded beep that sounded at appropriate intervals to allow participants to adjust their running speed as they passed through specific zones of the field (i.e., a cone placed every 20 m). Particular attention was focused on ensuring that the subject reached the required running speed within at
least 5 s [participants had to be near an additional cone, placed 10–12 m (depending on their running speed) from the starting line; i.e., within 5 ± 1 s]. If adjustment to the required running speed was not satisfactory (i.e., subjects passed a cone outside of a 1-s difference compared with expected time), the test was stopped, and the subject was asked to recommence the test after a 5-min period of passive recovery.

SE. The SE test chosen was adapted from previous repeated sprint running tests (6, 45). Before the study, all subjects were familiarized with the repeated-sprint protocol. Subjects performed six repetitions of maximal 25-m sprints, departing every 25 s (Wireless Timing-Radio Controlled, Brower Timing System). During the first ~18 s of recovery between sprints, subjects performed an active running recovery (2.0 m/s). Three seconds before the commencement of each sprint, subjects were asked to assume the ready position and await the start signal. During recovery, audio feedback (i.e., time countdown) was provided so that participants could maintain the required running speed. Participants were instructed to complete all sprints as fast as possible, and strong verbal encouragement was provided to each during all sprints. To assess eventual neuromuscular fatigue, the percentage of speed decrement (%Dec) was calculated for each session (6).

Measurements

Cardiorespiratory measures. Respiratory gas exchanges and HR were measured during the 30–15 sFT, Mod1, and Mod2 using an automated breath-by-breath system (K4b2, Cosmed, Rome, Italy) (21). Before each test, the O2 and CO2 analysis systems were calibrated as recommended by the manufacturer. Data were automatically filtered for aberrant data points [i.e., greater than 4 SDs from the local mean (39)]. For the 30–15 sFT, data were averaged in 20-s increments. Estimated \( \theta_t \) was defined as the VO2 at which CO2 output began to increase disproportionately in relation to VO2, with a systematic rise in the minute ventilation-to-VO2 ratio and end-tidal PO2, while the minute ventilation-to-CO2 output ratio and end-tidal PCO2 remained stable. VO2peak was arbitrarily defined as the highest VO2 values attained during two consecutive 20-s periods (5). A HR peak attained near predicted maximum (220-age), a lactate blood concentration ([La]b) higher than 8 mmol/l, and a respiratory exchange ratio > 1.1 were additionally required to confirm the maximal nature of the test (5).

NIRS measurements. The portable NIRS apparatus (Portamon, Artinis, Medical System, Zetten, The Netherlands) used in this study was a two-wavelength, continuous-wave system, which simultaneously used the modified Beer-Lambert and spatially resolved spectroscopy methods. The procedure used to collect data was the same as that used by Van Beekvelt et al. (47) using a similar nonportable device. Changes in tissue oxy-Hb/myoglobin concentration (oxy-[Hb+Mb]), deoxy-[Hb+Mb], and \( \text{Hb} \) were measured using the differences in absorption characteristics of light at 750 and 850 nm. The photodiodes detect changes in the transmission of radiation as a function of time, distance, and wavelength. In the present study, a differential pathlength factor was not determined, but a value of 3.83 was used to allow comparison with previous studies (18, 19, 29). Values for oxy-[Hb+Mb], deoxy-[Hb+Mb], and \( \text{Hb} \) were reported as a change from baseline (30-s averaging before each test) in micromolar units (\( \mu \)M) (17), are thus considered as “arbitrary units” throughout this paper. As the deoxy-[Hb+Mb] signal can be regarded as being essentially blood volume independent during exercise (15), the method was assumed to be a reliable estimator of changes in intramuscular oxygenation status and O2 extraction in the area of interrogation (15, 17). The NIRS probe was positioned on the vastus lateralis muscle of the right leg, ~10 cm from the knee joint and along the vertical axis of the thigh. A surgical marker and a numerical picture were used to mark the probe placement for accurate repositioning during the four trials. The probe and the skin were covered with black tape to prevent contamination from ambient light. Skinfold thickness at the site of application of the NIRS probe was determined before the testing sessions using Harpenden skinfold calipers (British Indicators). The calculated value of skin and subcutaneous tissue thickness was less than one-half the distance between the source and the detector. During all tests, the NIRS system was connected to a personal computer by Bluetooth for data acquisition (10 Hz), analog-to-digital conversion, and subsequent analysis.

Blood lactate measurement. Three minutes after the end of each exercise set, a fingertip blood sample (5 \( \mu l \)) was collected, and [La]b was determined (Lactate Pro, Arkray). The accuracy of the analyzer was checked before each test using standards. The suitability and reproducibility of this analyzer have been previously established throughout the physiological range of 1.0–18.0 mmol/l (42).

Data Analysis

Assessment of cardiorespiratory kinetics. VO2 values recorded during the four sets of two moderate-intensity running bouts were automatically filtered for aberrant data points (39), interpolated to 1-s intervals, time-synchronized, and ensemble-averaged to yield two single responses for each subject (i.e., Mod1 and Mod2). VO2 on-transient kinetics were modeled using an iterative technique (Sigmaplot 10, SPSS Science; Chicago, IL) using a monoeponential function (Eq. 1) (39, 48):

\[ \text{VO2}(t) = \text{VO2}_b + A_1 \times \left[ 1 - e^{-t / \text{TD1}} \right] \times U_1 \] (1)

where \( U_1 = 0 \), when time (t) is less than the time delay 1 (TD1) from the onset of exercise; \( U_1 = 1 \), for t \( \geq \) TD1; \( \text{VO2}_b \) is the VO2 before the onset of the rest-to-exercise transition (ml), \( A_1 \) is the asymptotic amplitude for the exponential term (ml); \( t \) is the time constant of the exponential (s); and TD1 represents the TD for each equation (s). The initial cardiodynamic component was excluded by deleting the first 20 s of data after the onset of exercise, so that the primary component parameters were not distorted by any early cardiodynamic influence (48). Beat-by-beat HR data were filtered for aberrant beats, time-aligned, and averaged to 1-s time bins. These data were then fit from exercise onset with a monoeponential model of the form in Eq. 1.

NIRS data and assessment of deoxyhemoglobin kinetics. The NIRS-derived oxy-[Hb+Mb], deoxy-[Hb+Mb], and \( \text{Hb} \) data were time-aligned and ensemble averaged to 1-s time bins to yield two single responses for each subject (i.e., Mod1 and Mod2). As previously proposed (e.g., Ref. 22), the first 90 s of deoxy-[Hb+Mb] were modeled using an exponential function of the form given in Eq. 1 to determine the time course of muscle deoxygenation, where deoxy-[Hb+Mb] is substituted for VO2p. Our choice to analyze the deoxy-[Hb+Mb] changes within only the first 90 s was motivated by previous observations made by Ferreira et al. (22). That is, only minor changes in deoxy-[Hb+Mb] tend to be shown from 90 to 360 s (~1 \( \mu \)M) (e.g., Refs. 17, 18, 22). Therefore, including the 360-s period in the monoeponential fitting would likely lead to distortion of the initial TD and the \( \tau \) of the early, primary response of interest. The amplitude of the increase in deoxy-[Hb+Mb] from the start to the end of exercise was also calculated. The mean response time (MRT) (deoxy-[Hb+Mb]) is defined as the deoxy-[Hb+Mb] TD + \( \tau \) deoxy-[Hb+Mb]), was also calculated to provide a description of the overall time course for muscle deoxygenation. The oxy-[Hb+Mb] and \( \text{Hb} \) signals did not approximate an exponential response, and, as a result, these data were not modeled.

Muscle capillary blood flow. One-second time interval VO2p and deoxy-[Hb+Mb] responses were first synchronized using time markers set during each recording (i.e., start of exercise). The Qm response to exercise was derived from the kinetics of VO2p and deoxy-[Hb+Mb], as previously proposed by Ferreira et al. (23). The present estimation is based on the fact that it) during constant work rate exercise, \( \tau \text{VO2}_p \) has been shown to approximate the VO2m kinetic

\[ \text{Qm} = \frac{\Delta \text{Hb}_O2}{\Delta t} \]
(\(\tau V\O_2_{m}\)) (e.g., Ref. 2), and 2) deoxy-[Hb +Mb] measured by NIRS is thought to be a function of the \(V\O_2_{ar-to-Qm}\) ratio (19, 20). Therefore, if one assumes that arterial \(O_2\) saturation does not change substantially during moderate-intensity exercise, the evolution of deoxy-[Hb +Mb] can then be considered as being proportional to \(O_2\) extraction (i.e., arteriovenous \(O_2\) difference). By rearranging the Fick equation, the temporal characteristics of \(Qm\) were estimated using the following formula:

\[
Q_m(t) = \frac{V\O_2_{p}}{\text{deoxy-[Hb +Mb]}}(t)
\]

It is worth noting, however, that, because the precise proportional contribution of arterial and venous blood to the deoxy-[Hb +Mb] signal is unknown for skeletal muscle, the amplitude of \(Qm\) is quantitatively uncertain. Nevertheless, the temporal characteristic of deoxy-[Hb +Mb], and thus \(Qm\), is likely to be preserved. \(V\O_2_{m}\) kinetics were thus estimated using the kinetic parameters of \(V\O_2_{p}\) obtained from the curve fitting (i.e., by assuming that \(V\O_2_{m}\) rose exponentially at time zero with the \(\tau\) and amplitude determined for the primary component of the \(V\O_2_{p}\) response). Finally, the time course of variance was found for the fit between modeled and measured \(V\O_2_{m}\) data exponentially at time zero with the

\[
Q_{\text{m,MRT}} = A_1 \times [1 - e^{-U_2 t}] + U_1 + A_2 \times [1 - e^{-U_2 t}]
\]

where \(U_1 = 0\), when time \(t\) is less than the \(TD_1\) (s) from the onset of exercise; \(U_2 = 1\), for \(t \geq TD_1\); \(U_2 = 0\), when time \(t\) is less than the \(TD_2\) (s) from the onset of exercise; \(TD_1 = 1\), for \(t \geq TD_2\); \(Q_{\text{m,MRT}}\) is the \(Qm\) before the onset of the rest-to-exercise transition (arbitrary units); \(A_1\) and \(A_2\) are the asymptotic amplitude for exponential terms (arbitrary units); and \(\tau_1\) and \(\tau_2\) are the time constants of the exponential (s).

A double-exponential model was used when a significant gain of variance was found for the fit between modeled and measured \(Qm\) data compared with a single-exponential model. The overall time course of the response was determined from the MRT (\(Qm\), MRT, s), which was calculated from a weighted sum of the delay and the \(\tau\) of each component. The MRT was equivalent in time to the point at which the response reached \(\sim 63\%\) of the difference between the baseline \(Qm\) and the new steady-state value, independent of the model used (23):

\[
Q_{\text{m,MRT}} = [A_1/(A_1 + A_2)] \times (TD_1 + \tau_1) + [A_2/(A_1 + A_2)] \times (TD_2 + \tau_2)
\]

where the parameters are from Eq. 2 and subsequent text.

Statistical Analysis

The distribution of each variable was examined with the Shapiro-Wilk normality test. Homogeneity of variance was verified by a Levene test. Possible differences in anthropometric characteristics and cardiorespiratory fitness variables between the two groups were tested with an independent \(t\)-test. Changes in \([La]_{b}\), cardiorespiratory and NIRS data, and estimated \(Qm\) values for the two moderate exercise bouts in both groups were analyzed using a two-way repeated-measures ANOVA, with one within factor (i.e., “repetition”, Mod1 vs. Mod2) and one between factor (i.e., “groups”, MFK vs. VFK). When a significant interaction was identified, a Bonferroni post hoc test was used to further delineate differences between repetition and/or groups. Linear regressions with Pearson’s coefficients (±95% CI) were used to establish relationships between cardiorespiratory kinetics. Other polynomial regressions were rejected on the basis of importantly higher residuals. For all analyses, the level of significance was set at \(P \leq 0.05\). All statistical analyses were carried out using Minitab 14.1 (Minitab, Paris, France). Data are presented as means ± SD.

RESULTS

Subjects were placed into two groups, according to their \(\tau V\O_2_p\) determined in response to the transition to moderate-intensity running exercise without prior high-intensity exercise (i.e., Mod1, MFK, \(\tau V\O_2_p of > 16\ s\); VFK, \(\tau V\O_2_p of < 16\ s\)). The groups were the same (all \(P > 0.05\)) with respect to age, stature, body mass, and maximal cardiorespiratory variables, whereas peak \([La]_{b}\) was higher in VFK than in MFK (Table 1).

During the entire experimentation, only three subjects (5%) were asked to restart their test (Mod1) because of excessive running speed at the initiation of the run exercise. Their repeated performances were thus included in the final analysis. As expected, running at 45% \(V\IFT\) was just below a moderate-intensity exercise level; mean steady-state \(V\O_2\) was just below \(\theta_1\) (60 ± 7 and 59 ± 3% \(\tau V\O_2_{peak}\), corresponding to 92 ± 5 and 93 ± 4% of \(\theta_1\) for MFK and VFK, respectively), and the \(V\O_2\) response was well fitted by a monoeXponential function in all cases (see below). During all tests, none of the participants presented with \(V\O_2\) values higher than that at \(\theta_1\). Repeated sprint exercises elicited similar cardiorespiratory responses in both groups: 81 ± 10 and 79 ± 8% of \(\tau V\O_2_{peak}\) (\(P = 0.42\)), and 94 ± 7 and 94 ± 4% of peak HR (\(P = 0.45\)) in MFK than in VFK, respectively. Mean \([La]_{b}\) 3 min after exercise was lower in MFK than in VFK (9.3 ± 1.9 vs. 11.3 ± 0.5 mmol/l, \(P = 0.02\)). Nevertheless, expressed as percentage of peak \([La]_{b}\), \([La]_{b}\) was similar in both groups (90 ± 15 vs. 93 ± 12% in MFK and VFK, respectively, \(P = 0.33\)). The %Dec during the sequence of repeated sprints was similar in both groups (3.3 ± 0.9 vs. 3.4 ± 0.7% in MFK and VFK, respectively, \(P = 0.42\)).

Table 1. Characteristics of the participants

<table>
<thead>
<tr>
<th></th>
<th>MFK</th>
<th>VFK</th>
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<tbody>
<tr>
<td>(n)</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>Age, yr</td>
<td>23.1 ± 4.5</td>
<td>23.2 ± 3.6</td>
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<tr>
<td>Stature, cm</td>
<td>180 ± 6</td>
<td>179 ± 5</td>
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<tr>
<td>Body mass, kg</td>
<td>76.1 ± 9.2</td>
<td>78.2 ± 9.8</td>
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<td>(V\IFT), km/h</td>
<td>19.7 ± 0.8</td>
<td>19.5 ± 0.6</td>
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<tr>
<td>(V\O_2_{peak}), l/min</td>
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<td>4.00 ± 0.62</td>
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<tr>
<td>(V\O_2_{peak}), ml·min(^{-1})·kg(^{-1})</td>
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<td>51.2 ± 8.5</td>
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<td>(%\O_2) at (V\IFT), %\O_2)peak</td>
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<td>67.1 ± 5.4</td>
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<tr>
<td>(HR)peak, beats/min</td>
<td>183 ± 8</td>
<td>183 ± 8</td>
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<tr>
<td>([La]_{b,peak}), mmol/l</td>
<td>10.4 ± 1.7</td>
<td>12.3 ± 1.4*</td>
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<tr>
<td>RER</td>
<td>1.16 ± 0.1</td>
<td>1.12 ± 0.1</td>
</tr>
<tr>
<td>RPE</td>
<td>8.9 ± 0.7</td>
<td>9.1 ± 0.6</td>
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</table>

Values are means ± SD of anthropometric characteristics of the participants; \(n\), no. of subjects. Maximal velocity (\(V\IFT\)), peak oxygen uptake (\(V\O_2_{peak}\), expressed in absolute and relative units), \(V\O_2\) at the first ventilatory threshold (\(V\IFT\)), peak heart rate (\(HR\)peak), peak blood lactate (\([La]_{b,peak}\)), gas exchange ratio (RER), and rating of perceived exertion (RPE) reached during the graded aerobic test in individuals exhibiting moderately fast (MFK) and very fast \(O_2\) uptake kinetics (VFK) without prior supramaximal warm-up are shown.
using the monoeXponential model in all subjects \((r = 0.99 \pm 0.01)\), with a mean 95% CI of \(\tau \pm 2 s\) for both Mod1 and Mod2, respectively. As illustrated in Table 3, there was neither a group nor a repetition effect for \(\tau V\bar{O}_{2,p}\) amplitude and \(\tau V\bar{O}_{2,p}\) TD (all \(P > 0.09\)). For baseline \(V\bar{O}_{2,p}\), there was a repetition effect \((P < 0.001)\), but no group effect \((P = 0.84)\). For \(\tau V\bar{O}_{2,p}\), we observed a repetition effect \((P < 0.01)\), as well as a group \((P < 0.01)\) and repetition \(\times\) group interaction \((P < 0.01)\). Post hoc tests revealed that \(\tau V\bar{O}_{2,p}\) for Mod1 in MFK was longer than values observed for Mod2 in MFK, but also higher than values calculated for Mod1 and Mod2 in VFK (all \(P < 0.01)\). There was, however, no difference between Mod1 and Mod2 in VFK \((P = 1.00)\). There was no significant effect on \(V\bar{O}_{2,p}\) TD (all \(P > 0.19)\).

HR Kinetics

HR data are presented in Table 4 and illustrated in Fig. 4 for two representative subjects in MFK and VFK groups. Data were very well fitted using the monoeXponential model in all subjects \((r = 0.98 \pm 0.01)\), with a mean 95% CI of \(\tau \pm 2 s\) for Mod1 and Mod2, respectively. We found a repetition effect for baseline HR, HR amplitude, and \(\tau HR\) (all \(P < 0.001)\), but neither a group (all \(P > 0.09)\) nor a group \(\times\) repetition interaction (all \(P > 0.21)\). There was also neither a repetition nor a group effect for HR TD (all \(P > 0.19)\).
Table 4. Heart rate kinetics during the first and second bouts of moderate field running

<table>
<thead>
<tr>
<th></th>
<th>MFK</th>
<th>VFK</th>
<th>MFK</th>
<th>VFK</th>
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<tbody>
<tr>
<td></td>
<td>Mod1</td>
<td>Mod2</td>
<td>Mod1</td>
<td>Mod2</td>
</tr>
<tr>
<td>Baseline HR, beat/min</td>
<td>79±2</td>
<td>110±13*</td>
<td>80±9</td>
<td>118±10*</td>
</tr>
<tr>
<td>HR amplitude, beat/min</td>
<td>53±10</td>
<td>36±8*</td>
<td>63±8</td>
<td>38±5*</td>
</tr>
<tr>
<td>HR TD, s</td>
<td>1±1</td>
<td>2±3</td>
<td>0±1</td>
<td>0±2</td>
</tr>
<tr>
<td>tHR, s</td>
<td>18.2±7</td>
<td>30±10*</td>
<td>16±7</td>
<td>33±6*</td>
</tr>
</tbody>
</table>

Values are means ± SD for baseline, amplitude, TD, and time constant of the primary component for heart rate (tHR) in subjects exhibiting MFK and VFK without prior supramaximal warm-up. *Significant repetition effect (P < 0.05).

Deoxyhemoglobin Kinetics

Figure 5, top, illustrates the profiles of deoxy-[Hb+Mb] during transition from rest to moderate running exercise for two representative subjects in MFK and VFK groups. The deoxy-[Hb+Mb] response, restricted to the first 90 s, was accordingly fitted with a monoexponential model in all subjects (r = 0.99 ± 0.01, with a mean 95% CI of τ ± 1 and τ ± 2 s for Mod1 and Mod2, respectively). As shown in Table 5, there was neither a group (all P > 0.10) nor repetition (all P > 0.22) effect on baseline oxy-[Hb+Mb], baseline deoxy-[Hb+Mb], deoxy-[Hb+Mb] amplitude, and deoxy-[Hb+Mb] MRT. In contrast, there was a repetition effect for baseline tHb, deoxy-[Hb+Mb] TD, and τ deoxy-[Hb+Mb] (all P < 0.001), but neither a group (all P > 0.18) nor a group × repetition interaction (all P > 0.33). The increase in deoxy-[Hb+Mb] within the first 90 s (Δdeoxy-[Hb+Mb]), for a given increase in VO₂p (ΔVO₂p), was similar in all subjects and did not change after the SE [i.e., there was neither a repetition (P = 0.70) nor a group effect (P = 0.38)]. Values were 3.5 ± 2.0, 3.3 ± 2.3, 2.9 ± 1.6, and 2.4 ± 1.6 μM·L⁻¹·min⁻¹ for Mod1 and Mod2 in MFK and VFK groups, respectively. Similar results were obtained when considering deoxy-[Hb+Mb] and VO₂p steady-state and end-exercise values.

Estimated Muscle Capillary Blood Flow Kinetics

Normalized Q_m profiles during transition from rest to moderate exercise (as a percentage of the final response) are illustrated in Fig. 5, bottom, for two representative subjects in MFK and VFK groups; MRT Q_m values are presented in Table 5. Data from Mod2 in MFK, and from both Mod1 and Mod2 in VFK, did not display the two expected phases at the start of exercise (i.e., rapid increase, phase 1, followed by a less pronounced increase, phase 2). Thus responses were fitted with a monoexponential model (r = 0.99 ± 0.01). For Mod1 in the MFK group, visual examination of six subjects’ responses revealed that a biexponential function was more suitable (Fig. 5, bottom left), which was confirmed by a slightly higher goodness of fit (SE of estimate = 0.49 ± 0.09 and r = 0.998 ± 0.001 vs. SE of estimate = 1.3 ± 0.1 and r = 0.999 ± 0.001 for the bi- vs. monoexponential model). Mean 95% CI of kinetics for τQ_m was mean ± 1 s for both Mod1 and Mod2. There was no effect on Q_m TD and overall Q_m amplitude (all P > 0.21). Baseline Q_m was increased before Mod2 (significant repetition effect, P < 0.001). For Q_m MRT, we observed a repetition effect (P = 0.05), as well as a group (P < 0.01) and repetition × group interaction (P < 0.01). Post hoc tests revealed that Q_m MRT for Mod1 in MFK were higher than values observed for Mod2 in MFK, but also higher than values calculated for Mod1 and Mod2 in VFK (all P < 0.01). There were significant correlations between Q_m MRT and τVO₂p for Mod1 [r = 0.90 (0.71, 0.97), P < 0.001] and Mod2 [r = 0.89 (0.68, 0.96), P < 0.001] (Fig. 6). Difference between τVO₂p for Mod1 and Mod2 was significantly related to MRTQ_m measured at Mod1 [r = 0.71 (0.29, 0.90), P < 0.01] but not Mod2 [r = −0.19 (−0.65, 0.38), P = 0.53].

DISCUSSION

The aim of the present study was to investigate, for the first time, the effect of a repeated sprint running sequence on VO₂p kinetics, muscular deoxygenation, and estimated Q_m responses during transition to moderate-intensity field-running exercise. The main findings were that 1) prior SE quickened the VO₂p kinetics during subsequent moderate-running exercise only in subjects presenting with relatively MFK without warm-up; 2) the magnitude of the speeding of the VO₂p kinetics after heavy-intensity exercise was related to the “lag” in the Q_m (and VO₂p) kinetics during moderate-intensity exercise without prior heavy-intensity exercise, with a greater shortening in τVO₂p seen in those individuals with the longer initial Q_m MRT; 3) HR and tHb were elevated at baseline before the onset of Mod2 compared with Mod1, suggesting that cardiac output and muscle perfusion were elevated before the onset of Mod2; 4) the TD before muscle deoxygenation was reduced in both
the respective determinants regulating $\dot{V}O_2$ at the onset of the exercise intensities in our protocol to allow comparison of perform a preceding high-intensity exercise bout), we chose moderate intensities, and older or diseased populations do not col is not initially evident (i.e., athletes do not compete at Exercise in the Field

Use of a Repeated Running Sprint Sequence as a Priming Exercise in the Field

While the practical application of the present study’s protocol is not initially evident (i.e., athletes do not compete at moderate intensities, and older or diseased populations do not perform a preceding high-intensity exercise bout), we chose the exercise intensities in our protocol to allow comparison of the respective determinants regulating $\dot{V}O_2$ at the onset of moderate-running exercise, as previously described on ergo-

Table 5. Deoxyhemoglobin and estimated muscle capillary blood flow kinetics during the first and second bouts of moderate-intensity, field-based running

<table>
<thead>
<tr>
<th></th>
<th>MFK</th>
<th>VFK</th>
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<tr>
<td></td>
<td>Mod₁</td>
<td>Mod₂</td>
</tr>
<tr>
<td>Baseline $\text{oxy-}[\text{Hb+Mb}]$, µM</td>
<td>30.7±4.4</td>
<td>34.5±6.2</td>
</tr>
<tr>
<td>Baseline $\text{Hb}$, µM</td>
<td>53.2±2.9</td>
<td>58.1±4.2*</td>
</tr>
<tr>
<td>Baseline $\text{deoxygeny-}[\text{Hb+Mb}]$, µM</td>
<td>23.5±1.3</td>
<td>23.7±1.6</td>
</tr>
<tr>
<td>End-exercise $\text{deoxygeny-}[\text{Hb+Mb}]$, µM</td>
<td>30.9±1.9</td>
<td>30.1±2.4</td>
</tr>
<tr>
<td>$\text{Deoxy-}[\text{Hb+Mb}]$ amplitude, µM</td>
<td>6.3±3.9</td>
<td>5.2±4.0</td>
</tr>
<tr>
<td>$\text{Deoxy-}[\text{Hb+Mb}]$ TD, s</td>
<td>15±3</td>
<td>9±2*</td>
</tr>
<tr>
<td>$\tau_{\text{Deoxy-}[\text{Hb+Mb}]}$, s</td>
<td>8±4</td>
<td>12±7*</td>
</tr>
<tr>
<td>$\text{Deoxy-}[\text{Hb+Mb}]$ MRT, s</td>
<td>22±4</td>
<td>21±6</td>
</tr>
<tr>
<td>Baseline $\dot{Q}_m$, AU</td>
<td>17.5±8.9</td>
<td>30.2±7.2*</td>
</tr>
<tr>
<td>$\dot{Q}_m$, TD, s</td>
<td>0±0</td>
<td>0±0</td>
</tr>
<tr>
<td>$\dot{Q}_m$, MRT, s</td>
<td>17±4</td>
<td>11±4*</td>
</tr>
</tbody>
</table>

Values are means ± SD for baseline, amplitude, TD, and mean response time (MRT) for muscle deoxy-hemoglobin/myoglobin concentration (deoxygeny-[Hb+Mb]) and muscle blood flow ($\dot{Q}_m$), as well baseline values for oxy-hemoglobin/myoglobin concentration (oxy-[Hb+Mb]) and total hemoglobin (Hb) in subjects exhibiting MFK and VFK without prior supramaximal warm-up. *Significant repetition effect ($P < 0.05$). †Significant group effect ($P < 0.05$). Note that near-infrared spectroscopy-derived baseline values are based on a differential pathlength factor of 3.83.
Presenting with VFK before warm-up, a speeding of the V˙O2p kinetics by Gurd et al. (29), our results confirm that, in individuals exercise onset and neither do interventions such as hyperoxic recruitment during Mod2 (7, 12). Muscle fatigue (38), which could have influenced motor unit 46), the %Dec during the repeated sprints (i.e., /H11015 [i.e., changes in metabolic processes intrinsic to the specific fibers involved, allowed by the faster delivery of other substrates, such as acetyl CoA, ADP, Pi, NADH, FADH2 after priming exercise), the lack of improvement of V˙O2p kinetics in subjects experiencing VFK [i.e., presumably less likely to be limited by O2 delivery (40)] suggests that the heavy warm-up did not affect local factors controlling O2 utilization (i.e., did not reduce the metabolic inertia). Nevertheless, our results suggest that, in individuals having slower V˙O2p kinetics, the likelihood of observing a measurable speeding of the response is greater than for those individuals that have relatively faster V˙O2p kinetics. Although this lack of a change is likely to be physiologically driven, it is also possible that our inability to measure a “true” reduction in V˙O2p may be due to a combination of limited precision in our technology, along with response variability (31).

Effect of Prior SE on V˙O2p Kinetics

After the priming exercise, baseline V˙O2p values were higher than before the first exercise bout (Table 3 and Fig. 2). Nevertheless, as per previous work (8, 10), we postulated that the “speeding effects” of priming exercise would be independent of baseline V˙O2p [i.e., changes in τV˙O2p are not likely to be an artifact of an unchanged response superimposed on an elevated baseline V˙O2p (10)] and felt confident with the acceleration of V˙O2p kinetics observed here. Consistent with previous findings made in running studies (13, 14, 33, 36), we have also shown moderate-to-fast V˙O2p kinetics (i.e., 18 s on average). The relatively fast V˙O2p adaptation at exercise onset was likely due to both the young age of our participants and the field-running exercise mode used (14, 36). Individuals who experienced MFK but not VFK at the initiation of moderate exercise without warm-up presented a shorter τV˙O2p following the 2-min repeated running sprint exercise (Fig. 2). Moreover, the magnitude of τV˙O2p shortening was strongly related to τV˙O2p at the transition of the first exercise without warm-up (P = 0.01), Fig. 3). Extending on the findings made by Gurd et al. (29), our results confirm that, in individuals presenting with VFK before warm-up, a speeding of the V˙O2p kinetics is not likely to occur with increases in O2 delivery. Indeed, in young reasonably fit subjects, prior exercise generally does not alter the phase II τ of the V˙O2p response following exercise onset and neither do interventions such as hypoxic gas breathing or erythropoietin use (12, 40, 41, 49, 51). Although the shortening of τV˙O2p in individuals having initially MFK [i.e., subjects presumably more likely to be limited by O2 delivery (40)] could have been related to both an increased bulk of O2 delivery and an accelerated oxidative phosphorylation (i.e., changes in metabolic processes intrinsic to the specific fibers involved, allowed by the faster delivery of other substrates, such as acetyl CoA, ADP, Pi, NADH, FADH2 after priming exercise), the lack of improvement of V˙O2p kinetics in subjects experiencing VFK [i.e., presumably less likely to be limited by O2 delivery (40)] suggests that the heavy warm-up did not affect local factors controlling O2 utilization (i.e., did not reduce the metabolic inertia). Nevertheless, our results suggest that, in individuals having slower V˙O2p kinetics, the likelihood of observing a measurable speeding of the response is greater than for those individuals that have relatively faster V˙O2p kinetics. Although this lack of a change is likely to be physiologically driven, it is also possible that our inability to measure a “true” reduction in V˙O2p may be due to a combination of limited precision in our technology, along with response variability (31).

Effect of Prior SE on Indexes of Central and Peripheral O2 Delivery

The marked metabolic acidosis induced by the priming SE probably contributed to an enhanced vasodilatation early in exercise, combined with an acidosis-induced rightward Bohr shift in the oxyhemoglobin dissociation curve, with improved O2 off-loading from Hb after high-intensity exercise (12). However, since intracellular acidosis is known to have a direct inhibitory effect on mitochondrial function (35), blood acidosis in itself cannot be held accountable for the shortening of τV˙O2p (as shown by the significant correlation between [La]b before Mod1, and the difference between τV˙O2p for Mod1 and Mod2 (r = −0.68, P = 0.01)), but might instead be used as a proxy variable for another process modulating the V˙O2p response to exercise [i.e., improved O2 delivery (10)]. Although absolute [La]b was higher in the VFK group, observation of comparable relative values (i.e., as a percentage of peak blood lactate reached during the maximal graded test, Table 1) suggest that, on an individual basis, subjects from both groups might have benefited to a similar extent from the acidosis. Absolute HR values were significantly elevated before the second exercise bout in both groups, suggesting improved central O2 delivery (Fig. 4). Moreover, HR kinetics, thought to reflect changes in cardiac output [as stroke volume changes minimally with exercise above baseline levels (16)], were similar in the two groups during both moderate-intensity exercise bouts. An elevated local O2 delivery (i.e., a relative hyperemia) was also confirmed in both groups before Mod2 by the greater tHb, reflecting an increased volume of Hb and myoglobin within the field of NIRS interrogation (Table 5). Taken together, these changes likely contributed to the elevated convective O2 delivery to the muscle at the start of the second exercise bout (29).

As previously proposed (22, 23), we estimated Qm by rearranging the Fick equation and used V˙O2p and deoxy-[Hb+Mb] as surrogates of VO2 m and arteriovenous O2 difference. Limitations of this technique are known, and estimated Qm kinetics likely represent the true Qm kinetics to within.
Effect of Prior Exercise on Muscle Deoxygenation

At the transition to Mod₁, we found similar deoxy-[Hb+Mb] dynamics in both groups (i.e., similar TD, τ, and thus MRT), despite noticeable differences in τV₂O₂p (discussed above). This would suggest that muscle deoxygenation dynamics were not likely directly responsible for the slower V₀₂m kinetic at exercise onset without warm-up in the MFK group. Conversely, it is probable that the slower Qₘ characterizing these subjects might have limited muscle O₂ delivery, which could, in turn, have slowed V₀₂m. The similar final deoxy-[Hb+Mb] asymptote value in both groups, as well as a similar Δdeoxy-[Hb+Mb] to ΔV₀₂p ratio, was thus consistent with a similar O₂ extraction (i.e., arteriovenous O₂ difference) in all subjects. Associated with the slower Qₘm, this was likely to be responsible for the slower V₀₂m (and consequently slower V₀₂p) kinetics in the MFK subjects. Nevertheless, whether V₀₂m limitations always result in slower Qₘ kinetics and muscle O₂ delivery is uncertain; faster microvascular deoxygenation leading to lower PO₂ may also lead to a possible impairment in diffusive O₂ delivery and, consequently, to slower V₀₂m.

After SE, a similar shortening of deoxy-[Hb+Mb] TD occurred in both groups, whereas V₀₂p kinetics were only accelerated in the MFK group. This finding suggests the absence of a relationship between the changes in the response of muscle deoxygenation at exercise onset and those seen for τV₂O₂p (22, 32), as well as an “alteration of the dynamic interaction between V₀₂m and Qₘm following exercise onset” (22). However, since muscle deoxygenation is generally expected to relate to O₂ utilization along with Qₘm, this particular observation warrants further examination. The faster muscle deoxygenation at exercise onset following high-intensity warm-up has been consistently reported in the literature (19, 22, 29), but has generally been associated with faster V₀₂p kinetics (1, 18, 29). It is now believed that the deoxy-[Hb+Mb] delay reflects a complex balance between Hb/Mb deoxygenation, O₂ delivery, and the effect of the muscle pump on microvascular volume. As suggested by Delorey et al. (17), it is thus possible that V₀₂m is actually increased during the delay, and any increase in deoxy-[Hb+Mb] might be “masked” by other factors, which impact on the volume of Hb in the field of NIRS interrogation. The shortening of the deoxy-[Hb+Mb] TD might, therefore, reasonably account for a faster and/or greater activation of muscle O₂ utilization relative to the increase in local Qₘ at exercise onset. It is possible that the prior exercise affected metabolic pathways and substrates for oxidative phosphorylation, so that these probably contributed to the more rapid adaptation of deoxy-[Hb+Mb]. Delorey et al. (18) propose that the observation of a slower rate of deoxy-[Hb+Mb] adaptation in conjunction with a shorter phase II τ provide evidence that an O₂ delivery limitation in the control condition would have been alleviated by priming exercise. Indeed, it is apparent that improved blood flow redistribution and local muscle perfusion consequent to prior SE contributed to the slower deoxy-[Hb+Mb] response seen in both groups during Mod₂. However, as observed by Ferreira et al. (22), the shortening of the deoxy-[Hb+Mb] TD was adjusted for by the longer τ deoxy-[Hb+Mb], so that the mean deoxy-[Hb+Mb] response time was not affected by prior exercise. In the MFK group, despite no change in mean deoxy-[Hb+Mb] response time, the higher Qₘ observed after warm-up was presumably responsible for the faster V₀₂p adaptation. In the VFK group, we can hypothesize that, without change in Qₘm, a faster V₀₂p response could only have been reached with a shorter deoxy-[Hb+Mb] MRT (1, 18, 29). The lack of a V₀₂p kinetic change observed in these subjects is thus consistent with the unchanged response in local hemodynamic measures (i.e., similar Qₘm and deoxy-[Hb+Mb] MRT).

To conclude, present results show for first time that local O₂ delivery (i.e., Qₘm) could be a possible factor regulating the V₀₂ kinetic during the onset of moderate-intensity, field-based running exercise. However, up to certain fast V₀₂p kinetic rates (i.e., <16 s), improvement in O₂ delivery is likely to have no further effect on the V₀₂m adaptation following moderate-intensity exercise onset.

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